The Jaffna Medical Journal

volume 27 issue 1 November 2011

The Journal of Jaffna Medical Association

Jaffna Medical Association

Council - 2011

President
Dr.S.Raviraj

Senior Vice President
Dr.G.Bavani

Junior Vice President
Dr.S.K.Arulmoli

President Elect
Dr.G.Selvaratnam

Secretary
Dr.S.Premakrishna

Treasurer
Dr.S.Sivakonesan

Editor
Dr.P.Lakshman

Librarian
Dr.S.Jeyaratnam

Asst. Librarian
Dr.T.Peranantharajah

Council members
Prof.K.Sivapalan
Dr.S.Sivayokan
Dr.T.Kumanan
Dr.K.Umapathy
Dr.M.Guruparan
Dr.Thuraisamy Sarma
Dr.P.Jeevahan
Dr.S.Umahsuthan
Dr.B.Abarna
Dr.S.Karthigan

Contents

Editorial

The Need to Give Primacy to the Institution over the Individual

A major challenge for healthcare professionals all over the world is to ensure that they are building a health care system, which could foresee and withstand difficulties that may lie ahead in the future. It would be a herculean task to be able to predict the future challenges because these challenges largely depend on multiple and complex external factors. The difficulty is much more acute in developing countries since the existing socio-cultural and political systems could pose further challenges in achieving this goal. I will take this space to point to three areas in which I think immediate attention is needed.

Firstly, the need to enhance the role of the patient in the treatment process. Patients in countries like Sri Lanka are mostly ignorant of their rights and responsibilities. Thus the responsibility of managing an illness is placed entirely on the doctor. This would sometimes lead to the violation of the rights of the patients and have negative implications on the management of the disease. This situation has to be remedied. This is essential to ensure and improve the quality of service rendered by the health care professionals.

Secondly, the need for change in the attitudes of doctors towards taking a more wholistic approach to their patients. Patients have to be seen in the backdrop of their socio-cultural systems as well as through individual factors. In other words factors such as age, gender, social class, level of education and religion should be considered by the doctor while treating a patient. At the same time it is also important that medical professionals are not affected by the consumer culture, a necessary evil of the globalization and continue to deal with the patient with a human touch.

Thirdly we should focus on redesigning hospital management structures that give primacy to institution-building over individuals. The existing management systems of various service sectors within the medical terrain, usually follow a top down approach as far as the decision making is concerned. Thus the individual becomes more important and prominent than the service sector for which he or she works. This would have serious negative consequences to the functioning of the service sector especially in the absence of the particular individual. The institution or service sectors should be given importance rather than individuals in order to ensure the quality of the services rendered. By focusing on the institution we need to work on building overall institutional knowledge and capacity. The strengthening of the institutions or service sector would be pivotal to the sustainability and would enhance the team spirit, which is very important for ensuring the quality of service in the health sector.

Let us sincerely hope that positive changes, both on the side of the patient and on the side of the health sector, would strengthen the existing mechanisms to ensure the health care of the population and to prepare them to face the challenges in the future.

Editor

Presidential address - JMA 2010

Optimal care in changing reality

The chief guest Dr. H.N Rajaratnam, the president and members of the Jaffna Medical Association, distinguished guests, colleagues and friends,

It is my pleasure to welcome you all for the inauguration ceremony of the academic sessions of Jaffna Medical Association.

The Jaffna Medical Association, popularly known as JMA, has a prestigious history in the medical field in Sri Lanka. Since its inception in 1941 it has been providing an esteemed platform to the medical professionals working in the Northern Sri Lanka. It has continuously been engaging with upgrading the medical education of the doctors and medical students through clinical meetings, case conferences, seminars, workshops, guest lectures, publications and providing library services. Its academic sessions and journal provide an opportunity to learn and an arena to many young doctors to mix up with their senior colleagues. The JMA has been chaired by many celebrities and I am privileged to be here as the president of JMA for this session.

The present council took over JMA in 2006 and within months a full blown war was resumed again. The mindset and the environment were very challenging during the period. However, we managed to continue our educational programmes. The weekly clinical sessions and guest lecturers have been continued without much interruption. We didn't stop there. A monthly newsletter was trailed for one year in 2007. A telemedicine service was piloted in 2008. Internet service has been set up in the library since 2008. Night time library services were provided during 2008-2009 period. Nevertheless, we were unable to have our academic sessions during these four years. It was planned to have the annual session in November 2008 but unfortunately the situation was not conducive and the sentiments were collectively hurt during those days and we had to postpone it.

I am glad today that we are almost going with the same plan we had in 2008. The chief guest, the guest speakers, the symposiums..... Everything remains the same and we are actually implementing the plans we had in 2008. I have to express my sincere thanks to the honorable chief guest of this occasion and all the other colleagues and guest lecturers who have readily accepted our invitation and expressed their willingness to attend and contribute to this session.

It is interesting to see that we have been living in a world which is changing continuously. Invariably changes do occur in the medical field too. Some drugs and intervention methods which seem very promising and fascinating one day may turn out to be as ineffective ones in another day. The understanding about the causes of illness and the possible interventions the medical professionals can accomplish do change constantly. Even defining the problems and illnesses change from time to time. As a result, the medical professionals need to update their knowledge and skills continuously. Academic sessions are one of the many avenues where we could update ourselves.

We could see that over the past years, there was a great shift in the attitude and conceptualization of the doctor patient relationship. The patients no more want to adapt the powerless role in the doctor patient relationship. Even the terminology of patients has started to change in many medical institutions. Some use the term clients and some use the term consumers, and some use the term service users. This shift reflects the changing trend in the service delivery model

S.Sivayokan
President, JMA 2010.

among the different health services. If someone has an illness what is wrong with if we call him or her as a patient? But in the traditional doctor patient relationship the patients become powerless and the doctors become demigods. And the demigods are overwhelmed by their poor patients' illnesses. We could notice that over the years our patients become more interested in knowing about their problems and possible treatment or management options. We could also experience that nowadays they have started discussing or even challenging some of our ideas and advices.

The concept of free medical services in this country is also changing. Free medical services provide a very good chance for majority of the people who come under low earning category. But in the meantime the paid services are growing very fast and we can see the private medical institutions in almost all the corners of cities and suburbs. Ironically sometimes we could observe that the poorer tend to pay and get the services in the private sector while the affluent richer influence the free medical services and get what they want to get.

The trends in the diagnosis, investigations and the treatment options are changing rapidly and if we think seriously we could realize that majority of these trends are directly and/or indirectly influenced by the manufacturers and distributors of medical supplies. These entrepreneurs have the abilities for systematically influencing the hearts and minds of the medical professionals. The craving for change and novelty is very normal to the mankind and the medical profession is not an exception to it.
There is a change in the attitude among the patients and their relations. Nowadays our patients have gradually been challenging the views of the medical professionals. There were warning signs that litigations against medical profession would become more and more in future. This will reflect in our practice and we may have to shift our usual service oriented model to a more legal oriented model. In other words we may have to involve with more paper work and defensive practice than what we did in the past.

Along with the changes that occur globally and nationally there are many changes occurring at a local level too. We have just entered in to a post war era. In the past we faced numerous difficulties and challenges due to the direct and indirect effects of war and due to the change of powers and ruling styles. Now a great change has happened. The roads have opened; boats have gained access to the seas; and the time has come with no curfews. Our place has become a popular and crowded visiting area; roads are full of vehicles and smoke. But the hearts are still filled with pain and deep sorrow.

We have to deal with a community which has gone through enormous damages, losses and scars. The community has lost its traditional cohesiveness and enthusiasm. There is a huge amount of suspiciousness, mistrust, selfishness and ennui. The values and cultural norms which were considered as the finest at one time have been deteriorating very fast. The sudden exposure and accessibility towards the information technology and the multicultural interactions and exchanges are greatly contributing to this type of changes happening in the community. Like in the war era, sometimes I personally feel confusion and perplexity in adjusting with the rapid changes and forced normalization.

From the health point of view, we have been observing a change in the morbidity and mortality patterns. For the first time we had a bad Dengue outbreak and it took a long time for us to keep it under control though we are still afraid of future outbreaks. Road traffic accidents are on the increase and become the leading killer. Substance abusers and related health problems are on the increase. Premarital sexual relationships and teen age pregnancies are being reported in high numbers. Early warning signs of sexual transmitted diseases especially HIV infections are visible. Hospital beds, especially cardiac beds are being occupied by young people with a history attempted suicide which is a very much disturbing reality at present. The number of reported carcinomas are also on the increase. From the psychological side, people are suffering from the ill effects of their past life; many do suffer from unresolved grief and depression. From a sociological point of view suddenly many people have become homeless and/or abandoned without care givers. Day by day we are getting requests for long term more like a permanent placements for these disables, orphans and elders.

It is interesting to see the changes among the medical profession too. For a long period, and probably even now, the doctors from Jaffna have a good recognition and reputation for their commitment and hard work

towards their profession. Many of these doctors tend to spend their days and nights in the hospitals. They never deserted their patients and faced all the hardships along with their patients. In fact some were even killed while on duty with the stethoscope the prestigious symbol of medical profession. But I could see an alarming transformation in the doctors' attitude over the years. Few doctors tend to breach the ethics and etiquettes of our profession. It appears that a shift has been happening from a service oriented attitude to a materialistic craving one over the years.

If this is the reality, a change which cannot be prevented since it is the nature's law, and then we have to seriously think about how best we could optimize our service deliveries in the changing context. A service delivery which depends on evident based findings; recognizes the patients' rights; respects the ethical principles; holds the cultural and gender sensitiveness; and values the legal obligations is needed at present. The clinicians should be competent enough to handle the machines and technologies properly without allowing them to handle them. And the preventive and curative sectors should go along with hand in hand to optimize the health deliveries. Mankind has the great capacity to adapt towards any changes, pleasant or unpleasant. I am sure we could find better ways and solutions amidst the ever-changing scenarios.

I would like to take this opportunity to thank all the old and new council members of the JMA and other active members who stood behind organizing this wonderful session. Our new president is very keen on having this missed session during our period. Our editor took great pain to re-launch the esteemed Jaffna Medical Journal after 15 years and I humbly request you to consider this as a trial version and we are certain that our next issue will meet all the standard criteria for a scientific journal. I would also like to express our sincere gratitude to the entire expatriate medical communities and their organizations for their tireless support to improve the health services of the needed regions and sectors.

I wish that the JMA should take an active role in educating the medical professionals both in the curative sector and in the preventive sector. It should also take an active role in imparting the right knowledge to the community. Being an academic body it should be able to come out with guidelines in order to update the doctors and staff in the health sector. More over it should voice against all the health hazards in our area including environmental pollution. I have great hope that the association can achieve this with its charismatic leadership.

Before finishing my address, let me quote a poem from *Geethanjali* by the great poet and Nobel Prize winner *Rabindranath Tagoore*.

> Where the mind is without fear and the head is held high
> Where knowledge is free
> Where the world has not been broken up into fragments by narrow domestic walls
> Where words come out from the depth of truth
> Where tireless striving stretches its arms towards perfection
> Where the clear stream of reason has not lost its way -
> into the dreary dissert sand of dead habit
> Where the mind is led forward by thee into ever widening thought and action
> Into that heaven of freedom, my father, let my country awake.

Thank you for your patience.

Oration

Medical Education in Jaffna

Professor C. Sivagnanasundram Memorial Oration - 2010

President, Members of the council of the Jaffna Medical Association, Members of the Family of Late Professor Sivagnanasundram, Ladies and Gentleman, colleagues and Students: In commemorating a revered leader of our profession and society and a teacher and friend of most of us, I have the privilege to talk on a subject that has been the life line of Professor Sivagnanasundram and relevant to all of us.

Introduction

I can relate my association with Prof. Sivagnasundram in his own words he used to introduce me at the chairman's address in the JSA Annual Sessions: "Sivapalan was my student, colleague and now my boss". I was his student in Peradeniya, became member of staff in Jaffna and he considered all grades of staff as his colleages and he was very happy when I was elected as the Dean that was when he started to regard me as his boss even though it was very embarrassing for me but he insisted on respecting the "chair". But on academic matters he was always my teacher. For example, he instructed me to show the full script of the chairman's address well in advance which he corrected thrice and I prepared my final presentation from that script.

About Prof. Sivagnasundram

Date of Birth is 30.03.1928. He published his first short story in Weerakesari (1947) when he was 19 years old. He entered the Faculty of Medicine, Colombo in 1950 and graduated in 1955. He was awarded the nick name "Nanthi" by the president of India, Honerable C. Rajagobalachari (Rajagi) in 1956. He kept saying, "I got MBBS in 1955 and Nanthi in 1956. For me both are equal". He obtained DPH in 1968 and Ph. D. in 1971 from University of London.

After completing his internship in General Hospital, Kurunagalla in 1955, he was posted as Medical Oficer in Charge of PU Hiripitiya in 1956. In 1958 he was transferred to Lady Ridgeway Children's Hospital for 6 months and he went to Nawalapitiya as MOH in 1958. In 1961he came to GH Jaffna and in 1961he became the MOH at Jaffna Municipality. He then worked as the JMO Jaffna after 1963for two years.

Having obtained wide ranging experiences as a Medical Officer, he entered the academic world in 1965 as a lecturer at the Faculty of Medicine, Peradeniya. He went for postgraduate studies in London School of Hygiene and Tropical Medicine in 1967. On return with his PhD in 1971, he was promoted as Senior lecturer and became Associate professor in 1975.

When the Jaffna Faculty of Medicine was established in 1978, he was appointed as Professor of Community Medicine. He was elected as the Dean in January 1984 to July 1988. He was the First President University Teachers Association in 1980, President of the Faculty of Medicine Teachers Association in 1983 and the President of the Jaffna Science Association during the same year. He was elected the representative of the Senate in the University Council repeatedly for several years and the Council has been nominated for the post of Vice Chancellor thrice. He continued teaching Community Medicine and engaged in community Development programs as a Saye Devotee till his demise on 04.06.2005

K. Sivapalan
Dean, Faculty of Medicine
University of Jaffna.

He held several prestigious postings as follows

- 1972- Resource person: seminars on Human Reproduction & Population Dynamics and Family Planning in Sri Lanka
- 1977- WHO fellowship for postgraduate teaching of community medicine in Singapore, Bangkok and Calcutta
- 1980- WHO Fellowship- in Medical Education at the Liverpool school of Tropical Medicine- Teacher Training course for primary health care
- 1981- Consultancy on paramedical education in Jordan [1 year]
- 1985, 1988- WHO Consultant- Health Systems Research in Malaysia
- 1989 - WHO Consultant on Health Systems Research Zimbabwe
- Member of the board of study in community Medicine at the PGIM for several years

For Prof, there was hardly any difference between his home and the Department of Community Medicine. The Faculty was the extended family for him and teaching was the job, hobby and everything. Retirement did not take him away from the Faculty. Even when the university administration refused to give him extension of appointment he continued his most needed service to the Faculty and to the Students. When he was terminally ill, he called me to discuss certain issues and how the teaching Community Medicine should continue in his absence.

Medical Education

Even though educating is the trade of teachers, Prof. Sivagnasundram loved the word "learning". He used to say, no point of our educating if the receiver does not learn. One incident that made this idea clear to me was during the Medical Exhibition when I was a medical student. I was placed in the snake section and was writing the first aid of snake bite on a board for display. I designed the letters to be wavy and everybody in the team said it looked good and I was almost completing the work when Prof and some other teachers walked in to see how things were progressing. Prof. was attracted by my writing and praised me saying snakes in each letter. He started to read it and halfway through he said, "Siva, it is straining eyes. Everybody will see this and admire the art work but nobody will read it. Your work will be worth only if people coming to the exhibition can get the message. Erase the whole thing and write in plain letters". I wrote the whole thing again till late in the night.

What is learning? In professor's words, "it results in change of behavior". Some other definitions are,

- Learning is acquiring new knowledge, behaviors, skills, values, preferences or understanding, and may involve synthesizing different types of information. The ability to learn is possessed by humans, animals and some machines.
- Human learning may occur as part of education or personal development. It may be goal-oriented and may be aided by motivation.
- Carl Rogers said in 1983, "I want to talk about learning. But not the lifeless, sterile, futile, quickly forgotten stuff that is crammed in to the mind of the poor helpless individual tied into his seat by ironclad bonds of conformity! I am talking about LEARNING - the insatiable curiosity that drives the adolescent boy to absorb everything he can see or hear or read about gasoline engines in order to improve the efficiency and speed of his 'cruiser'. I am talking about the student who says, "I am discovering, drawing in from the outside, and making that which is drawn in a real part of me." I am talking about any learning in which the experience of the learner progresses along this line: "No, no, that's not what I want"; "Wait! This is closer to what I am interested in, what I need"; "Ah, here it is! Now I'm grasping and comprehending what I need and what I want to know!"
- Thirukkural, the ancient Tamil Literature says to learn thoroughly by clearing all doubts and act accordingly.

கற்கக் கசடறக் கற்பவை கற்றபின்
நிற்க அதற்குத் தக

Medical education is the process of developing medical professionals. It involves the students and teachers who respond to the medical needs of the

society. The society keeps changing. As a result, medical needs and awareness of the needs keep changing. As a result, medical education also must keep changing. Five ongoing transitions that influence medical needs of any society are demography, environment, epidemiology, economy and technology. The transition that changes the awareness of the medical needs is the level and nature of education in the society.

Evolution of Medicine and Medical Education

Prof insisted on learning the history of Medicine and Medical Education to understand the evolution of Modern Medicine. He persuaded me to read the book on history of medicine when I was a probationary lecturer.

The practice of medicine and teaching of medicine could be traced back to 2500 BC in ancient universities of India. Prior to 19th century, when scientific method was first applied to medicine, all medical practices were what we call now as "Traditional Medicine". Different systems developed in different parts of the world. A common feature probably was the attribute to "Karma" or committed sin and hoping to heal through prayer. Medications were considered as second option or accessory in healing. Allopathic system appears to have originated in the fifth century BC in Greece by Hippocrates. He introduced observation and reasoning. This system developed into the modern medicine.

Jaffna received Siddha system of medicine from South India, Aurvedic System from North India, Unani system with the arrival of Muslims and Homeopathy and Allopathy with the arrival of Europeans.

In the early days medical professionals were regarded as persons gifted with healing powers. The teaching was mainly by apprenticeship. The students followed the "Guru" or the Master. One became the healer when the master decided on the competency. It was a family profession in many instances. When the healer dies another member of the family became the healer. If a system of educating existed, the dominant student became the healer.

First examination for medical practitioners was held in Britain in 1551 by establishment of Royal College of Physicians. GMC was established in 1858to supervise and regulate medical education. In South East Asia medical schools were established based on British system as in the following table:

Country	year
India	1824
Indonesia	1851
Sri Lanka	1870
Thailand	1889
Myanmar	1924
Nepal	1978

The medical schools remained with British curricula until 1950s. By then it was realized that the education did not suite the population served. After that changes began taking place.

Medical Education in Sri Lanka

The medical school run by Dr. Samuel Green in Jaffna does not seem to have gone into records of history. I was happy when Dr. Rajaratnam said, in his Chief Gust Address at the inauguration of the sessions, that his close relatives were students at this medical school and Professor Naratha Varnakulasooriar of Faculty of Medicine, Srijeyawardenapura has written about this last month. Unfortunately it had to be discontinued and Colombo medical school was established in 1870. A second medical faculty was opened in Peradeniya in 1964 with modified thinking of medical teaching while Colombo remained strictly traditional at that time. Faculty of Medicine Peradeniya deviated from "teaching" of medicine and engaged in "Active Learning". Students were stimulated to ask questions and learn instead of passively sit at lectures and clinical material was introduced early, from the first year. The presentations at the Kandy Society of Medicine conducted every Tuesday at 6.00 PM in the Physiology Lecture theatre and the Clinico-Pathological Conferences held every Thursday morning at 8.00 AM in the Hospital mortuary provided immeasurable informal teaching to students. Lively and open discussion of consultants and professors in front of the students on rights and wrongs, omissions and commissions, past, present

and future of treatment plans etc. impressed on the students and provided learning experiences available nowhere else. The first Medical Education Unit in Sri Lanka was established in early seventies in the administration block of the Faculty of Medicine, Peradeniya, with a view to make medical education more appropriate and to train staff in medical teaching when Professor Varagunam returned with training in Medical Education. This made a big impact on the teachers there initially and later in all Faculties of Medicine and later development of Staff Development Centers in all Universities.

The next two faculties of medicine were established in Jaffna and Karapitiya in 1978. More medical faculties came into existence with time in Kelaniya, Srijayawardanapura, Batticaloa, and Anurathapura. Two attempts to establish private medical schools - North Colombo and North Lanka failed.

Medical Education in Jaffna

Prof. C. Sivagnnaasundram, Prof. Hoover and Dr. S. V. Parameswaran [later promoted as professor] were the founder academic staff of the Faculty of Medicine, University of Jaffna. Prof. Hoover came from Colombo Medical Faculty but the other two were from Peradeniya and exposed to the changing trends in medical education there. Almost all staff members who joined the Faculty thereafter were influenced by Peradeniya education system as students or as staff. Probationary lecturers were sent to MEU Peradeniya for two week course in Medical Education in early eithties.

The Faculty in Jaffna formed a Curriculum Committee which discussed the curricular matters. Professor Sivagnanasundram played a key role in designing the curriculum of the Jaffna Medical Faculty. The committee had been chaired by Professor Sivagnanasundram for most of the years and Prof. Parameswaran had been the chairman for the rest of the years with few others in between. The driving force of the curriculum committee had always been Prof. Sivagnanasundram as long as he was in the Faculty irrespective of whether he was the chairman or a member. I was privileged to serve as the secretary of this committee to Prof Sivagnanasundram especially when he organized a workshop in Jaffna for Faculty and Extended Faculty staff for curriculum development under the sponsorship of WHO, in 1983. That was probably the first instance of respecting the consultants in the Teaching Hospital as Teachers and involving them in Formal Educational Activity.

Superficially, the curriculum of Jaffna Medical Faculty appeared to be a traditional, subject based one, it had several modifications to make the course more relevant and meaningful. For example, physiology incorporated aspects of statistics done by the Department of community medicine and behavioral science done by Department of Psychiatry. Applied anatomy and applied physiology classes were conducted by consultants from the teaching Hospital. All examinations had clinicians as external examiners. The clinical subjects focused on problem based approach for teaching. The JMA sessions were made compulsory for final year students and topics related to patients discussed in the JMA were not redone in the ward classes or lectures. Clinico - Pathological conferences were planned and incorporated in the curriculum. AI these progressive approaches had to be abandoned or cut short because of the war situation and dearth of staff.

Public Health was a phobia for many in the seventies. One Professor told, "We don't train doctors to look down the drains" at the in-service training for Medical Officers. Peradeniya started to change this image and introduced community based teaching for medical students with Hinthagala project area and introduced the concept of Health Promotion. The community based curriculum of the Department of Community Medicine Jaffna, designed by the Professor at the inception had been maintained so far and we are planning to extend it further.

The Medical curriculum was heavily biased towards curative medicine and only one subject was there to deal with preventive medicine and Health Development of the society. Preventive health concepts are being integrated into all subjects due to untiring efforts of Prof and others.

Medical Curriculum

Curriculum is an educational policy. Such a policy can be enshrined in a document, and / or be a process enacted in practice. As a written document it is an

attempt to communicate the essential principles and features of an educational proposal in such a form that it is open to critical scrutiny and capable of effective translation into practice. This amounts to far more than simple choices about what doctors should learn and how they should learn it. The term refers to all the activities, all the experiences and all the learning opportunities for which an institution or a teacher takes responsibility either deliberately or by default. This includes: the formal and informal, the overt and the covert, the recognized and overlooked, intentional and unintentional learning opportunities.

Educationists must be mindful of the following risks in developing a curriculum:

- The temptation to ignore the fundamental role of values which underlie all curriculum design and development issues, which are the driving force of sound education.
- Temptation to invent simplified version rather than do justice to realities.
- The creeping assault of management discourse in reference to matters educational- like assessment as management tool rather than a part of educational practice.
- The assumption that components of practice will 'cash out' into simple and visible behavior and design curriculum around visible elements of practice at the expense of invisible ones like professional judgment.
- The view that quality can be couched in quantitative terms.

The Jaffna Faculty of Medicine became the first Faculty in the island to possess a documented curriculum by 1985-86 because of the efforts of Prof. Sivagnanasundram and the cooperation of the Heads of Departments. The characteristics of the medical graduate of the Jaffna Medical Faculty had been defined in that document long before the concept of quality assurance came up and the concept of 'Benchmark' for each course was thought about. A Subject Benchmark Statement in Medicine was developed by representatives from all faculties of medicine and published by the Committee of Vice Chancellors and Directors in 2004.

The curriculum of our Faculty has been subjected to a major revision under QEF grant of the IRQUE project in 2009. The Faculty is now committed to develop Integrated Spiral Curriculum and adopt SPICES model of teaching in the years to come. The SPICES stands for student centered, problem based, integrated, community based, Electives and systematic learning activities. Even though this comes as a modern concept in Medical Education, elements of all these had been in operation in our Faculty through the efforts of Prof. Sivagnanasundram and others. His Dr. V. T. Pasupathy Memorial Lecture in 1987 on "teaching and learning medicine: ideas from eleven British medical schools" remains a monument of his critical thinking on medical curriculum.

At the moment we are synchronizing the subjects with additional necessary components incorporated. One major inclusion is the Personnel Professional Development Stream. Another area to be introduced into the curriculum is the subject of Family Medicine to keep with the changing trends in the country. At the moment it is to be a part of community medicine.

Student Research

Many health problems are not investigated scientifically because the doctor trained through the traditional curriculum does not know research methodology and the ad hog investigation carried out by such doctors may have errors and biases. The WHO suggested in 1988, "students should be trained in HSR; the degree and the depth of training could vary from introduction to the concept of HSR, teaching of HSR methodologies, decisions of HSR, results and involvement of students in HSR activities". Professor Sivagnanasundram incorporated student research project into the curriculum of Community Medicine in 1990. To facilitate this, Prof has written a book on "Learning Research" which has become very popular among students and teachers of all Medical Faculties. Laboratory research was introduced at pre-clinical level through biochemistry by Prof. Balasubramaniam

Reproductive Health

Prof was very worried about the inadequacy or even negligence of reproductive health in the curriculum. Topics such as sexuality, parenthood, adolescent sexuality are not openly discussed in our cultural background. Prof was instrumental in procuring books in these fields for our library. He used to say that we know the least about the most important

aspects of healthy life. Considerable number of student research has been in this field and he was very happy when I talked on reproductive health in JSA. We need to work more in this area to bring about individual, family and social health.

Challenges in developing Medical Curriculum

At the moment the most important question in developing the curriculum is to decide on the course content of each section that could be justified in terms of future usefulness. For example, the needs of a graduate to work in periphery at the primary health care level or to go into Family practice and the needs of graduates who would pursue post graduate studies in all possible medical specialties would vary widely. The specialties span out in wide ranging fields in curative and preventive perspectives. The present curriculum of all medical schools incorporating all needed aspects comprises more than 300 credits where as the standard load for university education in Sri Lanka is 30 credits per year. If we allow students to choose the material that could be relevant for their future prospects, this load could be reduced but we will have to design suitable examination procedure.

As the immediate goal is to prepare the students for work as an intern medical officer in a tertiary hospital, the preventive and social aspects taught at the Faculty are neglected by students. We can't change the final examination as we like it because of the need to prepare a common merit list and the Faculties of medicine are to move towards a common examination.

Para- Medical Personnel and Education

When the concept of "Health" was developed, Preventive Medicine became a necessity in medical education. Another concept that challenged the traditional medical curriculum was the 'Primary Health Care'. These two concepts had been the "mantras" chanted by Prof. Sivagnanasundram every now and then. The historic International conference on Primary Health Care held in Alma Ata in 1978, affirmed the principle of 'Health for all 2000' and declared that the primary health care approach was the means of achieving health for all. One of the prime requirements to achieve the goal of health for all was the deployment of appropriately trained health personnel of the right quality and in the right numbers within the health care system and this was considered by many to be difficult while the doctors were trained in the traditional way.

As mentioned at the beginning, medical education is the process of developing medical professionals. In ancient times the healer or the doctor was the one and only category of medical personnel. As medicine developed scientifically and the understanding of illnesses and their management widened, many categories of personnel came into the scene. But they were regarded merely as assistants to carry out the instructions of doctors.

Prof Sivagnanasundram played a key role in converting the Public Health Midwife who was attending only to maternal and child health into a Family Health Worker to provide care with respect to care of ante-natal, post-natal, natal, infants, preschool children, family planning, and chronically or acutely ill at elementary level and to health educate people at all levels.

Professor Sivagnanasundram wanted to educate the paramedical personnel and wanted to regard them as members of a health team. He has participated in several international seminars and received several WHO fellowships related to Community Medicine and Medical Education. They included education of paramedical personnel as well. In 1981 he had been appointed as consultant to the Ministry of Health in Jordan on training paramedical personnel. He was very keen on developing a complete facility for paramedical education in Jaffna. He tried different techniques to achieve this. He got the paramedical school incorporated in the corporate plan of the University of Jaffna. He had another idea to initiate courses in paramedical sciences in the Faculty of Medicine. As a key to this ambition, he got this idea into the institutional objectives of the Faculty of Medicine. The objective number 4 is: to provide training programs for supporting staffs in the health care system.

The need for educating health professionals at the Degree level has generally became accepted as indicated by the newer Faculties of Medicine named them as "Faculty of Medical Sciences" and made provision to admit students for degrees in allied health

sciences. But the established Faculties could not change their image of being Faculties of Medicine conducting the MBBS course only. Prof.Sivagnanasundram has at times proposed the change of our Faculty into a Faculty of Medical Sciences but the situation here and shortage of staff prevented progress in that direction. Prof. Sivagnanasundram had been contributing to and also helping in the implementation of the change in the focus of the WHO from developing the staff for the new medical schools in the 1960s and 1970s to Reorientation of Medical Education and later to development of Health Manpower.

Para-Medical Courses in Jaffna

In mid eighties, there was a severe need for supporting staff in the hospitals of North and East and attempts were made to establish private paramedical training centre with the support of Jaffna cooperative stores. A series of meetings were held in the Faculty and given up after selecting the students at the last minute due to worsening war situation.

The department of Community Medicine organized a training program for PHI around 1998 with the approval of the Ministry of Health and the Public Health Inspectors produced by that program proved to be very efficient and effective health workers. Now the RDHS is conducting the training program for PHIs with the help of the staffs in the Faculty.

The Faculty organized a Health Studies Unit in the Department of Community Medicine and conducted certificate course in Pharmacy, diploma in Community Health, Diploma in Physiotherapy and Diploma in Medical Laboratory Sciences with a view to meet the health needs of the area making use of the facility given by UGC to start self funded courses. One batch of students completed the course. The HSU faced problems of shortage of staff and delay in approving the curriculum by the Ceylon Medical Council for various reasons. This happened to be the first instance of giving university degree or certificate for courses in paramedical sciences in Sri Lanka. This has stirred the health system and initially all universities were stimulated to commence similar diploma courses. Soon trade unions and the Ministry of Health requested the UGC to commence degree courses in Allied Health Sciences and we have utilized the opportunity to commence degree courses in Nursing, Pharmacy and Medical Laboratory Sciences. More degree courses are to be commenced. The degree courses have increased the work load tremendously and as a result, the Faculty has temporarily halted admitting students for self funded diploma courses. It is now expected that the Allied Health Sciences will grow into a Faculty of their own leaving Faculty of Medicine to deal with MBBS course only.

However, the medical curriculum should train medical students to understand the role of other health professionals in providing health care and to regard them as members of the health team. Professor Sivagnanasundram was keen to develop this aspect and as a first step he introduced 2 weeks of clinical clerkship in nursing. Even though it was a good move, it had to be abandoned because of shortage of Nursing Tutors to do this appointment.

AMP course

In early eighties, the Government of Sri Lanka introduced training of AMPs to meet the health needs of the periphery and Prof. Sivagnanasundram took charge of the Jaffna centre and acted as the coordinator for the program which served to satisfy the burning needs of North and East of the country at that time.

Other systems of Medicine

Another area of interest was other systems of Medicine. Prof. Sivagnanasundram used to keep saying that the best in all systems should be useful to the society and there must be mutual reference of patients between the practitioners of all systems of medicine especially with Siddha medicine. He wished that medical students should learn about these systems at some point. Further, he has helped the development of the Unit of Siddha Medicine, Jaffna in all possible ways he could. His expectation was what is regarded as Tamil system of medicine, namely the Siddha Medicine develop and flourish in our area.

Public Education and Social Reforms

For Professor Sivagnanasundram, medical education did not end with educating the Health Professionals. His main concern has been raising the health

awareness of the society. He, as a youngster, could not tolerate the misconceptions and malpractices in the society and took his pen as a means of social reform. He has authored several short stories published in three collections, three novels, health education materials in the form of letters and books for human resource development.

Summary of books:

- அருமைத் தங்கைக்கு (தாயாகப்போகும் தங்கைக்கு பிரபல டாக்டர் எழுதிய கடிதங்கள்) 1960
- அன்புள்ள நந்தினி (குழந்தை வளர்ப்பு பற்றிய கடிதங்கள்) 1973
- உங்களைப் பற்றி- சிறுவர் அறிவு நூல் 1973
- தம்பி தங்கைக்கு- இளைஞர் அறிவு நூல் 1996
- மலைக்கொழுந்து- நாவல்- 1964 (சமூக சீர்திருத்தம்)
- குரங்குகள்- நாடகம் 1975
- தங்கச்சியம்மா- 1977 (மருத்துவ மாது மற்றும் கொலரா கட்டுப்படுத்தல்)
- நம்பிக்கைகள்- நாவல்- 1989 (மருத்துவ மாணவரின தும் வைத்தியர்களினதும் எதிர்பார்ப்புகளும் நடைமுறைச் சிக்கல்களும் அவர்களைப்பற்றிய சமூக நம்பிக்கைகளும்)
- ஊர் நம்புமா 1966-1947 முதல் எழுதியவை
- கண்களுக்கு அப்பால்- சிறுகதைத் தொகுப்பு 1984
- நந்தியின் கதைகள் 1994
- தரிசனம்- சிறுகதைத் தொகுப்பு 2002
- அன்பார்ந்த சாயி அடியார்களுக்கு 1992
- சாயியின் குரலும் வள்ளுவர் குறளும்- மனித மேம்பாட்டுக் கல்வி நூல் 2000
- சத்திய சாயி மனித மேம்பாட்டுக் கல்வி- பாடசாலை ஆசிரியருக்கான கை நூல் 2003

Public Education in Medical Curriculum

The Department of Community Medicine introduced the Field Health Work for students to gain experience in the same way as that of the Professor.

Conclusion

Prof has lived and his commitments are living as example of "Preach what you Practice" "சொல்லியவண்ணம் செயல்". He has laid a very strong foundation for good, complete and successful Medical Education in Jaffna. He enjoyed good cooperation from all around him. It is up to all of us to build on his foundation.

References

1. Teaching and learning medicine: ideas from eleven British medical schools. (1987) By C. Sivagnanasundram. Dr. V. T. Pasupathy Memorial Lecture. Jaffna Medical Journal, Vol. XXII Nos.1 & 2.
2. Development of a system of health care for rural Sri Lanka - a new role for the Public Health Midwife as a Family Health worker. C. Sivagnanasundram, (1976). S.L.A.A.S. mini sessions towards Healthier village.
3. Need for reforms in the present system of education- medicine. C. SivagnanasundramUnpublished.
4. Medical education in south-east Asia. (1996) Ed. By P. Abeykoon and N. Mattock, published by WHO
5. Medical education - developing a curriculum for practice. Published by- open university press [England]. (2005).
6. Essentials of Medical Education. (1996) Ed. By, R. K. Adhikari and P. T. Jayawickramarajah. Published by Health Learning Centre, (WHO colaborationg centre), Institute of Medicine, Tribhuvan University, Kathmandu, Nepal.
7. Medical Teacher, vol. 30, no. 1, February 2008.
8. Memories of our late Professor C. Sivagnanasundram, 2005. Published by Medical Students Union, Faculty of Medicine, University of Jaffna.

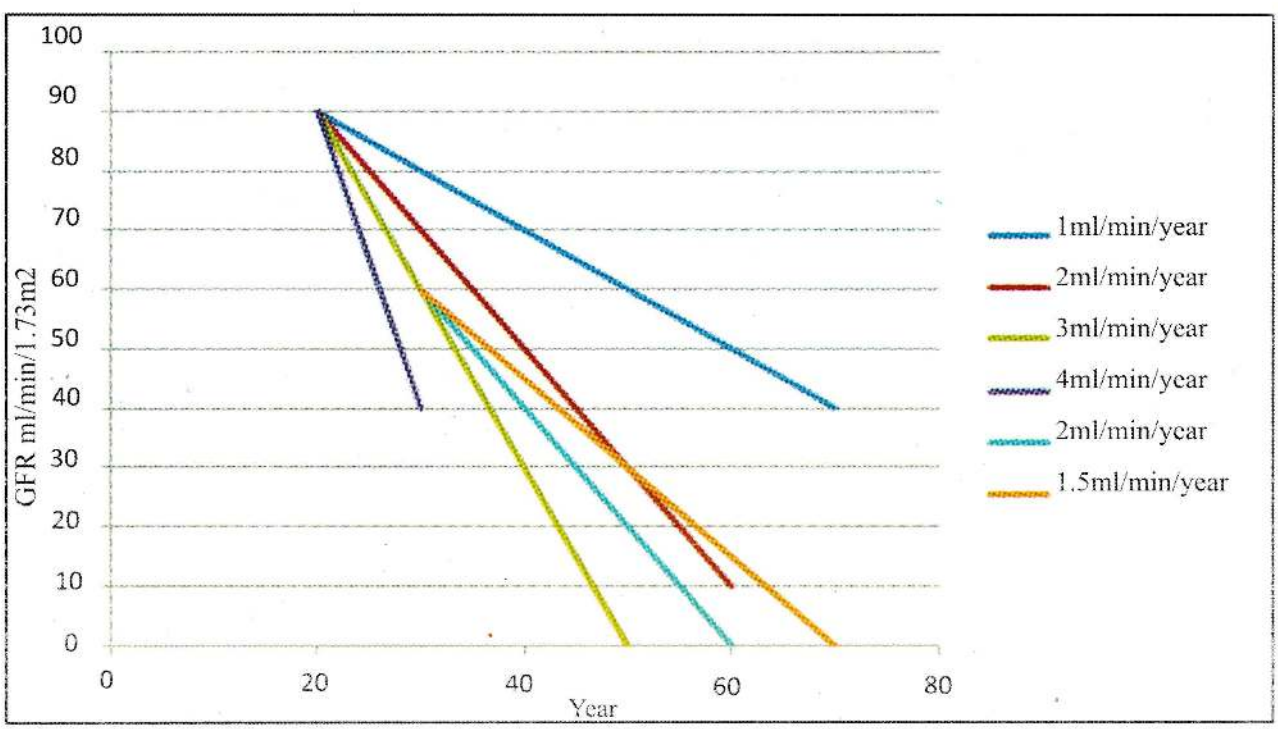
Review

Renoprotection : Emphasis on controlling proteinuria

Renal disease that progresses to end-stage kidney disease (ESKD) imposes a great burden on the affected individual, family and on society. Thus, there is a great need to identify therapies that arrest the progression mechanisms common to all forms of renal disease. Progress is being made.

Hypertension and proteinuria are the most important independent risk factors for renal disease progression. Therefore, current therapeutic strategies to prevent progression aim at controlling blood pressure and reducing urinary protein excretion. Renin-Angiotensin-Aldosterone-System (RAAS) inhibition therapy to slow the "progression of renal disease" is the corner stone of the treatment strategy. RAAS antagonists preserve kidney function not only by lowering blood pressure but also by their antiproteinuric, antifibrotic, and anti-inflammatory properties. Nevertheless, this therapy alone rarely stops renal disease progression. A multiple-risk-factor intervention strategy based on inhibiting the progression mechanisms believed to be common to most forms of progressive renal disease needs to be applied on each individual with CKD. Renal disease generally progresses slowly [Glomerular filtration rate (GFR) loss of about 3 mL/min/year (01). Thus even small improvements in slowing renal disease progression can provide large benefits (figure 1).

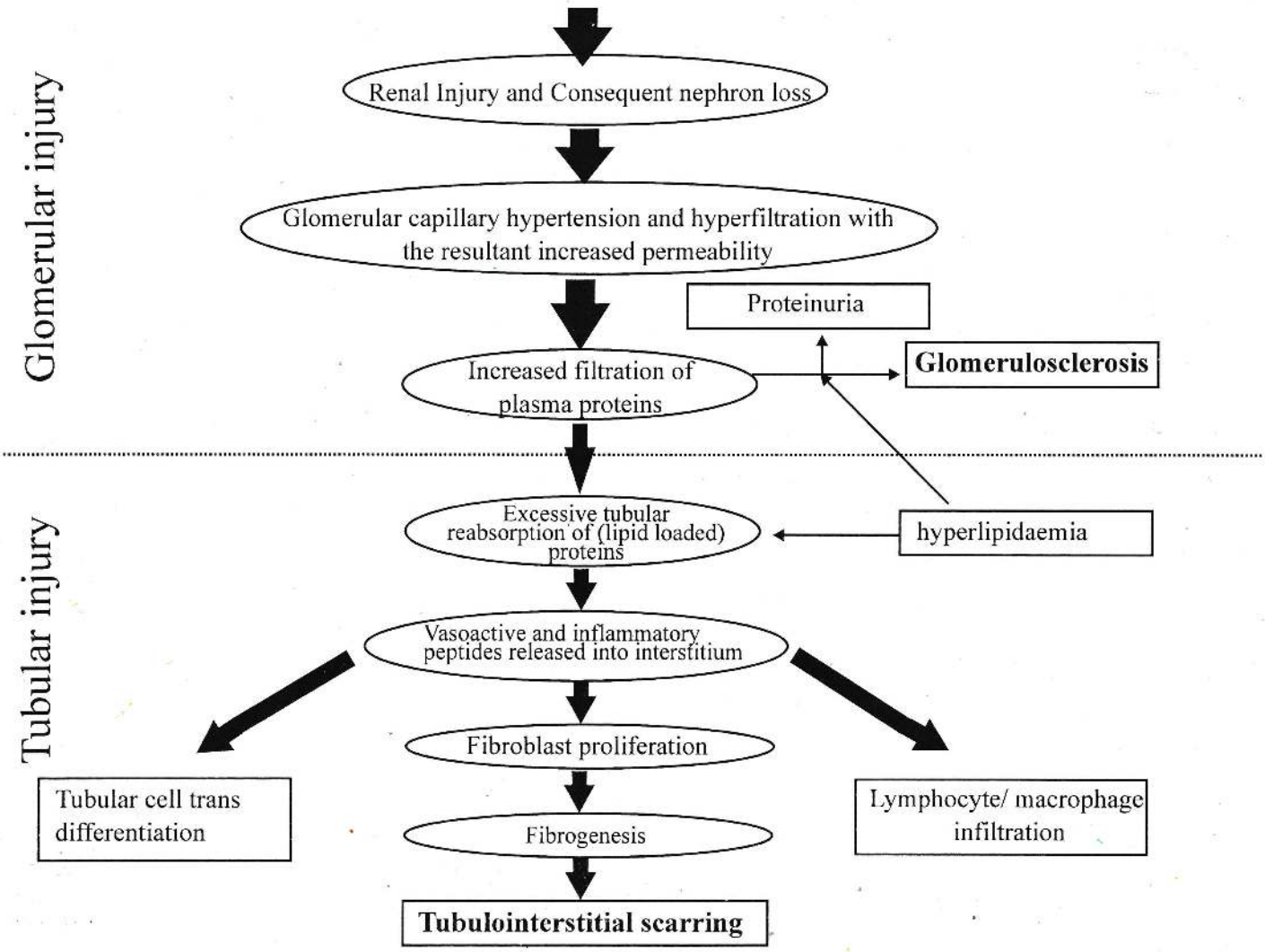
Figure 1. Rate of GFR decline in hypothetical patients with onset of progressive renal disease at the age of 20 years. Effects seen subsequent to renoprotective measures commencing at the age of 30 years, in those with an initial GFR decline of 3 ml/min/year.



S.Mathu
Consultant Nephrologist
Teaching Hospital Anuradhapura

Understanding the pathophysiological basis of progressive renal dysfunction is important to recognize the renoprotective treatment strategies (figure 2). Three important hypotheses are relevant. Firstly, Brenner and co-workers postulated that loss of nephrons as result of renal injury can elicit a vicious circle of progressive renal function decline (02). They hypothesized that after loss of functioning nephrons, the remaining nephrons demonstrate a compensatory response aimed at preserving the GFR, at the expense of glomerular hypertension that eventually leads to glomerular capillary damage, glomerular protein leakage, and finally glomerulosclerosis, and consequent further nephron loss. The fact that antihypertensive treatment exerts a beneficial effect on renal function is, according to this hypothesis, due to the reduction of glomerular pressure that results from the systemic blood pressure lowering, and/or specific reduction of glomerular pressure by post-glomerular vasodilation, as occurs during blockade of the RAAS. Secondly, Remuzzi and co-workers demonstrated that proteinuria is not merely a marker of severity of glomerular injury but also a direct tubular toxic agent and plays a key role itself in progressive renal function loss (03,04). They demonstrated that protein that leaks into the tubular lumen exerts tubulotoxic effects leading to renal scarring, and that antiproteinuric treatment can prevent these sequelae (03). Accordingly, they postulated that proteinuria reduction is pivotal for renoprotection, as subsequently supported by the results of the REIN trial and several subsequent studies. Thirdly, the notion that high blood pressure is not merely a symptom of renal failure but also an important risk factor in the progression of renal function decline was a crucial step forward in renoprotective treatment (05).

Figure 2 Pathophysiological basis of progression of renal dysfunction



Blood pressure(BP) is an important risk factor for renal function loss. In the MRFIT study (06) blood pressure was a strong predictor for the development of end-stage renal failure during 16 years of follow-up in middle-aged men. The study identified a strong graded relation between both systolic and diastolic blood pressure and end-stage renal disease. Several other studies pointed out that a more aggressive blood pressure control is beneficial to the course of renal function loss in renal patients. Bakris *et al.* performed a meta-analysis of long-term clinical trials directed to blood pressure reduction in both diabetic and non-diabetic patients (07).

This analysis showed a linear relationship between the obtained blood pressure and the rate of decline of renal function across the different studies. Thus, the available evidence indicates that blood pressure reduction exerts a beneficial effect on the decline of renal function.

Proteinuria is a major risk factor for both diabetic and nondiabetic renal disease progression (08,09,10,11). Nonselective proteinuria contains numerous toxic/inflammatory systems that promote the progression of renal disease (12, 13,14,03, 15). Protein filtered by glomerulus is reabsorbed by proximal tubular cells. This induces a proliferation of proximal tubular cells accompanied by an increased synthesis of many vasoactive and proinflammatory substances. The appearance of interstitial cellular infiltrates, a well-known finding in proteinuric diseases, precedes progressive tubulointerstitial fibrosis (16).

In chronic kidney diseases, the greater the proteinuria the greater is the risk of progression (17,18). The Modification of Diet in Renal Disease Study (MDRD) and study (Table 1) and Ramipril efficacy in Nephropathy (REIN) study (table 2) clearly demonstrates it.

Table 1

MDRD study Baseline GFR appx 39ml/min			
Baseline proteinuria (g/d)	<1.0	1-3	>3.0
GFR decline (mml/min/y/1.73m2)	1.7+/-0.3	4.9+/-0.5	8.3+/ -0.7

Table 2

REIN study Baseline GFR appx 44mil/min				
Baseline proteinuria (g/d)	<2.0	2-3	3-4.5	>4.5
GFR decline (mml/min/y/1.73m2)	2.5=/-0.04	4.6+/ -0.1	6.5+/ -0.1	9.5+/ -0.2

The exceptions are glomerulonephritis that manifest highly selective proteinuria such as minimal change disease and certain forms of hereditary glomerulopathy (19). In these conditions, heavy proteinuria can be present for years without evidence of kidney damage.

Non immunological treatment of proteinuria

Clinical studies have shown that reduction in proteinuria is associated with GFR reduction (08,20,21) and uncontrolled proteinuria is associated with faster GFR decline (14,22). The MDRD showed that for each 1-g/d reduction in proteinuria observed at 4 month of the antiproteinuria therapies (the BP and dietary interventions), subsequent GFR decline was slowed by about 1 ml/min per year (08).The REIN study showed that for each 1-g/d reduction in proteinuria observed at 3 months of ACEI therapy, subsequent GFR decline adjusted for baseline GFR was slowed by about 2.0 ml/min per year (23). A proteinuria reduction of 1g/d prolongs the time to ESKD and may reduce cardiovascular deaths as CKD is independently related to cardiovascular deaths (24). The goal of antiproteinuric therapy is to reduce the

proteinuria to as low as possible and preferably to less than 500mg/d (25,26,84).

1) Control of hypertension

BP should be taken in the sitting position and after taking the antihypertensive medications at the usual times. However, if the antihypertensive medication has rapid onset (clonidine tablets, labetalol, captopril, hydralazine), this needs to be taken into account. At the first evaluation, BP is taken in both arms. The arm with the higher BP is used for future BP measurement (27). MDRD study (08) the Appropriate Blood Pressure Control in Diabetes (ABCD) study (28), and the African American Study of Kidney Disease and Hypertension (AASK) (22) Randomized CKD patients to two different levels of BP control, usual goal (approximately 140/85 mmHg) or lower goal (approximately 125/75 mmHg) and assessed the effects on proteinuria. The lower BP target reduced proteinuria by 50% (08) or prevented the two fold to threefold increase in proteinuria observed in the usual BP target (22,28). The lower BP was well tolerated by the participants of these trials. In addition lower BP goal reduced stroke rate (28) and left ventricular mass index (29). Benefits of the low goal have also been confirmed in hypertensive non-kidney disease patients (30,31). Thus, the low BP goal, sitting systolic BP in the 120s or less if tolerated is recommended. A target of at least 130/80 mmHg should be achieved in CKD. The systolic BP is specified because it correlates better than diastolic pressure with kidney disease progression (08,14).

Antihypertensive regimes
Non-pharmacological

Reduced salt intake
Regular exercise and loss of excess weight
Avoiding excess alcohol (i.e. more than 2 drinks a day)
Avoiding NSAID, high dose oestrogen therapy, cocaine, anabolic steroids amphetamines, and decongestants

Pharmacological

The recommended first line therapy is Angiotensin Converting Enzyme inhibitors (ACEI) or Angiotensin Receptor Blockers (ARB), not diuretics. ALLHAT study (Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack) showed that chlorthalidone reduced certain cardiovascular risks better than ACEI, dihydropyridine calcium channel blocker (DH CCB), or doxazosin (32). On this basis, diuretics are recommended as first-linc therapy in hypertension (33). However, diuretics stimulate the RAAS, which is probably undesirable in CKD. Furthermore, in ALLHAT, the main benefit of diuretics was reduction in congestive heart failure and stroke in African Americans. Both are salt-sensitive states. Thus, unless heart failure or another edema-forming state is present, the preferred initial antihypertensive/antiproteinuric therapy in CKD is ACEI or ARB because they attenuate Angiotensin II effects. This should not compromise BP control in CKD. The year 2003, joint statement of the American Society of Nephrology and the National Kidney Foundation recommend that ACEI, ARB, or both is the preferred initial therapy of hypertension in CKD. If diuretic therapy is needed in CKD, frusemide is recommended (12). Thiazide diuretics are ineffective once the GFR falls below 30ml/min.

The following sequence is recommended until the patient reaches his or her blood pressure goal: (*08*) Low-dose ACEI therapy plus dietary salt restriction; (*09*) Higher dose ACE I therapy plus dietary salt restriction; or (*10*) Higher dose ACE I therapy plus dietary salt restriction plus diuretic therapy. In Autosomal Dominant Polysystic kidney Disease (ADPKD) patients, diuretic therapy may promote cyst growth (34) and is associated with more rapid progression, compared with ACEI therapy (abstract; Ecder *et al, Am Soc of Nephrol* 10:415, 1999). Thus, avoid diuretic therapy and emphasize salt restriction in ADPKD patients.

If the blood pressure goal is not being met with this regime, their drug and dietary compliance be assessed. To estimate dietary salt intake accurately, 24-hour urine creatinine and sodium excretion should be measured. If the patient is receiving $NaHCO_3$ therapy, 24-hour urine chloride should be measured. Note that unless advanced renal insufficiency is present, $NaHCO_3$ therapy does not usually lead to sodium retention (35) if the patient is compliant and blood pressure still is not at goal, triple antihypertensive therapy is recommended.

Triple antihypertensive therapy

ACE I, diuretic, and nondihydropyridine calcium channel blocker (ND-CCB, diltiazem and verapamil).

ND CCB are antiproteinuric and may be renoprotective (12,14). Sustained release forms are recommended. Diltiazem has fewer side effects, but is considerably more expensive. The combination of an ACE inhibitor and a ND-CCB is more antiproteinuric at the same blood pressure level than either drug taken alone (36,37). Avoid DH CCBs (for example, nifedipine, amlodipine, felodipine, nicardipine) use except for the BP control. DH CCBs are excellent antihypertensive agents. However, in the ABCD trial, despite good BP control, nisoldipine resulted in threefold greater albuminuria rate compared with enalapril (38). In the IDNT, despite substantial BP reduction in the amlodipine group, proteinuria remained at baseline. By contrast, at the same BP level as the amlodipine group, the irbesartan group achieved a 33% reduction in proteinuria (39). In the AASK, although amlodipine achieved better BP control than ramipril or metoprolol, proteinuria increased about twofold on amlodipine therapy but generally remained at or below baseline levels on ramipril or metoprolol therapy (22). These results confirm previous observational studies suggesting that DH CCB are not antiproteinuric and may actually promote proteinuria and more rapid CKD progression, if strict BP control(Systolic BP<110mmHg) is not achieved. (14,83). Also, the AASK showed that, compared with ACEI and beta-blocker therapy, DH CCB increased the risk of the composite endpoint of doubling of serum creatinine, ESRD, or death, compared with ACEI or beta-blocker (14). If DH CCB is needed for BP control, concomitant use of ARB (40), ACE, combination ACEI/ARB, or beta-blocker may mitigate the vasodilatory effects of DH CCB to cause glomerular hypertension (22) and, apparently, promote proteinuria. This strategy may also limit RAAS stimulation by DH CCB. Combination NDH CCB and DH CCB is potent antihypertensive therapy (07).

ACE I, diuretic, -blocker. This combination is appropriate for patients with coronary artery disease. -Blockers can slow recovery from insulin-induced hypoglycemia but are not contraindicated in diabetes if needed for blood pressure control. In patients prone to diabetes, -blocker therapy appears to increase (by 28%) the risk of developing diabetes (41). This should be taken into account when selecting antihypertensive therapies. The AASK showed that sustained-release metoprolol had antiproteinuric effects nearly equal that of ramipril and better than that of amlodipine (22).

ACE I, diuretic, clonidine. This combination is recommended for individuals receiving insulin (clonidine does not importantly affect glucoregulation) and for those who may have difficulty with -blocker therapy (bronchospasm, cardiac conduction). Clonidine tablets are inexpensive but must be taken at least twice daily. The clonidine patch is convenient but more expensive.

ACE I, diuretic, -1 blocker. This combination is generally well tolerated and effective. The -1 blockers ameliorate the symptoms of prostatism. The ALLHAT study discontinued the arm that compared the -1 blocker doxazocin 1 to 8 mg daily to chlorthalidone 25 mg daily as monotherapy to control hypertension. They found a 25% greater incidence of hospitalization for congestive heart failure in those randomized to doxazocin. The congestive heart failure seen in the doxazosin-treated ALLHAT patients may have been the result of inadequate diuretic therapy(12).

If triple therapy does not achieve the blood pressure goal, the patient should be re-evaluated for drug and dietary compliance. If that evaluation is negative, the patient should be studied by ambulatory blood pressure monitoring to determine whether sustained hypertension is present. If sustained hypertension is documented by ABPM, secondary causes of hypertension should be sought, particularly renal artery stenosis.

If the evaluation for secondary causes of hypertension is negative, more intensive triple therapy is recommended. The initial step is usually an increase in diuretic therapy. A further increase in ACE I therapy is not recommended to achieve the blood pressure goal. Rather, high-dose ACE I therapy should be reserved for those who have achieved their blood pressure goal and dietary goals (for salt and protein) but have not achieved their proteinuria goal.

If more intense triple therapy does not achieve the blood pressure goal, additional antihypertensive therapy is recommended.

Multiple antihypertensive therapy

ACE I, diuretic, -blocker, and DH-CCB. This combination is usually highly effective in controlling blood pressure. The DH-CCBs are more effective antihypertensive agents than the ND-CCBs, but may blunt the renal protection provided by ACE I therapy. Thus, D-CCBs are recommended only if essential to achieving the blood pressure goal.

ACE I, diuretic, -blocker, and minoxidil. Minoxidil may be unacceptable in women because it causes generalized hair growth. Minoxidil may worsen proteinuria, similar to D-CCB. Thus, minoxidil is recommended only if needed to achieve the blood pressure goal.

ACE I, diuretic, -blocker, and -1 blocker. The addition of a DH-CCB or minoxidil will increase the effectiveness of this combination.

ACE I, diuretic, -blocker, and clonidine. The combination of a -blocker and clonidine can induce bradycardia but usually is well tolerated. The addition of a DH-CCB or minoxidil will increase the effectiveness of this combination.

1) RAAS blockade

There is now adequate evidence based on prospective, randomized, and controlled clinical trials that ACE I is renoprotective, independent of its antihypertensive effects, in both diabetic and nondiabetic nephropathies (13,42). By ameliorating size selectivity of the glomerular capillary barrier, ACE inhibitors and ARBs reduce proteinuria, which leads to renoprotection (43).

However, RAAS inhibition does not confer additional renoprotection over non RAAS-inhibiting antihypertensive therapy in patients with hypertension, diabetes or increased cardiovascular risk, but without proteinuria (44). ACEI, rather than ARB, is the initial choice because, although both ACEI (12,13,25,45) and ARB (40,39, 46) are antiproteinuric and renal protective, it is unclear whether ARB are cardioprotective to the level of ACEI(84). The HOPE trial ($n > 9000$ patients) showed that ramipril significantly reduced the composite endpoint of death, stroke, and myocardial infarction, and each component of the composite endpoint (47). In the LIFE trial ($n > 9000$ patients), the composite endpoint of death, stroke, and myocardial infarction was reduced significantly by losartan but of the individual components of the composite endpoint, only stroke was significantly reduced (48). Furthermore, in the OPTIMAAL trial ($n = 5477$ patients), which compared losartan to captopril in patients with acute myocardial infarction and heart failure (49), captopril was numerically better than losartan in reducing death, the primary endpoint ($P = 0.069$), and in reducing each of the twelve secondary cardiac and non-cardiac endpoints, although statistical significance was reached only for cardiovascular death, $P = 0.032$. In the captopril trial (50), the combined endpoint of death or ESRD was reduced significantly. By contrast, such benefit was not observed in RENAAL or the IDNT study (40, 39, 51). Theoretical advantages of ACEI over ARB include the increased bradykinin levels during ACEI therapy, which can be additionally vasodilatory and antifibrotic (52). Both ACEI and ARB appear to reduce the rate of new-onset diabetes mellitus: ACEI, 34% reduction in HOPE; ARB, 25% reduction in LIFE. Importantly in a resource limited setting generic ACEi are cheaper than ARBs. Some suggest that the lower rates of new-onset diabetes in the ACEI or ARB groups in these trials are not truly protection against diabetes but rather reflect induction of diabetes by the beta-blocker or diuretic therapy of the control group. The ALLHAT results, however, suggest true protection against diabetes by ACEI because the chlorthalidone group had more new cases of diabetes mellitus and the ACEI group had fewer new cases of diabetes mellitus than the group receiving DH CCB, which does not affect glucose metabolism.

ARBs are recommended in ACEI-intolerant patients (cough, angioedema, or allergy).

ACE I therapy is renoprotective even in those with substantially impaired kidney function (serum creatinine 2.5 mg/dL) (12, 50, 53). Thus, impaired kidney function is not a contraindication to ACE I therapy; however, greater caution is advised.

If hyperkalemia develops on ACE I therapy, it is important to determine whether the hyperkalemia occurred despite a restricted K^+ intake. A 24-hour urine for creatinine and potassium best assesses this question. If the 24-hour urine for K^+ exceeds 50 mEq/24 hours, a reduction in potassium intake should prevent serious hyperkalemia. However, if the hyperkalemia occurred when a 24-hour urine for K^+ was less than 40 mEq/24 hours, it is unlikely that hyperkalemia can be avoided by dietary measures alone. In that circumstance, an increase in diuretic therapy (if the blood pressure is above goal) or sodium bicarbonate therapy (if plasma bicarbonate is less than normal) may control the hyperkalemia (12). If the hyperkalemia persists despite these measures, it is best to stop the ACE I. ARB therapy should then be considered because of its lesser tendency to raise serum K^+ (12,54).

Usually serum creatinine increases slightly with ACE I therapy (for example, increases of 0.2 mg/dL for patients with serum creatinine near 2 mg/dL,) (53). If it is a stable increase in serum creatinine, there is no need to discontinue the ACE I therapy (12,22). Serum creatinine increases of up to 50% can occur with ACEI therapy. However, awareness to possible renal artery stenosis is indicated.

ACEI therapy reduces proteinuria by about one third (25,45). At maximum recommended doses, ACEI may be more antiproteinuric than ARB (55). Concomitant use of low-salt and reduced-protein diet enhances ACEI antiproteinuria (56) and slow GFR decline (12,57). Diuretic therapy (58) and statin therapy (59) also potentiates effects of ACI. The effectiveness of ACEI to slow CKD progression is greater with greater proteinuria (25). ACEI are antiproteinuric and renoprotective even in inflammatory glomerulopathies (60).

Some, but not all, studies show that doses of ACE inhibitors or ARBs above the maximum recommended doses achieve a greater reduction in proteinuria than do conventional doses in patients with chronic kidney disease both those with diabetes and those without diabetes (61). In the Renoprotection of Optimal Antiproteinuric Doses (ROAD) trial,(62) a strategy of increasing the dose of benazepril or losartan to achieve optimal antiproteinuric effects, as compared with conventional dosing, reduced the incidence of the doubling of serum creatinine, end-stage renal disease, or death over the course of a median 3.7-year follow-up. In other studies, high doses of lisinopril (80 mg per day)(63) or candesartan (128 mg per day)(64) have been shown to reduce proteinuria more than the standard doses.

Combination of ACEI and ARB

Evidence suggests that more effective blockade of the reninangiotensin system, rather than simply more effective blood-pressure control, confers maximal renoprotection.(65) Combination ACEI/ARB therapy is more antiproteinuric than ACEI or ARB alone (55,66,61,67). The optimum antiproteinuric strategy appears to be addition of ARB to maximum ACEI in those who fail to achieve their proteinuria goal on ACEI alone (55). The theoretical benefits of combination therapy include those of ACEI therapy (increased bradykinin, decreased aldosterone, decreased AngII levels) and those of ARB therapy (blockade of AngII produced by chymase, and increased AT2 receptor activation, which may be vasodilatory, antiproliferative, and antifibrotic).

ONTARGET study showed that dual RAAS blockade prevents microalbuminuria but facilitates transient renal function impairment in nonproteinuric patients with atherosclerotic vascular disease or diabetes. These findings should not be used as an excuse not to optimize RAAS inhibition and target urinary protein in patients with proteinuric nephropathies (68, 69).

Randomized trials are lacking to compare treatment with an ACEI or ARB at doses that are higher than the maximum recommended doses with treatment that consists of a combination of these two agents at conventional doses. Ongoing randomized trials (VALID and VANEPHRON-D, Clinical-trials.gov registry numbers NCT00494715 and NCT0555217, respectively) will prospectively address whether dual RAAS blockade results in more efficient renoprotection than single-drug RAAS inhibition.

Individualizing therapy

Ruggenenti *et al.* showed improved long-term outcomes with an individual titration regimen in 56 patients in their 'Remission Clinic' (70). The regime consisted of ramipril 510 mg/day, losartan 50100 mg/day, verapamil 80120 g/day and atorvastatin 1020 mg/day in successive steps, aiming for a low blood pressure target of less than120/80 mmHg and a proteinuria target of less than 0.3 g/day in patients who had proteinuria greater than 3 g/day despite treatment with ACIs. Median decline in estimated GFR was significantly slower among patients treated according to the Remission Clinic protocol than in a matched historical reference group. Only 3.6% of Remission Clinic patients reached end-stage renal disease, compared with 30.4% of the historical controls.

Following are other measures which can reduce proteinuria.

2) Controlled protein intake

In CKD with proteinuria, reducing protein intake from usual levels (about 1.0 to 1.5 g/kg ideal body weight per day) to about 0.7 g/kg ideal body weight per day decreases proteinuria about 50% (12) even with nephrotic-range proteinuria (71). Reduced protein intake lowers fibrinogen levels (71) possibly reducing cardiovascular risk (12) and inhibits glomerular

hypertrophy (72) possibly contributing to renoprotection (12). In patients with proteinuria < 250 mg/d, the low-protein diet does not reduce proteinuria; however, it does slow the progression from minor to major proteinuria, which is a strong risk factor for CKD progression (12). Thus, the low-protein diet is recommended even in low-level proteinuria (126). Substituting soy proteins for animal proteins is antiproteinuric and inhibits glomerulosclerosis (73,74). Soy proteins are high in antioxidants (isoflavones) and L-arginine, a nitric oxide donor, which may be renoprotective. A reduced protein diet is recommended in both diabetic and nondiabetic CKD (12). Dietary advice should be tailored to each individual to avoid malnutrition(75).To monitor dietary protein intake, measurement of 24-h urine urea excretion is recommended (12).

3) Salt control

The antiproteinuric effects of ACEI or NDH CCB is over ridden by high salt intake (e.g., 200 mmol NaCl/d or 4.6 g sodium/d)(12). Counseling the patient in dietary salt restriction is usually necessary. An arbitrary goal of 80-120 mmol NaCl/day is recommended (This is based on recommendation for BP control). However hyponatraemia due to salt loosing nephropathy is not uncommon in chronic interstitial nephritis which is seen in certain region of Sri lanka, in which case salt restriction is not advisable.

4) Cholesterol control

Clinical trials show an antiproteinuric effect of lipid-lowering therapy, especially statins (59,76). The mechanisms may include decreasing oxidative stress and prevention of lipid-induced podocyte damage from decreased nitric oxide production. Combining ACEI with statin may further reduce proteinuria (77).

5) Aldosterone inhibition

In CKD, 25 mg/d spironolactone added to ACEI therapy for 4 wk reduced mean proteinuria from 3.8 g/d to 1.8 g/d (78), perhaps by blocking the profibrotic effects of aldesterone. Combination spironolactone and ACEI therapy can cause serious hyperkalemia.

6) Smoking cessation

Cigarette smoking is associated with proteinuria and faster progression of CKD of all types (79,80)

7) Obesity reduction

Obesity is associated with glomerulomegaly, focal and segmental glomerulosclerosis (FSGS), and proteinuria that can be progressive (81). Reducing obesity can reduce proteinuria (81) but may not affect progression of primary glomerulopathies (82).

Any patient with a CKD is a candidate for antiproteinuric therapy, even in those with low-level proteinuria. CKD with low-level proteinuria generally manifests slow GFR decline, but progression of proteinuria during follow-up is the rule. Thus, those with low level proteinuria and slow GFR decline tend to become those with greater proteinuria and greater GFR decline. The ability of antiproteinuric therapy to slow GFR decline has been documented at proteinuria levels starting as low as 500 mg/d (25).

Chronic Kidney Diseases for which Antiproteinuric Therapies Usually Are Not Indicated Aggressive use of kidney protective therapies is not recommended in CKD at low risk for ESRD (12). These include steroid-responsive minimal change disease, a solitary kidney that is normal and acquired in adulthood (e.g., a kidney donor), hereditary nephritis, or thin GBM disease in the normotensive adult whose only renal manifestation is microscopic hematuria, and in elderly patients with idiopathic and moderately elevated serum creatinine (1.3 to 2.0 mg/dl) and minor proteinuria (<1 g/d) that have been stable for at least 1 yr(84).

In conclusion, hypertension and proteinuria are key players in renal disease progression. Therapeutic strategies to prevent progression should comprise blood pressure control and lowering of proteinuria. RAAS antagonists preserve kidney function, not only by lowering blood pressure but also through antiproteinuric and antifibrotic properties.

The patient and nephrologist share a dream, "No more dialysis", the march towards it will continue, until it's reached.

References

01. Klahr S, Levey A & Beck J et al. The effects of dietary protein restriction and blood-pressure control on the progression of chronic renal disease. N Engl J Med 1994; 330: 877884
02. Brenner BM, Meyer TW, Hostetter TH. Dietary protein intake and the progressive nature of kidney disease: the role of hemodynamically mediated glomerular injury in the pathogenesis of progressive glomerular sclerosis in aging, renal ablation, and intrinsic renal disease. N Engl J Med 1982;307:652-9
03. Abbate M, Zoja C, Rottoli D et al. Antiproteinuric therapy while preventing the abnormal protein traffic in proximal tubule abrogates proteinuria. J Am Soc Nephrol 1999;10:804-13
04. Remuzzi G, Bertani T. Pathophysiology of progressive nephropathies. N Engl J Med 1998;339:1448-56
05. Alvestrand A, Gutierrez A, Bucht H, Bergstrom J. Reduction of blood pressure retards the progression of chronic renal failure in man. Nephrol Dial Transplant 1988;3:624-31
06. Klag MJ, Whelton PK, Randall BL et al. Blood pressure and end-stage renal disease in men. N Engl J Med 1996;334:13-8
07. Bakris GL, Williams M, Dworkin L, Elliott WJ, Epstein M, Toto R, Tuttle K, Douglas J, Hsueh W, Sowers J: Preserving renal function in adults with hypertension and diabetes: a consensus approach. National Kidney Foundation Hypertension and Diabetes Executive Committees Working Group. PG - 64661. *Am J Kidney Dis* 36: 2000
08. Peterson JC, Adler S, Burkart JM, Greene T, Hebert LA,Hunsicker LG, King AJ, Klahr S, Massry SG, Seifter JL:Blood pressure control, proteinuria, and the progression ofrenal disease. The Modification of Diet in Renal Disease Study. *Ann Intern Med* 123: 754762, 1995
09. de Zeeuw D, Remuzzi G, Parving HH, Keane WF, ZhangZ, Shahinfar S, Snappin S, Cooper ME, Mitch WE, BrennerBM: Proteinuria, a target for renoprotection in patientswith type 2 diabetic nephropathy: Lessons from RENAAL.*Kidney Int* 65: 23092320, 2004
10. Atkins RC, Briganti EM, Lewis JB, Hunsicker LG, Braden G, Champion de Crespigny PJ, DeFerrari G, Drury P, Locatelli F, Wiegmann TB, Lewis EJ: Proteinuria reduction and progression to renal failure in patients with type 2 diabetes mellitus and overt nephropathy. *Am J Kidney Dis* 45: 281287, 2005
11. Lea J, Greene T, Hebert L, Lipkowitz M, Massry S, Middleton J, Rostand SG, Miller E, Smith W, Bakris GL: The relationship between magnitude of proteinuria reduction and risk of end-stage renal disease: Results of the African American Study of Kidney Disease and hypertension. *Arch Intern Med* 165: 947953, 2005
12. Hebert LA, Wilmer WA, Falkenhain ME, Ladson-Wofford SE, Nahman NS Jr, Rovin BH: Renoprotection: One or many therapies? *Kidney Int* 59: 12111226, 2001
13. Taal MW, Brenner BM: Renoprotective benefits of RAS inhibition: from ACEI to angiotensin II antagonists. *Kidney Int* 57: 18031817, 2000
14. Remuzzi G, Ruggenenti P, Benigni A: Understanding the nature of renal disease progression. *Kidney Int* 51: 215, 1997
15. Hebert L, Agarwal G & Sedmak D et al. Proximal tubular epithelial hyperplasia in patients with chronic glomerular proteinuria. Kidney Int 2000; 57: 19621967
16. MANUEL PRAGA and ENRIQUE MORALES:Renal damage associated with proteinuria kidney int 62: S42-S46, 2002
17. Keane WF: Proteinuria: its clinical importance and role in progressive renal disease. *Am J Kidney Dis* 35: S97S105, 2000
18. Ruggenenti P, Perna A, Mosconi L, Matalone M, Pisoni R, Gaspari F, Remuzzi G: Proteinuria predicts end-stage renal failure in non-diabetic chronic nephropathies. The "Gruppo Italiano di Studi Epidemiologici in Nefrologia" (GISEN). *Kidney Int Suppl* 63: S54S57, 1997
19. Branten AJ, van den Born J, Jansen JL, Assmann KJ, Wetzels JF: Familial nephropathy differing from minimal change nephropathy and focal glomerulosclerosis. *Kidney Int* 59: 693701, 2001
20. GISEN: Randomised placebo-controlled trial of effect of ramipril on decline in glomerular filtration rate and risk of terminal renal failure in proteinuric, non-diabetic nephropathy. The GISEN Group (Gruppo Italiano di Studi Epidemiologici in Nefrologia). *Lancet* 349: 18571863, 1997
21. Rossing P, Hommel E, Smidt UM, Parving HH: Reduction in albuminuria predicts a beneficial effect on diminishing the progression of human diabetic nephropathy during antihypertensive treatment.*Diabetologia* 37: 511516, 1994

22. African American Study of Kidney Disease and Hypertension: Effect of blood pressure lowering and antihypertensive drug class on progression of hypertensive kidney disease: Results from the AASK trial. *JAMA* 2003,
23. Ruggenenti P, Perna A, Remuzzi G: Retarding progression of chronic renal disease: The neglected issue of residual proteinuria. *Kidney Int* 63: 22542261, 2003
24. Muntner P, He J, Hamm L, Loria C, Whelton PK: Renal insufficiency and subsequent death resulting from cardiovascular disease in the United States. *J Am Soc Nephrol* 13: 745753, 2002
25. Jafar TH, Schmid CH, Landa M, Giatras I, Toto R, Remuzzi G, Maschio G, Brenner BM, Kamper A, Zucchelli P, Becker G, Himmelmann A, Bannister K, Landais P, Shahinfar S, de Jong PE, de Zeeuw D, Lau J, Levey AS: Angiotensin-converting enzyme inhibitors and progression of nondiabetic renal disease. A meta-analysis of patient-level data. *Ann Intern Med* 135: 7387, 2001
26. Ruggenenti P, Schieppati A, Remuzzi G: Progression, remission, regression of chronic renal diseases. *Lancet* 357: 16011608, 2001
27. Lane D, Beevers M, Barnes N, Bourne J, John A, Malins S, Beevers DG: Inter-arm differences in blood pressure: when are they clinically significant? *J Hypertens* 20: 10891095, 2002
28. Schrier RW, Estacio RO, Esler A, Mehler P: Effects of aggressive blood pressure control in normotensive type 2 diabetic patients on albuminuria, retinopathy and strokes. *Kidney Int* 61: 10861097, 2002
29. Schrier R, McFann K, Johnson A, Chapman A, Edelstein C, Brosnahan G, Ecder T, Tison L: Cardiac and renal effects of standard versus rigorous blood pressure control in autosomal-dominant polycystic kidney disease: Results of a seven-year prospective randomized study. *J Am Soc Nephrol* 13: 17331739, 2002
30. Group: Effects of ACE inhibitors, calcium antagonists, and other blood-pressure-lowering drugs: results of prospectively designed overviews of randomised trials. *Lancet* 355: 19551964, 2000
31. Lewington S, Clarke R, Qizilbash N, Peto R, Collins R: Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. *Lancet* 360: 19031913, 2002
32. Group AS: Major outcomes in high-risk hypertensive patients randomized to angiotensin-converting enzyme inhibitor or calcium channel blocker vs diuretic. *JAMA* 288: 29812997, 2002
33. Appel LJ: The verdict from ALLHAT-Thiazide diuretics are the preferred initial therapy for hypertension. *JAMA* 288: 30393041, 2002
34. Grantham J. Mechanisms of progression in autosomal dominant polycystic kidney disease. Kidney Int 1997; 52 Suppl: S93S97.
35. FALKENHAIN M. Nutritional management of water, sodium, potassium, chloride and magnesium in renal disease and renal failure,. inNutritional Management of Renal Disease 1997; edited by Kopple J, Massry S Baltimore, Williams & Wilkins pp 371394.
36. Ritz E, Orth S & Strzelcyk P. Angiotensin converting enzyme inhibitors, calcium channel blockers, and their combination in the treatment of glomerular disease. J Hypertens 1997; 15: S21S26.
37. Bakris G, Weir M & Dequattro V et al. Effects of an ACE inhibitor calcium antagonist combination on proteinuria in diabetic nephropathy. Kidney Int 1998; 54: 12831289.
38. Estacio RO, Jeffers BW, Gifford N, Schrier RW: Effect of blood pressure control on diabetic microvascular complications in patients with hypertension and type 2 diabetes. *Diabetes Care* 2 [23 Suppl]: B54B64, 2000
39. Lewis EJ, Hunsicker LG, Clarke WR, Berl T, Pohl MA, Lewis JB, Ritz E, Atkins RC, Rohde R, Raz I: Renoprotective effect of the angiotensin-receptor antagonist irbesartan in patients with nephropathy due to type 2 diabetes. *N Engl J Med* 345: 851860, 2001
40. Brenner BM, Cooper ME, de Zeeuw D, Keane WF, Mitch WE, Parving HH, Remuzzi G, Snapinn SM, Zhang Z, Shahinfar S: Effects of losartan on renal and cardiovascular outcomes in patients with type 2 diabetes and nephropathy. *N Engl J Med* 345: 861869, 2001
41. Gress T, Nieto F & Shahar E et al. Hypertension and antihypertensive therapy as risk factors for type 2 diabetes mellitus. N Engl J Med 2000; 342: 905912.
42. Bakris GL. Slowing nephropathy progression: Focus on proteinuria reduction. Clin J Am Soc Nephrol 3: S3-S10, 2008
43. Perico, n., Benigni, A. & Remuzzi, G. Present and future drug treatments for chronic kidney diseases: evolving targets in renoprotection. Nat. Rev. Drug Discov. 7, 936953 (2008)
44. Casas, J. P. et al. effect of inhibitors of the reninangiotensin system and other antihypertensive drugs on renal outcomes: systematic review and meta-analysis. Lancet366, 20262033 (2005)

45. Giatras I, Lau J, Levey AS: Effect of angiotensin-converting enzyme inhibitors on the progression of nondiabetic renal disease: a meta-analysis of randomized trials. Angiotensin-Converting-Enzyme Inhibition and Progressive Renal Disease Study Group. *Ann Intern Med* 127: 337345, 1997
46. Hilgers KF, Mann JF: ACE Inhibitors versus AT(1) Receptor Antagonists in Patients with Chronic Renal Disease. *J Am Soc Nephrol* 13: 11001108, 2002
47. Yusuf S, Sleight P, Pogue J, Bosch J, Davies R, Dagenais G: Effects of an angiotensin-converting-enzyme inhibitor, ramipril, on cardiovascular events in high-risk patients. The Heart Outcomes Prevention Evaluation Study Investigators. *N Engl J Med* 342: 145153, 2000
48. Dahlof B, Devereux RB, Kjeldsen SE, Julius S, Beevers G, Faire U, Fyhrquist F, Ibsen H, Kristiansson K, Lederballe-Pedersen O, Lindholm LH, Nieminen MS, Omvik P, Oparil S, Wedel H: Cardiovascular morbidity and mortality in the Losartan Intervention For Endpoint reduction in hypertension study (LIFE): A randomised trial against atenolol. *Lancet* 359: 9951003, 2002
49. Dickstein K, Kjekshus J: Effects of losartan and captopril on mortality and morbidity in high-risk patients after acute myocardial infarction: The OPTIMAAL randomised trial. Optimal Trial in Myocardial Infarction with Angiotensin II Antagonist Losartan. *Lancet* 360: 752760, 2002
50. Lewis EJ, Hunsicker LG, Bain RP, Rohde RD: The effect of angiotensin-converting-enzyme inhibition on diabetic nephropathy. The Collaborative Study Group. *N Engl J Med* 329: 14561462, 1993
51. Berl T, Hunsicker LG, Lewis JB, Pfeffer MA, Porush JG, Rouleau JL, Drury PL, Esmatjes E, Hricik D, Parikh CR, Raz I, Vanhille P, Wiegmann TB, Wolfe BM, Locatelli F, Goldhaber SZ, Lewis EJ: Cardiovascular outcomes in the Irbesartan Diabetic Nephropathy Trial of patients with type 2 diabetes and overt nephropathy. *Ann Intern Med* 138: 542549, 2003
52. Schanstra JP, Neau E, Drogoz P, Arevalo Gomez MA, Lopez Novoa JM, Calise D, Pecher C, Bader M, Girolami JP, Bascands JL: In vivo bradykinin B2 receptor activation reduces renal fibrosis. *J Clin Invest* 110: 371379, 2002
53. Maschio G, Alberti D & Janin G et al. Effect of the angiotensin-converting-enzyme inhibitor benazepril on the progression of chronic renal insufficiency. N Engl J Med 1996; 334: 939945.
54. Bakris GL, Siomos M, Richardson D, Janssen I, Bolton WK, Hebert L, Agarwal R, Catanzaro D: ACE inhibition or angiotensin receptor blockade: Impact on potassium in renal failure. VAL-K Study Group. PG - 208492. *Kidney Int* 58: 2000
55. Laverman GD, Navis G, Henning RH, De Jong PE, De Zeeuw D: Dual renin-angiotensin system blockade at optimal doses for proteinuria. *Kidney Int* 62: 10201025, 2002
56. Ruilope L, Casal M & Praga M et al. Additive antiproteinuric effect of converting enzyme inhibition and a low protein intake. J Am Soc Nephrol 1992; 3: 13071311.
57. De Jong PE, Navis G, de Zeeuw D: Renoprotective therapy: titration against urinary protein excretion. *Lancet* 354: 352353, 1999
58. Buter H, Hemmelder MH, Navis G, de Jong PE, de Zeeuw D: The blunting of the antiproteinuric efficacy of ACE inhibition by high sodium intake can be restored by hydrochlorothiazide. *Nephrol Dial Transplant* 13: 16821685, 1998
59. Bianchi S, Bigazzi R, Caiazza A, Campese VM: A controlled, prospective study of the effects of atorvastatin on proteinuria and progression of kidney disease. *Am J Kidney Dis* 41: 565570, 2003
60. Praga M, Gutierrez E, Gonzalez E, Morales E, Hernandez E: Treatment of IgA Nephropathy with ACE Inhibitors: A Randomized and Controlled Trial. *J Am Soc Nephrol* 14: 15781583, 2003
61. Berl T. Maximizing inhibition of the renin-angiotensin system with high doses of converting enzyme inhibitors or angiotensin receptor blockers. Nephrol Dial Transplant 2008;23:2443-7.
62. Hou FF, Xie D, Zhang X, et al. Renoprotection of Optimal Antiproteinuric Doses (ROAD) Study: a randomized controlled study of benazepril and losartan in chronic renal insufficiency. J Am Soc Nephrol 2007;18:1889-98.
63. Mehdi UF, Adams-Huet B, Raskin P, Vega GL, Toto RD. Addition of angiotensin receptor blockade or mineralocorticoid antagonism to maximal angiotensin-converting enzyme inhibition in diabetic nephropathy. J Am Soc Nephrol 2009;20:2641-50.
64. Burgess E, Muirhead N, Rene de Cotret P, et al. Supramaximal dose of candesartan in proteinuric renal disease. J Am Soc Nephrol 2009;20:893-900.
65. Remuzzi G, Benigni A, Remuzzi A. Mechanisms of progression and regression of renal lesions of chronic nephropathies and diabetes. J Clin Invest 2006;116:288-96.

66. Campbell R, Sangalli F, Perticucci E, Aros C, Viscarra C, Perna A, Remuzzi A, Bertocchi F, Fagiani L, Remuzzi G, Ruggenenti P: Effects of combined ACE inhibitor and angiotensin II antagonist treatment in human chronic nephropathies. *Kidney Int* 63: 10941103, 2003
67. Kunz, R., Friedrich, C., wolbers, M. & Mann, J. F. Meta-analysis: effect of monotherapy and combination therapy with inhibitors of the renin angiotensin system on proteinuria in renal disease. Ann. Intern. Med. 148, 3048 (2008).
68. Mann, J. F. et al. Renal outcomes with telmisartan, ramipril, or both, in people at high vascular risk (the ONTARGET study): a multicentre, randomised, double-blind, controlled trial. Lancet 372, 547553 (2008)
69. Piero Ruggenenti, Giuseppe Remuzzi.i the ON TARGET renal substudy actually off target. Nature review nephrology 5, 436-437 (2009)
70. Ruggenenti P *et al.* (2008) Role of remission clinics in the longitudinal treatment of CKD. *J Am Soc Nephrol* 19: 12131224
71. Giordano M, De Feo P, Lucidi P, DePascale E, Giordano G, Cirillo D, Dardo G, Signorelli SS, Castellino P: Effects of dietary protein restriction on fibrinogen and albumin metabolism in nephrotic patients. *Kidney Int* 60: 235242, 2001
72. Schrijvers BF, Rasch R, Tilton RG, Flyvbjerg A: High protein-induced glomerular hypertrophy is vascular endothelial growth factor-dependent. *Kidney Int* 61: 16001604, 2002
73. Sakemi T, Ikeda Y, Shimazu K, Uesugi T: Attenuating effect of a semipurified alcohol extract of soy protein on glomerular injury in spontaneous hypercholesterolemic male Imai rats. *Am J Kidney Dis* 37: 832837, 2001
74. Velasquez MT, Bhathena SJ: Dietary phytoestrogens: a possible role in renal disease protection. *Am J Kidney Dis* 37: 10561068, 2001
75. Pifer TB, McCullough KP, Port FK, Goodkin DA, Maroni BJ, Held PJ, Young EW: Mortality risk in hemodialysis patients and changes in nutritional indicators: DOPPS. *Kidney Int* 62: 22382245, 2002
76. Fried LF, Orchard TJ, Kasiske BL: Effect of lipid reduction on the progression of renal disease: A meta-analysis. *Kidney Int* 59: 260269, 2001
77. Zoja C, Corna D, Rottoli D, Cattaneo D, Zanchi C, Tomasoni S, Abbate M, Remuzzi G: Effect of combining ACE inhibitor and statin in severe experimental nephropathy. *Kidney Int* 61: 16351645, 2002
78. Chrysostomou A, Becker G: Spironolactone in addition to ACE inhibition to reduce proteinuria in patients with chronic renal disease. *N Engl J Med* 345: 925926, 2001
79. Orth SR: Smoking and the kidney. *J Am Soc Nephrol* 13: 16631672, 2002
80. Pinto-Sietsma SJ, Mulder J, Janssen WM, Hillege HL, de Zeeuw D, de Jong PE: Smoking is related to albuminuria and abnormal renal function in nondiabetic persons. *Ann Intern Med* 133: 585591, 2000
81. Kambham N, Markowitz GS, Valeri AM, Lin J, D'Agati VD: Obesity-related glomerulopathy: an emerging epidemic. *Kidney Int* 59: 14981509, 2001
82. Cattran D, Bartosik LP, Scholey J, Duncan J, Miller J: Does obesity initiate or worsen renal function in the progressive nephropathies [Abstract]. *J Am Soc Nephrol* 12: 96A, 2001
83. Tarif N & Bakris G. Preservation of renal function: The spectrum of effects by calcium-channel blockers. Nephrol Dial Transplant 1997; 12: 22442250
84. Wilmer WA, Rovin BH, Hebert CJ, Rao SV, kumor K & Hebert LA. Management of glomerular proteinuria: A commentary. *J Am Soc nephrol* 14: 3217-3232, 2003

Case report

A case of symptomatic Morgagni's hernia

Introduction:
Congenital diaphragmatic abnormalities occur in 1/2,000 to 1/4,000 membranes. The most common defect is congenital diaphragmatic hernia, and 90% of these hernias are Bochdalek's type. Morgagni's hernias are less common. Although Morgagni's hernias are generally asymptomatic and frequently found coincidentally during routine diagnostic tests for other pathologies, rarely they may be symptomatic due to intestinal obstruction or respiratory distress. We present an adult Morgagni's hernia case with acute abdominal symptoms secondary to transverse colon herniation and intestinal obstruction as a consequence.

Case report Clinical course:
A 73 year old female patient was admitted to the primary care unit of our hospital with the history of abdominal pain, abdominal distension and absolute constipation of three days duration and vomiting of one day duration. On examination the patient was dehydrated, tachycardic, abdomen distended and mild tenderness in the right hypochondrium and bowel sounds weak. Digital rectal examination showed dilated empty rectum. She has no past history of chest or abdominal trauma.

Clinically intestinal obstruction was suspected. Supine abdominal X-ray and erect chest X-ray were done. Erect chest X-ray showed the bowel loop in the right thorax. Supine abdominal X-ray showed dilated ascending colon. A lateral chest X-ray was performed which showed herniation of bowel loop through the anterior part of the Diaphragm.

Picture 1: Chest and Abdominal X- Rays.

Erect chest x ray Supine abdomen Lateral chest

S.Mathievaanan[1], R.M.P.S.Ratnayake[1], G.K.S.P.P.Premaratna[2], Ajith de silva[3].
[1]Registrar, [2]Resident Surgeon, [3]Consultant Surgeon
Colombo North Teaching Hospital, Ragama.

Diagnosis was made as intestinal obstruction due to diaphragmatic hernia and emergency laparotomy was performed. The finding was Morgagni hernia containing transverse colon and omentum with proximal bowel dilatation.

The hernial contents were reduced and defect repaired with the non absorbable suture. Patient recovered and post op Chest X-ray was normal.

Discussion:

Morgagni hernia was reported for the first time by Morgagni in 1769 and accounts for 3% of all diaphragmatic hernias. In Morgagni hernia, intra-abdominal organs are herniated into the thoracic space through a right retrosternal fissure in the diaphragm. In Morgagni hernia, the transverse colon and the great omentum are likely to herniate into the thoracic space. Because herniated organs are usually covered with hernia sac, which is parietal peritoneum, patients usually do not have symptoms. In contrast, Bochdalek hernia, which is the most common type of diaphragmatic hernias, lack the hernia sac and usually patients have severe abdominal symptoms.

The Clinical presentation in adults tends to be very different from neonates. Many adults remain asymptomatic and congenital diaphragmatic hernias diagnosed incidentally. In Morgagni hernia, 30% of patients have abdominal symptoms such as upper abdominal pain and vomiting, 30% have respiratory symptoms such as dyspnea, cyanosis and cough, and 35% remain asymptomatic. Symptoms are self-limiting in most cases. However, there have been several reports of emergency operations because of small or large intestinal Morgagni hernias.

Plain Chest X- ray is useful in diagnosis. Unfortunately, in many cases radiographic appearances are either too small to detect or tend to be misinterpreted as consolidations, delaying Diagnosis CT Scans using a radiopaque contrast have offered a more sensitive diagnostic option. CT is a static evaluation it may not identify a diaphragmatic defect if there is no herniation at that time. It may not possible to visualize a diaphragmatic defect through the transverse cuts of the CT scans as they are parallel to the defect

Upper GI contrast study gives an excellent visualization of organo - axial rotation.
Direct visualization through the abdominal laparoscopy may be most useful.

All congenital Diaphragmatic hernias should be repaired. Minimally invasive surgical approaches are now gaining popularity for the repair of congenital diaphragmatic hernia with excellent outcome.

Conclusion:

This case report highlights the unusual presentation of obstructed viscera within a Morgagni's hernia in a senile and elderly patient. Although uncommon, herniation of the omentum and the bowel can occur in the adult through the foramen of Morgagni. This report highlights a case scenario where an elderly patient may require emergency intervention on presentation because of the risk of strangulation of the bowel.

References

1. Arora S, Haji A, NgP, Ann of R coll surg Engl 2008 Nov; 90(8)694-69
2. ArudchelvamJD, DiasP, Weerasekara D, Srilanka Journal of surgery 2009:27:17-8
3. Suat E, Hatice G, Adnan O, (2003) A rare cause of intestinal obstruction in the Adult: Morgagnis hernia 7:97-99
4. Y.Iso, T.sawada, K.Rokkaku, T.Furihata, M.Shimoda, J.Kita and k.Kubota, Hernia, The world journal of hernia, 16 Aug 2006
5. Barut I, Tarhan OR, cerci C,Akdeniz Y,Bulbul M, J Thoracic imaging 2005aug;20(3),220-22
6. A.J.Paily& A.K Lannigan: A case of obstructed Morgagni Hernia, The internet journal of surgery 2009 vol 19:2
7. Santosh PV Rai, Madhav kamath,Ashok shettty, Suresh shenoy,Ashvini Kumar, Journal of emergency trauma and shock,2010 jan-mar ;3(1):89-91

Review

Obesity an emerging pediatric problem

Childhood obesity has reached epidemic proportions worldwide and more children are presenting for medical care. It was first included in the international classification of diseases from 1948. The danger of childhood obesity is that it persists into adulthood and related pathological changes begin from childhood leading to non communicable diseases and complications at a younger age. Therefore early detection and correction of childhood obesity is of paramount importance. (1)

Epidemiology

The rise in childhood obesity over the past decade has been dramatic. It is not only the scale of childhood obesity that is challenging, but also the speed at which the prevalence has increased.

Overall in developing countries the prevalence is 12-16% of the childhood population. (2)

In Sri Lanka a survey conducted among urban schools in Colombo district in 2004 showed the prevalence of obesity, overweight and the under nutrition. The overall prevalence of obesity was 4% and overweight was 11 %.(3)

Diagnosis of obesity in children:

Obesity is defined as an excess fat in the body associated with morbidity. Many different methods are used to measure adiposity in children. Direct methods such as densitometry and scanning using dual energy x-ray absorptiometry are more accurate than indirect methods. The indirect methods are waist circumference, skin folds and BMI are used. Bioelectrical impedance and air displacement plethysmography provide reliable indirect methods which are often used in research studies.

Body mass index (BMI) is the only method that is feasible outside the realms of research. BMI is calculated from the formula BMI (kg/m^2) =weight (kg) /height^2 (m^2). Although consensus is lacking a body fat content of more than 20-25% in boys and 25-32% in girls is associated with morbidity. Gender specific BMI for age and WHO growth charts can be used for calculation of BMI. Obesity is diagnosed when BMI is >95th centile (or >2 SD). BMI between 85th to 95th centile (+1 to +2 SD) is considered as overweight or at risk of obesity. (1)

Aetiology

Obesity is caused by an imbalance between energy input and energy expenditure. The vast majority of children have nutritional or exogenous causes such as the societal changes. (4) Causes of obesity are listed in table 1.

Table 1: Causes of obesity

Common Causes
Nutritional
Psychological
Rare causes
Syndromes – Prader Wili, Bardet Beidl , Alstrom
Endocrine – hypothyroid, Cushing's , GH deficiency, hypoparathyroid
CNSpathology – hypothalamic causes

M.G.Sathiadas,
Senior Lecturer in Pediatrics,
Faculty of Medicine, University of Jaffna

Risk Factors associated with obesity

The relative contribution of sedentary life style and diet, to the development of obesity in children is unclear, partly because these variables are difficult to measure and the balance of energy is complex. A large number of studies have reported associations between a wide variety of risk factors and overweight or obesity in children. Randomised controlled trials and prospective studies provide the most robust evidence for identifying causal associations, but for obesity relatively little evidence comes from randomised controlled trials.

Several systematic and non systematic studies have shown a clear link of diet and the physical activity to childhood and adolescent obesity. Low levels of physical activity and inactive parents are associated with obesity. Considering the dietary pattern formula feeding, missing breakfast, snacking in between meals, eating large portions of meals and energy dense food and beverages are risk factors for obesity. A positive correlation exists between hours of television viewing and overweight, which is stronger with increasing age. (1)

Considering the genetics, reports of case series have suggested single gene defects in which obesity is the specific abnormality, are related to the leptin or the melanocortin system. The inheritance pattern also suggests that obese parents will have obese children. A cohort study done in the USA showed parental obesity more than doubles the risk of adult obesity in children under 10 years of age and 80% of children with two obese parents become obese.(5)

The ethnic origin also plays a key role in obesity. Combined longitudinal and cross sectional studies have shown that Hispanic, Native American, black, and south Asian children have an increased risk of obesity compared with white children.

Intrauterine exposures to diabetes and maternal obesity especially during pregnancy have shown a clear risk factor for childhood obesity. The birth weight of babies also predicts the outcome, small for gestational age babies who exhibit catch-up growth may be at risk of obesity in children. A non systematic review suggests large for date babies have an increased risk of adolescent obesity. (1)

Other risk factors associated with obesity are urban dwelling, socio-economic status of the family, single parent family and drugs. (1)

Consequences of obesity

The effects are both physical and psychological. Obesity was considered as a cosmetic problem until recently evidence has shown the association with non communicable diseases. A study in the USA has shown 58% of the children with BMI >95th centile had hypertension, hyperlipidemia and insulin resistance, 25% had more than one of these conditions.(5) A systematic review by Owen CG, Whincup PH, Orfei L,et al showed that BMI from childhood onwards is positively related to risk of coronary heart disease. (6) Studies also have shown the risk of the metabolic syndrome is nearly 50% in severely obese (BMI $\geq$ 40.6 kg/m^2) adolescents. The incidence of Non Alcoholic Fatty Liver Disease/Non Alcoholic Steato Hepatitis (NAFLD/NASH) depends on the detection method. It can be detected either by liver function tests or ultrasonography of the liver. In an autopsy study, the incidence of fatty liver was 38% in obese children 219 yr of age.(7)

The most common problem associated with obesity is psychosocial. The problems are depression, poor quality of life and low self esteem. Social acceptance is affected if children/adults are obese and they are more prone to get weight related victimisation. Twenty percent of girls and 32% of the boys of 15-19 year olds with major depression have a BMI of more than 95th centile. Incidence is also seen to increase with increasing BMI. When 12 year old girls with obesity and their non obese counterparts were compared there was a significant difference in self worth, athletic ability and acceptance of appearance. (8)Table 2 summarises the consequences of obesity.

Table 2: Consequences/comorbidities of Obesity

Metabolic	Physical	Psychological
Diabetes mellitus	Slipped upper femoral epiphysis	Poor self esteem
Cardiovascular disease	Blount's disease	Depression
Metabolic syndrome	Flat feet, genuvara	Poor quality life
NAFLD/NASH	Scoliosis	Social acceptance affected
Glomerulosclerosis	Obstructive sleep apnoea	Weight related victimisation
Polycystic ovarian disease		
Gall stones		
Pseudotumour cerebrii		

Management of obesity

Paediatricians find managing obesity a daunting experience and unrewarding as it is hard to help children to lose a meaningful amount of weight. Goals have to be defined in paediatric management of obesity. Goals are identification of a medical cause, identification of any consequences of obesity, promotion of weight loss/control and management of medical problems. A proper clinical evaluation is needed to achieve this. Table 3 summarises the clinical evaluation.

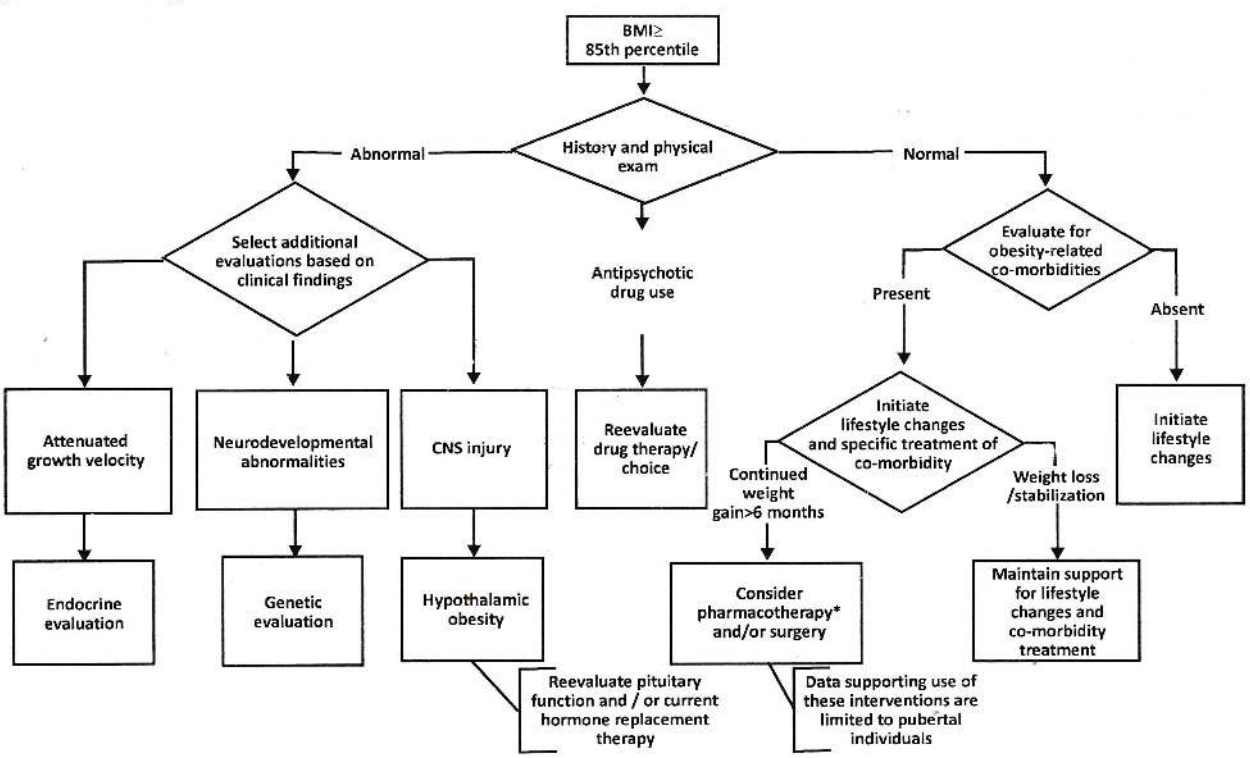
Table 3: Clinical evaluation of the obese child

History	Physical evaluation
Age of onset	Measurement s – height, weight, waist:hip ratio
Dietary habits	BMI
Physical activity	Dysmorphic features
School and social issues	Features of hypothyroidism/Cushings/ others
Sleep problems	Blood pressure
Hip/knee pain	Hirsutism
Menstrual irregularities	Acanthosis nigricans
Family history	
Co morbidities	
Features of endocrine abnormalities	

In a child (above 5 years) with the clinical suspicion of simple obesity, basic investigations are performed to evaluate associated co morbidities rather than to identify the aetiology. Investigations in an obese child below 5 years of age are usually done only when there is severe obesity with strong family history of co morbidities or resistance to initial therapy.

Fasting blood sugar, lipid profile, liver transaminases and ultrasound scan of the liver are performed initially and every 6-12 months thereafter. Features suggestive of a genetic or endocrine disorder warrant special investigations and /or referral to a specialised centre. Bone age is advanced in simple obesity but matches the height age of the child.

Figure 1 - management flow chart.



August G P et al. JCEM 2008;93:4576-4599

Mainstay of the management includes lifestyle modifications. It is based on Child's lifestyle, Co morbidities, willingness to change and support from family. Behavioural change occurs gradually over a long period of time. Targets such as weight maintenance if BMI> 98 or weight reduction of 0.5-1.0kg/month if BMI>99.6 are set depending on the stage of puberty. This is achieved by healthy eating, increasing physical activity and reducing sedentary behaviour. (9)

Dietary habits

A total energy intake must be reduced in addition to avoiding consumption of calorie dense, nutrient poor food. An increase of dietary fibre, fruit and vegetables should be encouraged in children. They should also be encouraged to eat regular timely meals and reduction of snacking. A reduction of consumption of saturated fat is recommended after 2 years of age. To maintain ideal body weight the intake of fruit and vegetables must be 33%; cereals, bread and rice 33%; milk and dairy products 15%; meat, fish and egg 12% and food and drinks with high fat 7% of the total intake. Calorie intake should be 80% of the normal. The practical way of avoiding consumption of high calorie containing refined food is by avoiding stocking them at home. Therefore the cooperation of the entire family is needed and by doing so it will help prevent obesity/overweight.

Physical activity

Physical activity not only reduces weight but also improves the cardiovascular fitness. Moderate to vigorous physical activity of 60 minutes per day is encouraged. Sedentary time (watching television, computer games and tuition class) must be reduced to two hours per day. Exercises like brisk walking, dancing, swimming and cycling on flat terrain should be encouraged. Children should also get involved in day to day household activities and must avoid using automated appliances.

Pharmacological management

Pharmacological agents are not recommended in children unless they have persistent severe co morbidities despite lifestyle modifications and a strong family history of type 2 diabetes mellitus (T2DM) or cardiovascular risk factors. Drugs that are used in children are Sibutramine (FDA for>16yrs), Orlistat (FDA for >12years) and Metformin (obesity and T2DM).

Bariatric surgery

Surgery is considered as the last option when there are severe persistent obesity and co morbidities despite life style modification along with pharmacological interventions. The procedures done are laparoscopic gastric banding and Roux-en-Y gastric bypass. Surgery is recommended only if the pubertal stage has reached 4-5 and near final height, BMI >50kg/m^2, or BMI >40kg/m^2 and significant co morbidities. Prior to surgery psychological evaluation is needed in view of the family unit and to assess the patient's ability to adhere to life style modifications.

Obesity intervention programmes

Some centres in the west have introduced obesity intervention programmes and strategies such as stimulus control, self monitoring, goal setting; rewards for achieving goals and problem solving are implemented. Praise and encouragement by parents to role model desired behaviour of children are also recommended. Whether these intervention programmes are successful is debatable. A recent study done in obese children with NALFD who underwent interventions as outpatients (12 days per 6 months) or as in patients (5 days in 6 months) with exercise (1hour/3 times per week) and dietary interventions showed statistically significant improvement in BMI and liver function tests. (10)

Prevention

Interventions regarding prevention should be initiated at home, school, community and clinical settings. Dietary intervention to prevent obesity include exclusive breast feeding for 6 months, avoiding excessive feeding in IUGR babies, reducing fat containing food at preschool level, improve quality of school lunch and reducing consumption of carbonated drinks.

Physical activity should be encouraged to maintain BMI. Society has to implement regulatory policies regarding media advertisement of food. Policies to stock healthy food in school cafeterias and vending machines must be implemented. Parental participation in school activities should be encouraged. Incentives must be given to retailers, who sell fruit and vegetables. Policies must be implemented to prevent fast food stalls especially near the school. There should be opportunities for safe walking or cycling to school, recreational activity and

athletic events which will encourage greater physical activity. As medical professionals the duty is to screen children for obesity, to identify the co morbidities, to initiate life style intervention and to establish obesity clinics. The whole family should be involved in the care.

References

1. Ruth R Kipping, Russell Jago, Debbie A Lawlor. Obesity in children, epidemiology, measurement and risk factors. BMJ 18 October 2008;Volume 337:922-27
2. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 1997;337:869-73.
3. Wickramasinghe P, Sathiadas G et al. Dietary and physical activity patterns of school children in an urban area of Sri Lanka, Sri Lanka Journal of Child Health 2005;34:44-49
4. Mary CJ Rudolf. Best practice on obese child. Arch Dis Child Educ Pract Ed 2004; 89:ep57ep62
5. ParsonsTJ,Power C,LoganS,SummerbellCD.Childhood predictors of adult obesity: a systematic review. Int J Obes Relat Metab Disord 1999;23(suppl 8):S1-107.
6. Baker JL, Olsen LW, Sorensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. N Engl J Med 2007;357:2329-3
7. Ram Weis, James Dziura et al. Obesity and the metabolic syndrome in children and adolescents. N Engl J Med 2004;350:2362-74. .
8. Melissa Wake, Louise Canterford,et al. Co morbidities of overweight/obesity experienced in adolescence:longitudinal study. Arch Dis Child 2010;95:162168
9. Management of obesity in children and young people: a national clinical guideline, Scottish Intercollegiate Guidelines Network 69. April 2003. Published by Royal College of Physicians, Edinburgh.www.sign.ac.uk
10. Christina Wei, Anna Ford et al. Abnormal liver function in children with metabolic syndrome from a UK-based obesity clinic.Arch Dis Child 2011;96:1003-1007
11. Reilly JJ, Methven E, McDowell ZC, et al. Health consequences of obesity. Arch Dis Child 2003;88:74852.
12. Wickramasinghe P. Treatment and prevention of childhood obesity. Ceylon Medical Journal 2011; 56: 77-80

The 2010 American College of Rheumatology/European League against Rheumatism classification criteria for rheumatoid arthritis

Who should be tested? Patients who

1) Have at least 1 joint with definite clinical synovitis
2) With the synovitis not better explained by another disease

Classification criteria for RA: score based algorithm: add score of categories A-D

A score of 6/10 is needed for a patient as having definite RA

A. Joint involvement	
1 large joint	**0**
2-10 large joints	**1**
1-3 small joints (with or without involvement of large joints)	**2**
4-10 small joints (with or without involvement of large joints)	**3**
>10 joints (at least 1 small joint)**	**5**
B. Serology (at least 1 test result is needed for classification)	
Negative RF *and* negative ACPA	**0**
Low-positive RF *or* low-positive ACPA	**2**
High-positive RF *or* high-positive ACPA	**3**
C. Acute-phase reactants	
(at least 1 test result is needed for classification)	
Normal CRP *and* normal ESR	**0**
Abnormal CRP *or* abnormal ESR	**1**
D. Duration of symptoms	
>6 weeks	**1**
<6 weeks	**0**

JMA Forum

Electronic Health Care: A review for a practising Physician

World Medical system has moved towards an electronic health care and paperless ward rounds and patient care. When and how this transformation is going to happen in Sri Lanka is unclear, but the knowledge about this evolving electronic health care delivery is essential for a practicing Physician in any part of the world. This is a general review with some focus on one of the best electronic health care delivery system in the world which is also facing major health care reform and challenges in history, the US health care delivery.

Abstract

Physicians are facing many challenges in electronic transformation of medical records and the health care delivery system. This is a review of health care informatics and information technology and how it affects the practices. Universal electronic medical records (EMR) are essential in re-structuring health care. It helps to improve quality of care and promotes evidence based medicine. Electronic health care delivery system needs standardization, privacy protection, use of universal medical terminology and inter-operability. The challenges are high implementation and operational cost, technological difficulties and knowledge, information overflow and lack of reimbursement for co-ordination of care.Health care reform is a shared responsibility of the government, insurers, medical organizations and Physicians.

Introduction

In the era of health care re-form busy Physicians are facing many challenges in electronic transformation of medical records and the health care delivery system. E-prescribing, digital records and Imaging, reporting quality measures electronically, public health reporting and datacollection for research and education, all have major impact on health care providers. The Physicians are not provided with in-depth knowledge of this electronic health care transformation and not well informed about the medical informatics. Physicians in small groups struggle with funding and resources to deploy electronic health records and Physicians in hospitals and large groups are seeing massive and rapid changes to digital records and imaging environment. They need to cope up with the challenges involved in this massive transformation without compromising patient care and the overall health care delivery at the bed side. In this review we are providing the basic knowledge of health care informatics and information technology, what it means to a busy Physician, challenges and impact on health care delivery and how does it affect specialties.

Electronic health records are the key to Health care Reform

Electronic health records are the main key to health care reform. Universal electronic medical records are essential in re-structuring the health care (1). This will increase efficiency, decrease medical errors and health care cost in a long run. It requires strict privacy protection. The concept of a health care bank account for each person where the medical records could be saved and health care providers could access and deposit the findings and test results with each patient encounter and universal access upon request under Health Insurance Portability and Accountability Act (HIPPA). This account will become the patient's private account and patient will have the sole authority to control the record. It will cost 1US$ per month to maintain these accounts and the benefits and heath care cost saving by this availability is many times high (2).

Interoperable electronic health records (EHR) will significantly increase accountability for care. It will help in payment reform, data use policies, Information Technology (IT) strategy, and data management. Data

Sri Sujanthy Rajaram
Assistant Professor of Medicine
Cooper Medical School at Rowan University (CMSRU)
University of Medicine & Dentistry New Jersey (UMDNJ)
Division of Critical Care, Department of Medicine
Cooper University Hospital, Camden, New Jersey 08103, USA

will become a strategic asset, in healthcare finances, research and improved patient outcome. Major advantages are improved quality of health care, promotion of evidence based medicine and help in proper record keeping for research and procedures associated with patient care. Major disadvantage is the initial high cost to establish a system which is universal and protect the patient privacy. But with the recent American recovery and reinvestment Act of 2009, providers are expected to take full risk of investing in healthcare IT. Medicare announced significant cost savings for providers if electronic health records are adopted. The HITECT Act, part of 2009 economic stimulus package also provide incentives for EHR and reducing payments for who do not use EHR(3). Another important area addressed in Health Information exchange (HIE), for hospitals to use meaningful exchange of information receives a stimulus funding. This will avoid duplicate of testing and procedures and reduce the cost. Other cost involves in adopting Universal electronic records in addition to the initial startup cost are training cost, software maintenance costs and implementation (3).

Health Informatics and electronic health records system have been already adopted in many other countries like Argentina, Brazil, Canada, and European Union member states, China, Hong Kong, Saudi Arabia and Australia. Canada has adopted a universal healthcare so as Australia and many European countries and their cost for medical care is far less compare to United States (4, 5). The current financial crisis and the rising health care cost in United States make health care reform a dire emergency and if not done, it is a threat to entire economy. If health care cost continues to be the same way in our country the existing system will completely collapse, and this makes the health-care reform an urgent priority. For the health care reform to take place and the change to occur it is important to establish a universal or an interchangeable or readable, electronic health record system first. Health informatics and electronic health records becomes the key to this reform in the current climate. Many hospitals and practices are already adopting the EHR and it is estimated that United States will at-least will require about 50,000 IT workers to reform the health care and manage the EHR. This brings many job opportunities for the students to go into Healthcare Informatics in the future (6).

Fundamentals of Health Care Informatics
History of Computers

Information technology and the first use of a computer type model can be traced back to 1793, when Joseph-Marie Jacquard (1752-1834), who is a weaver and ex-soldier from Lyons, France, used the punch cards to make the loom industry automatic and 24 times faster than the manual labor for weaving (7). Following this the first programmable electronic computer called ENIAC (8.5x3x80 feet total of 680 sq.feet, weight 27 tons) was constructed at University of Pennsylvania for the United Sates Army at a cost of $500,000 in 1946 (8). In 1964, IBM started manufacturing computers followed by the first microcomputers appeared in 1975, portable computers evolved in 1982 and handheld computers appeared in 1990.

Health care Industry has been well known for not utilizing innovative technology years after the invention and the first use of thermometers by French clinicians, almost 200 years after the invention in 1820 is a popular example (9). The current concept of electronic medical records was dreamed by an American Engineer, Vannevar Bush who is well known for his political role in the development of atomic bomb, and the device he called as Memex(memory extension), which is a microfilm based device which stores a person's books, records and communications and which can be consulted as needed with exceeding speed and flexibility(10).

Medical records werenon- existent in the 1800's, but there was an epidemiology history. Pierre Charles Alexandre Louis (1787-1872), who was a French Physician and one of the founders of medical statistics, for the first time in history, recorded a "numerical method" of data collection about a bloodletting treatment for some inflammatory diseases(11). Only until 1950 paper medical records were started, during the 1960's large organizations first started using computers in health care, the next two decades it became more prevalent and after 1990 internet was launched and sophisticated clinical software, human genome mapping and digital education were all part of health care industry (12).

Now the health care industry is moving from Analog (something physical or tangible form of information storage e.g. compact disc) to digital storage form in which the data are stored as discrete numbers, in electronic medical records. Advantage of the digital system is the data could be shared by many clinicians, in a secure way at the same time.

Medical Informatics

Medical Informatics is the field that deals with storage, sharing, optimal use of biomedical information, data and knowledge for problem solving and decision making in the areas on computing and communication. It links the online clinical knowledge and content into digital form, and creates electronic medical records. It utilizes rules and alerts, so that order entries could be monitored, such as allergies pop up when a medication is ordered. The system also

could create a follow up or preventive service plan. Because of the diversity of information entered in to the medical records there need to be standards developed, to improve quality, assess the effectiveness and to achieve inter-operability within various systems. As an example American Medical association and National electrical Manufactures association agreed on a standard specification of medical Imaging called DICOM, which is widely used for clinical images (12).

Another important aspect in this electronic transformation is the use of common medical terminology. Apelon Thesaurus is a worldwide leader in healthcare vocabulary management and enhancement system and has over 1.4 million names and 750,000 concepts (13). If a medical term is typed such as diabetes or high sugar it will be able to link the much recourseavailable to review and help improve the standards and clinical outcome.

If large amount of medical and personal information stored digitally in hospitals and Physicians offices there need to be adequate back up storage plans in case of any disaster and loss of computers and network system such as flooding or hurricanes. Electronic records are highly confidential and need to protect the patient's privacy; there need to be authentication methods to access the data by pass words, PIN numbers, finger print readers, and voice print readers by the authorized users only. EHR could be linked to the iPhones and blackberries, so that e-prescribing is made available. Another technology available is Air Strip Observer suites, such Air Strip Critical care, where the patient's bedside monitor and ventilator readings could be displayed in the Physician's mobile hand held device. Air Strip Obstetrics, Air Strip Cardiology, Air Strip Laboratory, and Air strip imaging are other available Air strip technologies. Same mobile computer technology is also used by nurses to support medication administration with bar coding (14). Similarly pharmacy could use an automated drug dispensary system.

Challenges and Impact of electronic health care Delivery

The transition from paper to electronic notes has been regarded as valuable to patient care and hospital operations, Physicians face many challenges. Physicians may face difficult work environment, understaffing, equipment problems, discomfort with technology, and a focus on short-term rather than long-term goals.

In a recent report, two years after the conversion to Electronic Medical Record (EMR), the challenges include time for note entry, and the perception that notes may be more difficult to understand and to find within the EMR (15). American Recovery and Reinvestment Act of 2009 allocated funds to hospitals and Physician practices to adopt EMRs, only 44% of hospitals are adopting EMRs according to a report from hospitals and health network. The top reasons for adopting EMRs are improving quality of care reduce dictation and transcription cost, to reduce general operating cost, to meet patient and market expectations and to support pay-for-performance. The main barriers to EMR adoption are cost, support, EMR make up, interoperability and implementation. Many Physicians perceive that EMR will take away the autonomy and increase work load. Independent practices seek customized EMR's and they are more difficult to maintain and interface with hospitals. With limited resources EMR implementation in Physician offices, ongoing maintenance and system upgrades are costly and time consuming for hospitals and IT departments (16).

In a recent study from 26 practices with commercial ambulatory care EMRs in place for at least 2 years, reported six major themes. EMRs facilitate within-office care coordination, during patient visits and through electronic messaging. EMRs are less able to support coordination and information exchangebetween clinicians and settings, due to lack of standardization. Managing the significant information overflow from EMRs is a challenge for clinicians. Physicians believe EMRsdid not adequately capture the medical decision-making process and future care plans, which support coordination of care. The practices needed operational processes. Finally the current fee-for-service reimbursement encourages EMR use for documentation of billable events such as office visits and procedures, but did not address care coordination which is not a billable activity, but consumes significant time and expertise (17). By reforming a payment policy to include care coordination, EMRs with technology for this interoperability which provides inter-practice data exchange and multi-provider clinical decision support are key elements for effective electronic health care delivery. Coordination of care in chronic care patients with diabetes in four different models using EMRs has shown the persistent challenges despite the continuity of information provided. When multidisciplinary team provides chronic care and longitudinal care plan, structured communication is still a challenge with EMRs (18).

In specialties like radiology although the imaging technology has undergone dramatic changes and innovation, the radiology reporting has been stagnant

for decades. A good radiology report has several components of "C"s such as clarity, correctness, confidence, concision, completeness and consistency. In order to enhance good clinical outcome and to reduce medico-legal liability, communication and consultation between radiologist and clinician is of greater importance. Timeliness and standardization are important aspects of radiology reporting especially when it comes to mammography and scans reporting potential tumors. With widespread adoption of computerized imaging and new imaging technologies such as PACS system access to medical imaging is ubiquitous and instantaneous. The most challenging in medical imaging is standardization of reports to meet the political, economical and clinical demands. This involves number of technical, clinical and psychological challenges within the radiology community (19).

In pediatrics development of Personal Health records (PHR) to improve quality of health care in children is a challenge. The information is gathered from multiple contributors, such as parents, caretakers, patient, family and other healthcare providers. In Pediatrics PHR use and maintenance, child- and adolescent-friendly standards for PHR content, portability, security, and privacy are important challenges. PHR is a lifelong and comprehensive record for a patient and should begin at birth. The basic principle of the ideal PHR are equal access to all children regardless of income, information availability at all the time, adequate data exchange capability between hospital systems and registries(Immunization, hearing screening, newborn metabolic screening) and pediatricians and content standards must be aligned . Other key challenges are to provide appropriate amount of useful data to provide to specific professionals, control of PHR access by the patient or parent or guardian, protections of PHR data, adolescent rights, special protection for specific personal data, specific health information such as mental illness, social conditions like incarceration, genetic susceptibility, auditing the data entry and integrity, extensible functionality of records in immunization, growth, childhood obesity, continuity of care and support for adoption programs (20).

The Massachusetts eHealthCollaborative has recently implemented EHRs in a diverse set of communities, about 500 physicians serving over 500,000 patients. Both EHR implementation and health information exchange at the community level has identified numerous challenges. The factors which are essential for success are strong financial backing, intensive practice support, commitment to collective action, clear goals, leadership from the physician community, governmental support, and a community-based focus. Important barriers identified were inadequate standards for data representation and vocabulary, concerns about vendor instability and system obsolescence, system limitations, privacy and security issues, contracting demands, and practice inertia. The lack of universal standards for vocabulary and data representation was a major obstacle. To achieve interoperability, to exchange even rudimentary data, competing vendors need to provide considerable technical and organizational commitment. Electronic health records play a key role in patient centered medical management and the payment reform that encourage this approach and sharing of clinical decision making may work better than providing financial support to purchase the necessary software and hardware (21).

In the emergency room and critical care units implementing electronic health records face unique challenges due to the large volume of patients and the high acuity respectively; care cannot be compromised or delayed due to transitioning and allowing the Physicians to adapt to the new technology.

Conclusion

Many practices and hospitals face the challenges of maintaining physician compensation as reimbursement declines, operating costs that are escalating faster than revenues, the threat of further cuts in Medicare reimbursement andimplementing an electronic health record (22).

EHRs are the key to reinforce medical competence, improve relationships with patients, implement disease management programs, and, by increasing transparency and accountability and help reduce some conflicts of interest. In addition to financial and technical support, Cooperation, collaboration and the responsibility should be shared by government, insurers, medical organizations and Physicians, are needed to implement these information technology tools in the United States' dispersed and fragmented medical care system (23).

References

1. Glaser J, Markell P, Stone J, The strategic importance of data, Healthcare Financial Management. 2010 Feb; 64(2):46-52.
2. WA.Yasnoff, Electronic records are key to health care reform, business week,dec.19,2008 daily flash
3. RSteinbrook, Health Care and the American Recovery and Reinvestment Act, NEJM 360;11 nejm.org march 12, 2009
4. http://en.wikipedia.org/wiki/Electronic_health_record
5. http://en.wikipedia.org/wiki/Health_informatics
6. David Blumenthal, Health IT buzz, Office of the coordinator for health IT, November 24, 2009; M.Minear, Lectures 1-2, Health Informatics, JHSPH, 2010
7. History of Science.com: "The Most Famous Image in the Early History of Computing" http://www.historyofscience.com/G2I/timeline/index.php?category=Computers+%26+the+Human+Brai n.
8. http://en.wikipedia.org/wiki/ENIAC
9. E.F.J. Ring The historical development of temperature measurement in medicine, Infrared Physics & Technology, Volume 49, Issue 3, January 2007, Pages 297-301
10. Buckland, Michael. "Emanuel Goldberg, Electronic Document Retrieval, andVannevar Bush's Memex." *Journal of the American Society for Information Science*43, no. 4 (May 1992): 284294.
11. Morabia A. (2004), *Pierre-Charles-Alexandre Louis and the Evaluation of Bloodletting*, [The James Lind Library] (www.jameslindlibrary.org
12. Michael N.Minear, Health Informatics, John Hopkins Bloomberg School of Public Health lectures, 2010.
13. www.apelton.com
14. Honeywell healthcare professions guide to scanning and mobility, Data capture solutions for healthcare applications, 2009 Honeywell international Inc.
15. Payne.PH, Transition from paper to electronic inpatient physician notes. J Am Med Inform Assoc. 2010 Jan-Feb; 17(1):108-11.
16. Runy LA, IT challenges in Physician practice management, H&HN: Hospitals & Health Networks (H&HN HOSP HEALTH NETW), 2009 Nov; 83(11). (5p
17. O'Malley AS, Grossman JM, Cohen GR, Kemper NM, Pham HH, Are electronic medical records helpful for care coordination? Experiences of physician practices, J Gen Intern Med. 2010 Mar; 25(3):177-85. Epub 2009 Dec 22
18. MacPhail LH, Neuwirth EB, Bellows J, Coordination of diabetes care in four delivery models using an electronic health record, Med Care. 2009 Sep; 47(9):993-9.
19. Reiner BI, The challenges, opportunities, and imperative of structured reporting in medical imaging, J Digit Imaging. 2009 Dec; 22(6):562-8.
20. Schneider JH, Marcus E, Del Beccaro MA, Benson KA, D'Alessandro DM, Drummond WH, Handler EG, Kim GR, Leu MG, Lund GC, Zuckerman AE, Simonian MM, Spooner SA, Mansour J, Policy Statement--Using personal health records to improve the quality of health care for children, ., Pediatrics. 2009 Jul; 124(1):403-9.
21. Goroll AH, Simon SR, Tripathi M, Ascenzo C, Bates DW, Community-wide implementation of health information technology: the Massachusetts eHealth Collaborative experience J Am Med Inform Assoc. 2009 Jan-Feb;16(1):132-9. Epub 2008 Oct 24.
22. Pope C, Margolis J., Medical practice today, what members have to say, MGMA Connex. 2008 Jul; 8(6):17-23, 1 MGMA Communications and Marketing Department, USA. cpope@mgma.com.
23. Mechanic D, Rethinking medical professionalism: the role of information technology and practice innovations, Milbank Q. 2008 Jun;86(2):327-58., Institute for Health, Health Care Policy and Aging Research, Rutgers University, New Brunswick, NJ 08901, USA.

Original Article

Bakery products and their glycaemic indices in Jaffna.

Abstract

This study was aimed at evaluating of glycaemic index (GI) values of bakery products available in Jaffna such as bread, normal bun, butter cake, hard bun and rusk. The results will be helpful to Physicians and public to decide on the consumption of bakery products particularly by the diabetic and coronary heart disease patients. Healthy volunteers (22 Nos.) of 24.62 (±1.43) years, 63.42 (±10.50) kg body weights, 1.70 (±0.07) m height and 21.90 (±2.75) kg m^{-2} body mass index, were selected with their written consent. After overnight fasting, 75g glucose and each test food containing 75g digestible carbohydrate were administered at different instances and blood glucose levels were measured half hourly for two hours. The GI values were calculated and analyzed by Randomized Complete Block Design using SAS analytical package. The mean GI values of bread, normal bun, butter cake, hard bun and rusk were 68.59 (±3.74), 67.30(±5.03), 64.72 (±6.44), 52.78 (±3.40) and 50.30 (±4.85) % respectively. The GI values of bread, normal bun and butter cake differed significantly ($P<0.05$) from hard bun and rusk. The GI values of bread and normal bun did not differ significantly ($P>0.05$) among them. The GI values of rusk and hard bun did not differ significantly ($P>0.05$) among them. The results suggested that the hard bun and rusk are lower GI and bread, normal bun and butter cake are medium GI foods.

Introduction

Glycaemic index is defined as the incremental area under the blood glucose response curve elicited over a two-hour period by a 75g carbohydrate portion of a food, expressed as a percentage of the response to the same amount of carbohydrate from a standard food taken by the same subject (Anderson, 1997; Wolver, 1999). Food with a GI value of 70 or more are considered to be high GI diet, with an index value between 55 to 69 as medium GI diet and less than 55 as low GI diet (Mendosa, 2007).

Lower glycaemic foods in diet may decrease metabolic risk (Vrolix et al., 2008; Augustin et al., 2002). High glycaemic index is also associated with liver steatosis (Valtuena et al., 2006). The reduction and stabilization of blood glucose level can reduce the insulin demand and have beneficiary effects on insulin sensitivity, lipid profiles (Rettersol et al., 2009; Ebbeling et al., 2007) and β-cell functions (Amano et al., 2007). Reductions in hyperglycemia and hyperinsulinemia may also lower oxidative stress (Abete et al., 2008) which exacerbates a number of features of metabolic syndromes (Vrolix et al., 2008; Jenkins et al., 2002), including obesity (Brand-Miller et al., 2002), insulin resistance (Burani et al., 2006; Sheard et al., 2004; Villegan et al., 2007) inflammation and hypertension (Augustin et al., 2002; Sone et al., 2007). Prolonged exposure to hyperglycemia can promote macro- (Beulens et al., 2007) and micro vascular diseases (Augustin et al., 2002; Beulens et al., 2007; Apekey et al., 2009; Oh et al., 2005; Beera and Rizzo 2009; Mursu et al.,2009).

The objective of this study was to determine the GI values of frequently consumed bakery products by Jaffna inhabitants of Sri Lanka. The evaluation of GI will help the local public to decide the diets which have to be consumed particularly by the diabetic and coronary heart diseases patients. Hence, in this study the GI values of frequently consumed bakery products (bread, normal bun, butter cake, hard bun & rusk) were studied.

S.Pirasath, K.Thayananthan,
S.Balakumar, V.Arasaratnam
Faculty of Medicine, University of Jaffna

Materials and methods

Foods considered for this studies

The pure glucose (Royal Pure Glucose, Smithkline Beecham Pvt Ltd, Moratuwa), bakery products (bread, normal bun, butter cake, hard bun & rusk) were purchased from a local bakery.

Preparations of foods

The bread (454.0g) was prepared in appropriate amount of water with wheat flour (333.33g), Sugar (3.33g), margarine (4.66g), bread improver (2.0g) and yeast (1.33g) and was baked for 50 min.

The one pair of normal bun was prepared in appropriate amount of water with wheat flour (75.0g), milk powder (1.0g), sugar (22.50g) and margarine (2.50g) and was baked for 20 min. Hard bun was prepared in appropriate amount of water with wheat flour (633.33g), sugar (316.0g), margarine (50.0g) and salt (3.33g) and was baked for 20 min.

Butter cake was prepared using with wheat flour (250.0g), sugar (250.0g) margarine (250.0g), baking powder (10.0g) and eggs (No 06) and was baked for 30 min. Rusk was prepared in appropriate amount of water with wheat flour (834.20g), sugar (22.02g) margarine (14.66g), salt (12.32g) and yeast (14.66g) and was baked for 20 min.

Analysis of foods

All foods were analyzed for moisture (Pearson, 1976), fat (Pearson, 1976), soluble dietary fiber (SDF) (Megazyme, 2007), insoluble dietary fiber (Megazyme, 2007) & total dietary fiber (TDF) (Megazyme, 2007) contents.

Selection of subjects

A group of 22 healthy volunteers between 20 to 24 years old was selected and the weight & height were determined and body mass index were calculated. The volunteers who had abnormal glucose tolerance, underweight or overweight, dieting or restricting their carbohydrate intake, suffering from any illness or food allergy were excluded from the studies.

Ethical clearance

The ethical clearance for this study was obtained from the 'Ethical Review Committee', Faculty of Medicine, University of Jaffna.

Estimation of blood glucose level of volunteers The blood samples were collected and measured using semiautomated biochemical analyzer (TC 3300).

Calculation of glycaemic response and glycaemic index values Glycaemic index and glycaemic response were calculated (Wolver et al., 1991).

Statistical analysis

Glycaemic response and glycaemic index values of different types of bakery products were analyzed by Randomized Complete Block Design (RCBD) using SAS analytical package.

Results

The mean age, weight, height and body mass index were 24.62 (±1.43) years, 63.42 (±10.50) kg, 1.70 (±0.07) m 21.90 (±2.75) kg m^{-2} respectively.

When 75g of glucose was orally administered to the volunteers, the blood glucose level reached the peak value at 30min. The mean fasting blood glucose level was 84.81 (±4.37) mg dL^{-1} and the mean blood glucose level at 30min was 147.43 (±11.67) mg dL^{-1}. The mean peak glycaemic response for pure glucose was 62.62 (±11.45) mg dL^{-1}. This value was considered to find out the GI of test foods.The proximate compositions of the different bakery products available in Jaffna are given in Table 1.

After overnight fasting (12h) 75g digestible carbohydrate containing test foods were administered to volunteers on separate days. All foods exposed peak glycaemic response at 30min.

The mean glycaemic response values of bread, normal bun, butter cake, hard bun and rusk were 43.0 (±2.32), 42.2 (±3.15), 40.5 (±4.30), 33.1 (±3.39) and 31.1 (±3.03) % respectively (Table 2).

The mean GI values of bread, normal bun, butter cake, hard bun and rusk were 68.59 (±3.74), 67.30 (±5.03), 64.72 (±6.44), 52.78 (±3.40) and 50.30 (±4.85) % respectively (Table 3).

Discussion

When fiber contents of the bread and normal bun were considered, bread contained less soluble dietary fiber (0.50%) and more insoluble dietary (2.73%) and total dietary fibers (3.23%) (Table 1), than normal bun (0.56, 2.43 and 2.99% respectively).The SDF, IDF and TDF of wheat bread and normal bun did not show much difference. Even though, bread contains higher IDF, the mean glycaemic response to bread is higher than the normal bun (Table 2). It could be due to the less amount of SDF than that in the normal bun. Thus

the SDF has more influence on glycaemic response than IDF. The fat contents of bread and normal bun were 1.40 (±0.08) and 3.01 (±0.04) respectively. Thus there was an influence of the fat contents on the glycaemic response after the consumption of these two foods.

When fiber contents of the normal bun and hard bun were considered, the hard bun contained more soluble dietary fiber (0.76%), insoluble dietary fiber (2.99%) and total dietary fiber (3.75%) than the normal bun (0.56,2.43 and 2.99%) (Table 1). The mean glycaemic response to normal bun is higher than the hard bun. This could be due to the effects of SDF, IDF and TDF in these buns. The fat contents of normal bun and hard bun were 3.01 (±0.04) and 1.69 (±0.05) % respectively (Table 1). Thus fat contents of the normal bun and hard bun did not show any direct relationship with the glycaemic index values.

The SDF (0.60%), IDF (2.57%) and TDF (3.17%) (Table 1) contents of the butter cake were higher than the bread and normal bun. The mean glycaemic response to the butter cake was 40.53 (±4.03) mg/dL and this was lower than that those to bread and normal bun, and this could be due to the higher dietary fiber contents. The fat content of the butter sake was higher than other bakery products (5.11%). But the influence of fat content did not show any direct relationship.

Rusk contained more soluble dietary fiber (0.87%), insoluble dietary fiber (3.18) and total dietary fiber (4.05%) than other bakery products. Among all the bakery products, rusk gave lowest glycaemic response. According to these results, it could be suggested that the soluble dietary fiber content highly influence on the glycaemic response than insoluble dietary fiber content.

The glycaemic response of glucose was significantly ($P<0.05$) higher than glycaemic response of bread , normal bun, butter cake, hard bun and rusk. The glycaemic response and glycaemic index value of bread differed significantly ($P<0.05$) from butter cake, hard bun and rusk and did not differ significantly ($P>0.05$) from normal bun. The glycaemic response and glycaemic index value of normal bun differed significantly ($P<0.05$) from hard bun. The glycaemic response and glycaemic index value of butter cake differed significantly ($P<0.05$) from bread, hard bun and rusk and did not differ significantly ($P>0.05$) from normal bun. The glycaemic response and glycaemic index value of rusk differed significantly ($P<0.05$) from bread, normal bun and butter cake and did not differ significantly ($P>0.05$) from hard bun.

These results showed that hard bun and rusk are better choice for those who need loe glycaemic index diets than bread, normal bun and butter cake. However recommendation of these types of bakery products should be made after analyzing the glycaemic load, energy, protein and fat contents.

Chulp et al. (2004) have showed that the blood glucose level reached at 30 and 60 min for white bread in mean glucose curve after the consumption of 85.0g of white bread consumed for breakfast and dinner respectively The mean glycaemic index value of white bread was 70.3%. Williams and Stubbs (2007) reported that glycaemic index values of white bread (1 oz slice), bagel bread (1 each,2 oz), sourdough bread (1.5 oz slice),100% stone-ground whole wheat bread (1.5 oz slice) and rye bread (1 oz slice) were 73.0, 72.0, 54.0, 53.0 and 52 respectively.

The mean glycaemic index value of bread [68.59 (±3.74) %] was closer to that of bread from Canada [69.0(±5.0) %)], USA (70.0%) and Australia (70.0%) (Foster-Powell et al., 2002). The mean glycaemic index value of the normal bun [67.30 (±5.03) %] was slightly higher than the hamburger bun (61.0%) from Canada (Foster-Powell et al., 2002).

Conclusion

The hard bun and rusk are lower GI diets (GI values < 55%). Bread, normal bun and butter cake are medium GI diets (GI values between 55-70%). Further studies have to be carried out on the prediabetes and diabetes.

Acknowledgement

Financial assistances by University of Jaffna and Thilliampalam Kanagasabai research fund.

Table 1: Proximate compositions of different bakery products available in Jaffna, Sri Lanka.

Constituents (%)	Bread	Normal bun	Hard bun	Butter cake	Rusk
Moisture	33.11(±1.01)	26.43(±0.34)	9.30(±0.14)	22.18(±0.67)	5.39(±0.12)
Fat	1.4(±0.08)	3.01(±0.04)	1.69(±0.05)	5.11(±0.04)	0.97(±0.01)
Soluble dietary fiber (SDF)	0.50(±0.02)	0.56(±0.03)	0.76(±1.01)	0.60(±0.02)	0.87(±0.01)
Insoluble dietary fiber (IDF)	2.73(±0.10)	2.43(±0.07)	2.99(±0.07)	2.57(±0.06)	3.18(±0.05)
Total dietary fiber (TDF)	3.23(±0.15)	2.99(±0.07)	3.75(±0.09)	3.17(±0.10)	4.05(±0.08)
Total digestible Carbohydrate	52.80(±0.98)	60.58(±0.87)	77.98(±0.45)	67.61(±0.56)	86.27(±0.98)

Table 2: Glycaemic response of different bakery products at 30 and 60 min.

Glycaemic response (mg/dL)		
Bakery products	**30 min**	**60 min**
Bread	43.0 (±2.32)	23.7 (±5.86)
Normal bun	42.2 (±3.15)	23.3 (±5.86)
Hard bun	33.1 (±3.39)	19.7 (±1.53)
Butter cake	40.5 (±4.30)	22.7 (±4.04)
Rusk	31.1 (±3.03)	22.3 (±3.21)

Table 3: Exact amount of different bakery products consumed and their glycaemic index value

Bakery products (Amount consumed in g)	Total digestible Carbohydrate (g)	Total dietary fiber (g)	Soluble dietary fiber (g)	Insoluble dietary fiber (g)	Fat (g)	Glycaemic Index (%)
Wheat flour bread (142.05)	75.00	4.59	0.71	3.88	1.99	68.59(±3.74)
Normal bun (123.8)	75.00	3.92	0.69	3.01	3.73	67.30(±5.03)
Hard bun (96.18)	75.00	3.61	0.74	2.88	1.62	52.78(±3.40)
Butter cake (110.93)	75.00	3.52	0.67	2.85	5.67	64.72(±6.44)
Rusk (86.93)0	75.00	3.52	0.76	2.76	0.84	50.30(±4.85)

References

1. Abete, I., D. Parr and J.A.Martinez, 2008. Energy-restricted diets based on a distinct food selection affecting the glycaemic index induce different weight loss and oxidative response. Clin. Nutr., 27: 545 551.
2. Amano, V., M. Sugiyama, J.S. Lee, K.K. Kawakubo, K. Mori, A.C. Tang and A. Akabayashi, 2007. Gylcemic index-based nutritional education improves blood glucose control in Japanese adults. Diabetes Care., 30 (7): 1874 1876.
3. Anderson, G.H., 1997. Sugars and health. Nutr. Res., 17(9):1485 1498.
4. Augustin, L.S., S. Franceschi , D.J.A. Jenkins, C.W.C. Kendall and C.L. Vecchia, 2002. Glycaemic index in chronic disease. Eur. J. Clin. Nutr., 56: 1049 1071.
5. Apekey, T.A., A.J.E. Morris, S.Fagbemi and G. J. Griffiths , 2009. Effect of low-fat and low GI diets on health. Nutr. Food Sci., 39(6): 663-675.
6. Berra, B. and A.M.Rizzo, 2009. Glycaemic index, glycaemic load, wellness and beauty: the state of the art. Clin. Dermatol., 27: 230-235.
7. Beulens, J.W.J., L.M.de Bruijne , R.P.Stolk, P.H.M. Peeters,M.L. Bots, D.G. Grobbee and Y.T. Vander Schouw,2007. High dietary glycaemic load and glycaemic index increases risk of cardio vascular disease among middle aged women. J. Am. Coll. Cardiol., 50 (1): 14-21.
8. BrandMiller, J.C., S.H.A. Holt , D.P. Powlak and J. McMillan ,2002. Glycaemic index and obesity. Am. J. Clin. Nutr.,76 (Suppl.): 281S 285S.
9. Burani, J. and P.J. Lango, 2006. Low Glycaemic Index carbohydrates: An effective behavioral change for glycaemic control and weight management in patients with Type 1 and 2diabetes. The Diabetes Educ., 32(1): 78-88.
10. Chlup R, Bartek J, Reznickova M. Zapletalova J, Doubravova B, Chlupova L, Seckar P, Dovorackova S, Simanek V. Determination of glycaemic index of selected foods (white bread and cereal bars) in healthy persons. Biomed Papers 2004; 148(1): 17-25.
11. Ebbeling, C.B., M.M.Leidig , H.A.Feldman , M.M.Lovesky and D.S. Ludwig,2007. Effects of low glycaemic load vs. low fat diets in obese young adults: A randomized trial. J. Am. Med. Assoc ., 297 (19): 2092 - 2102.
12. Foster-Powell, K., S.H.A. Holt and J.C.Brand miller, 2002.International table of glycaemic index and glycaemic load values. Am. J.Clin. Nutr.,76:5-56.

13. Jenkins, D.J.A., C.W.C.Kendall , L.S.A.Augustin , S.Franceschi , M.Hamidi , A.Marchie , A.L.Jenkins and M. Axelsen,2002. Glycaemic index: Overview of implications in health and disease. Am. J.Clin. Nutr ., 76 (Suppl.): 266S 273S.
14. Megazyme., 2007.Total dietary fibre assay procedure. http://www.megazyme .com/booklets/KTDFR.pdf, 2007.
15. Mendosa D.,2007. Glycaemic Index. http://www.mendosa.com/gi:htm.
16. Mursu, J., J.K.Virtanen , T.H.Rissanen , T.P.Tuomainen , I.Nykanen , J.A.Laukkanen , R.Kortelainen and S.Vaulilainen,2009 . Glycaemic index, glycaemic load, and the risk of acute myocardial infarction in Finnish men: The Kuopio ischaemic heart disease risk factor study. Nutr. Metabolism Cardiovasc. Dis ., 20: 1-6.
17. Oh, K., F.B.Hu , E.Cho , K.M.Rexrode , M.J.Stampfer , J.E.Manson , S.Liu and W.C.Willelt,2005. Carbohydrate intake, glycaemic index, glycaemic load, and dietary fiber in relation to risk of stroke in women. Am. J. Epidemiol ., 161(2): 161 169.
18 Pearson, D., 1976 editors. The Chemical analysis of foods. 7th ed. Edinburgh, London, Churchill. Livingstone.
19. Retterstol, K., C.B.Hennig and P.O. Jversen ,2009. Improved plasma lipids and body weight in overweight / obese patients with type III hyperlipoproteinemia after 4 weeks on a low glycaemic diet. Clin. Nutr ., 28: 213 215.
20. Sheard, N.F., N.G.Clark ,J.C. Brand-Miller , M.J.Franz ,F.X. Pi-Sunyer , E.MayerDavis , K.Kulkarni and P.Geil ,2004. Dietary carbohydrate (amount and Type) in the prevention and management of diabetes : A statement by the American Diabetes Association. Diabetes Care ., 27 (9): 2266 2271.
21. Sone, H., Y.Yoshimura ,S. Tanaka , S.Iimuro , Y.Ohashi , H.Ito , H.Seino , S.Ishibashi , Y.Akamuna and N.Yamada,2007. Cross-sectional association between BMI, glycaemic control and energy intake in Japanese patients with type 2 diabetes: Analysis from the Japan diabetes complication study. Diabetes Res. Clin. Pract ., 775: S23-S29.
22. Valtuena, S., N.Pellegrini , D.Ardigo ,D.D. Rio , F.Numeroso , F.Scazzina , L.Monti , I.Zavaroni and F.Brighenti,2006 . Dietary glycaemic index and liver steatosis. Am. J. Clin. Nutr ., 84: 136 142.
23. Villegan, R., S.Liu , Y.T.Gao , G.Yang , H.Li , W.Zheng and X.O. Shu,2007. Prospective study of diatery carbohydrates glycaemic index, glycaemic load and incidence of type 2 diabetes mellitus in middle-aged Chinese women. Achiev. Int. Med ., 167 (21): 2310 2316.
24. Vrolix, R., L.E.C.Van Meijl and R.P. Mensink,2008. The metabolic syndrome in relation with the glycaemic index and the glycaemic load. Physiol. Behav ., 90: 293 299.
25. Williams, S. and P.Stubbs ,2007. Theglycaemicindex. http://extension.usu.edu/files/ publications/factsheet/FN2004-01.pdf,2007
26. Wolver, T.M.S., 1999. Dietary recommendations for diabetes: high carbohydrate or high monounsaturated fat? Nutr. Today ., 34(2): 73-77.
27. Wolever, T.M.S., D.J.A.Jenkins,A.L.Jenkins and R.G.Josserg,1991. The Glycemic Index:Methodology and Clinical Implications.The Am. J. Clin. Nutr .,54:846- 854

Case report

Chloroquine resistant Plasmodium vivax in Jaffna First reported case in Srilanka

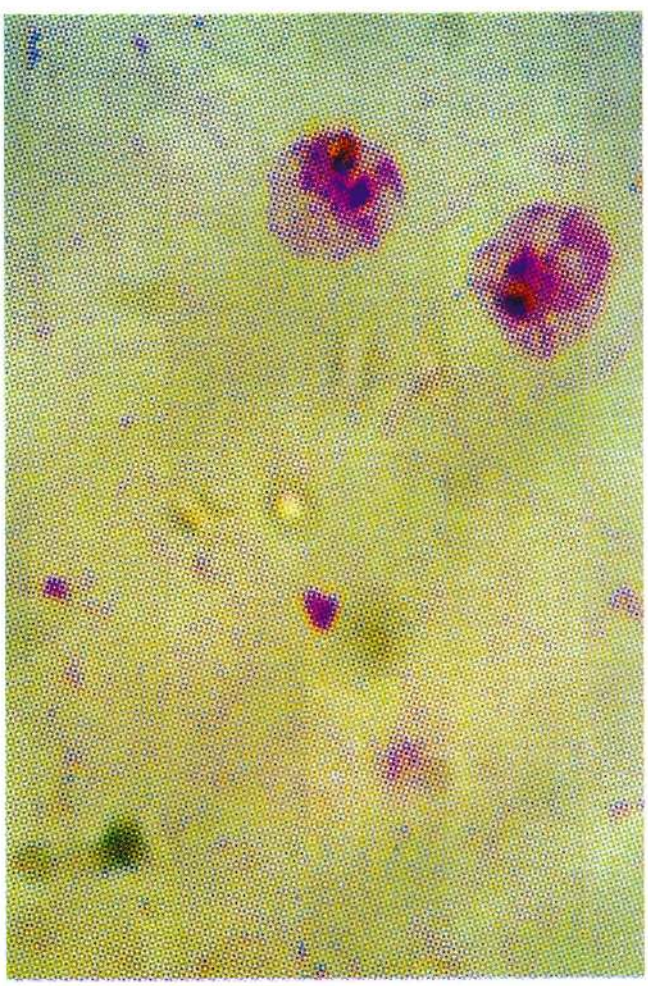
Case report

A 64 year old gentleman with a past history of ischemic heart disease was admitted with fever of 4 days duration following a recent travel to Tamil Nadu India three weeks back. It was a high spiking fever with chills, rigors and sweating and associated with cough and pleuritic chest pain. Examination revealed a febrile ill looking gentleman with dyspnoea without cyanosis with bilateral basal crepitations with the oxygen saturation being 92% in room air. As the treatment for a community acquired pneumonia was not responding, a prompt search for the possibility of malaria was carried out considering his clinical picture and his recent visit to India. Blood film revealed plasmodium vivax late trophocytes and gametocytes. The patient failed to respond to the standard Chloroquine therapy. Coartem was started according to the standard practice on the following day of completion of Chloroquine therapy on 0, 8, 24, 36, 48 & 60 hours. The patient had an uneventful recovery and discharged on the t 9th day of hospital admission. This is the first reported case of Chloroquine resistant plasmodium vivax found in Srilanka.

Discussion

Plasmodium vivax is the most widely distributed human malarial parasite with an at risk population of 2.5 billion persons. Although the exact burden of disease caused by *P. vivax* infection is still a matter of debate, this parasite causes approximately 100300 million clinical cases each year (1). *P vivax* mono infection could also be involved in multiple organ dysfunction and severe life- threatening disease as seen in *P. falciparum* infection (2). In Srilanka P. falciparum, resistant to Chloroquine was first reported in 1984(3) A study in the northern Srilanka highlighted the prevalence of Chloroquine resistant falciparum malaria among security force personnel in 2004(4) The first Chloroquine resistant isolates of *P vivax* were reported from Papua, Indonesia, and Papua New Guinea, and cases have also been reported from Myanmar, India and South America. (5, 6, 7) This case indicates the travel related transmission of resistant Plasmodium vivax infection to Srilanka.

Giemsa stain showing P. vivax parasite.



K.G.Ajith Kumara[1], G.J.Pratheepan[2], J.A.Pradeepan[3], T.Kumanan[4]
[1]Registrar, [2]Senior Registrar, [3] Lecturer
[4] Senior Lecturer, Department of Medicine, Teaching Hospital Jaffna.

References

1. Barbara S, 2002. Focus on *Plasmodium vivax. Trends Parasitol 18:* 287289.
2. Piscot S, 2006. Is *Plasmodium vivax* still a paradigm for uncomplicated Malaria? *Med Mal Infect 36:* 406413.
3. Ratnapala R, Subramaniam K, Yapabandara MGM, Fernando WP. Chloroquine resistant *Plasmodium falciparum* in Sri Lanka. *Sri Lanka Medical Journal* 1984; 29: 135-45.
4. Hapuarachchi HA, Dayanath MY, Abeysundara S, Bandara KB, Abeyewickreme W, de Silva NR. *Chloroquine resistant falciparum malaria among security forces personnel in the Northern Province of Sri Lanka.* Ceylon Med J. 2004 Jun; 49(2):47-51.
5. Rieckmann KH, Davis DR, Hutton DC. *Plasmodium vivax* resistance to chloroquine? *Lancet*. 1989; 334:118384.
6. Baird JK, Basri H, Purnomo, et al. Resistance to chloroquine by *Plasmodium vivax* in Irian Jaya, Indonesia. *Am J Trop Med Hyg*. 1991; 44:54752.
7. Dua VK, Kar PK, Sharma VP. Chloroquine resistant *Plasmodium vivax* malaria in India. *Trop Med Int Health*. 1996; 1:81619.

Classes of recommendations

Classes of Recommendations	Definition
Class I	Evidence and/or general agreement that a given treatment or procedure is beneficial, useful, and effective.
Class II	Conflicting evidence and/or a divergence of opinion about the usefulness/efficacy of the given treatment or procedure.
Class IIa	Weight of evidence/opinion is in favour of usefulness/efficacy.
Class IIb	Usefulness/efficacy is less well established by evidence/opinion.
Class III	Evidence or general agreement that the given treatment or procedure is not useful/effective, and in some cases may be harmful.

Level of evidence

Level of Evidence A	Data derived from multiple randomized clinical trials or meta-analyses.
Level of Evidence B	Data derived from a single randomized clinical trial or large non-randomized studies.
Level of Evidence C	Consensus of opinion of the experts and/or small studies, retrospective studies, registries.

Clinical Practice

Distraction Osteogenesis
A novel technique for limb lengthening

Limp length discrepancy (LLD) is a major burden to patients with incapacitating limps and challenges surgeons with management. Distraction osteogenesis (DO) is one of the method to treat patients with bone loss & LLD. We have summarized our experience with DO and identified the determinants of the management and outcome (including long-term functional outcome) of the patients.

This is an observational descriptive retrospective case-series of 16 patients underwent DO from 2005 to 2009 in Teaching hospital Jaffna. The data were collected by interviewer-administered questionnaires and Data-Extraction-form. Lower limb functions were assessed with Lower Extremity Functional Scale (LEFS) questionnaires.

Out of 16 (tibia 8, femur 8) patients 9(56%) were males. Injury due to firearm were in 7 (44%) and road traffic crash (RTC) were in 4 (25%). Three (19%) of them were smokers. Ultimate lengthening reached average of 9.8 cm. Six out of sixteen patients developed pin tract infection and treated with antibiotics. Lower limb functions were assessed after a mean follow-up of 2.4 ± 1.02 years. Overall function of lower extremity in patients underwent segmental bone transport was 'a little bit of difficulty' and compression distraction was 'moderate difficulty'. In posttraumatic injuries (RTC and firearm) functional results were good or excellent. The average LEFS score for posttraumatic injuries is 55(Moderate difficulty), for non traumatic cases is 38(quite a bit of difficulty).

S.Sashikaran
Registrar in orthopaedic Surgery
National Hospital Srilanka.

K.Umapathy
Consultant Orthopaedic Surgeon
Teaching Hospital Jaffna.

Non union with bone loss occur in various ways. It can be due to Open fractures (with Segmental fracture with extrusion of fragments Post debridement or Blast injuries), Congenital defects, Infection, Tumour resection, Osteonecrosis, and Failed arthroplasty. But the main cause of bone defect is high energy trauma such as gun shot or blast injuries. Which leads to significant segmental bone loss directly or due to the consequence of it complications. This gives a major burden to the patient with deformed incapacitating limps and a challenging to surgeon in order to manage the condition. There are several methods in current orthopaedic practice to manage a bone defect efficiently. These techniques include

- Grafting the bone to the defect (Auto, allo, xenograft),
- Use the technique called Distraction osteogenesis,
- Use of Biomaterials to fill the defect such as Demineralised bone matrix and Ceramics and
- Fixation of Prosthetic implants.

In these treatment modalities Distraction osteogenesis is the one method, which is used to treat the patients with non-union with significant bone loss with limp length discrepancy.

By definition "Distraction osteogenesis is a de novo production of bone between corticotomy surfaces which undergoing gradual distraction". Distraction osteogenesis, also called callus distraction, callotasis and osteodistraction; is a slow, gradual and controlled stretching of the bony callus after a low energy subperiosteal corticotomy to fill the bony defects. The basic principles of this procedure is to create an environment of a combination of conditions based on maximum preservations of blood supply, osteogenic elements and stable fixation coupled with appropriate rate and rhythm of distractions. According to the "Law of Tension Stress" on tissue regeneration, the gradual traction on callus stimulates osteogenesis and growth and regeneration of skin, muscles, nerves and blood vessels. (1, 2)

In 1905, Alessandro Codivilla introduced surgical practices for lengthening of the lower limbs. Early techniques had a high number of complications, particularly during healing, and often resulted in a failure to achieve the goal of the surgery. (3)The breakthrough came with a technique introduced by Russian orthopedic surgeon Gavril Ilizarov. Ilizarov pioneered in this treatment technique with a modular ring fixator and transosseous wires to stabilize the bone fragment and this technique carry his name. This technique reduced the frequency and severity of the complications, made the surgery safer, and allowed the goal of lengthening the limb to be achieved.

This technique can be classified according to the mode of transport as

- Compression distraction
- Bone transport.

In compression distraction the limb is shortened by compression at fracture site and distracted from a separate corticotomy site or a short limb is distracted by a corticotomy. This allows for reduction of the size of the soft tissue defect, and will often allow delayed primary closure or skin grafting of a defect that otherwise would require a soft tissue flap. In Bone transport the defect is closed by transportation of a bone segment but the limb length remain same. The advantage is that the limb can be functional, even weight bearing, during the process. It is also a useful simple and effective method in lengthening with immediate correction of deformity or angulation. These mode of transport can be used separately or combined (Bifocal lengthening) for large limb length discrepancies. (4, 5,6)

Although distraction osteogenesis is most often used in the treatment of post-traumatic injuries, it is increasingly used to correct limb discrepancies caused by congenital conditions and old injuries.

Patients and methods

Ethical approval for this study was granted through the Committee on Ethical Review of faculty of medicine, University of Jaffna.

This is an observational descriptive retrospective case-series on 16 patients underwent DO from 2005 to 2009 in Teaching hospital Jaffna. The data were collected by interviewer-administered questionnaires and Data-Extraction-forms. Lower limb functions were assessed with Lower Extremity Functional Scale (LEFS) questionnaires.

Results

Out of 16 patients 9(56%) were males and 7 (44%) were females. Tibia was involved in 8 patient (50%) and femur in 8 patients (50%). Bony defects due to firearm injury were in 7patients (44%) and road traffic accidents were in 4 patients (25%) (Figure 1).Out of 16 patients 3(19%) of them were smokers.(Table 1)

The ultimate bone lengthening in our group of patients reached an average of9.8 cm, with a minimum of 5.8cm and maximum of 14.9cm.The fixators were in place for an average of 13.3 months. The mean follow-up was 2.4 ± 1.02 years.(Table2)

Bone transport

Bone transport was performed in five patients: one femur and four tibias. The median age was 31years (range 1750 years) in 1 female and 4 male patient. The mean amount of lengthening was 7.5 cm for femoral and 10 cm for tibial distraction osteogenesis. The mean healing index was 5.3 months/cm in the femur and 1.18 ± 0.22 month/cm in the tibia. The mean external fixation index was 5.3 months/cm in the femur and 1.18 ± 0.22 month/cm in the tibia. The average LEFS score was 64 ± 11 points.

Compression distraction

Leg lengthening by Compression distraction was performed in eleven patients: seven femurs and four tibias. The median age was 28years (range 762 years) in six female and five male patients. The mean amount of lengthening was 9.9± 3.3 cm for femoral and 10.1± 1.43 cm for tibial distraction osteogenesis. The mean healing index was 1.5 ± .92 months/cm in the femur and 1.12± 0.35 month/cm in the tibia. The mean external fixation index was 1.39 ± 0.91 months/cm in the femur and 1.02 ± 0.33 month/cm in the tibia.The average LEFS score was 45.09± 20.9 points.

Outcome

In our patients over all bony and functional results are summarized in figure 2 and 3 respectively.

In patient underwent bone transport, the function of the affected leg was rated excellent in three patients, and good in two patients. The bony result is rated as excellent in all patients. The average LEFS score was 64± 11 points.

In patient underwent compression distraction, the function of the affected leg was rated excellent in three patient, good in five, fair in one and poor in two patients. The bony result is rated as excellent in nine patients, Fair in one & Poor in one patient .The average LEFS score were 45± 20.9 points.

In patients with non traumatic aetiology, the function of the affected leg was rated excellent in one patient, good in two patients fair in one and poor in one patient. The bony result is rated as excellent in four patients and fair in one patient. The average LEFS score was 39.8 ± 19.5 points.

In patient with traumatic aetiology of bone defect, the function of the affected leg was rated excellent in five patients, good in five patients and poor in one. The bony result is rated as excellent in ten patients and poor in one patient. The average LEFS score was 56± 19.1 points. (Table 3)

Complications

During the distraction, common problem was pain in ten out of 16 patients and swelling of leg in 4 patients. There were a total of 18 true complications found in this study. 3 patients limp lengthening were not associated with a complication; ten were associated with one complication; four, with two complications; and one, with three complications. The complications included pin site inflammation (six), mild to moderate contracture of knee or ankle(four), Residual shortening (three), Foot become equinus in position (three),fracture of the lengthened bone (one), Axial deviation (one) and Delayed union(one).

Discussion

Distraction osteogenesis has four phases (figure 4 & 5) named as

- Latency period/ Lag phase
- Distraction phase
- Hardening/ Consolidation Phase

The duration of the latency period in this study was ranged from 10 to 14 days. The latency period in most published clinical studies has ranged from 3 to 10 days. It varies depending on type and gentleness of osteotomy. This allows the fracture healing mechanisms to start.

The bone segments were distracted with a rate of one millimetre per day in four 0.25 mm increments in distraction phase. In literature optimal distraction rate varies from 0.3 mm/day to 1.5 mm/d. If the increments of distraction too large it may exceed the potential for vascular ingrowth at a diaphyseal site and inhibit osteogenesis. If the rate of distraction is too slow the gap may close by normal fracture healing. Any instability producing shear stress inhibits osteogenesis. In this phase the tissue between the cut bone surfaces develops into a bipolar fibro vascular zone with collagen fibers oriented parallel to the direction of pull. Bone begins to form by intra membranous ossification arising from the full width of the cut bone surface. This bone forms in a highly uniform, ordered fashion of columns or cones about 200 microns in diameter, surrounded by microvasuclar channels. Mineralization proceeds in proximity to the vessels, which grow parallel to the distraction force.Low intensity pulsed ultrasound stimulation (LIPUS) may used as a biophysical stimulation to accelerate the formation and maturation of regenerate bone during lengthening.(13)

Hardening/ Consolidation Phasemay take as much as twice as long as the distraction phase. At this time the soft bone that forms now hardens and a cortical tube is allowed to form. In this period careful radiological assessment of the patterns of callus formation is a useful method for the evaluation and monitoring of regenerate bone.(14) Transplantation of culture-expanded bone marrow cells (BMC) and platelet-rich plasma (PRP) during limb lengthening shorten the treatment period by accelerating callus formation.(15, 16)

After completion of the consolidation phase the fixator will be dynamized and the patient allowed the full weight bearing. The pixel value ratio (ratio of pixel value of regenerate versus the mean pixel value of adjacent bone) on radiographs can be an objective parameter for callus measurement and may provide guidelines for the timing of external fixator removal. (17)

All our segmental transport patients underwent an additional operation for autogenous bone grafting to allow consolidation at the docking site.

Assessment of a patient undergone limb lengthening is difficult due to the absence of a single standard measuring technique. Though there are several techniques used by various author for assessing the patient under gone distraction osteogenesis.

The healing index (HI) was defined as the time (days) needed for consolidation per cm of distracted osteotomy site (days/cm). Consolidation time (CT) was the time (days) between the end of distraction osteogenesis and total consolidation or removal of fixator. The external fixation index is calculated as the duration of external fixation in months divided by the total amount of bone transported and/or the amount of lengthening in centimetres. (24)

Calluses formed during limb lengthening were classified radiographically into 6 types: external, straight, attenuated, opposite, pillar, and agenetic. The healing indexes correlated well to the intrinsic periosteal and endosteal conditions of each type. This classification enabled us to estimate the intrinsic conditions, predict the healing index, control the daily lengthening speed, and decide to apply early augmentation of the callus.(25)

Recently developed Quantitative evaluation of regenerate using the regeneration formation index based on classic radiography and computer assisted image analysis, allows to precisely analyzing the course of distraction osteogenesis, particularly to precisely estimate the timing of fixator removal and avoid complications.(26)

Patient with Distraction osteogenesis also can be assessed by the functional and bony scoring described by Paley et al. Bony scoring system consists of 4 Criteria

1. Bony union
2. Absence of infection
3. Deformity less than 7 °
4. Length discrepancy less than 2.5cm.

An Excellent result consist of all four criteria, Good = union + two, Fair = union + one Poor if non-union only.

Functional scoring system consist of 5 Criteria's, these are

1. Noteworthy limp
2. Joint stiffness- knee, ankle
3. Soft tissue sympathetic dystrophy
4. Pain that reduces activity
5. Inactivity

An Excellent result consist of active pt, with none of the above, Good-active + one /two of other criteria, Fair-active + three/four criteria Poor-inactive and Poor means inactive(22)

Long-term functional outcomes can be evaluated with the Lower Extremity Functional Scale (LEFS). The LEFS is a 20-item self-report measure of physical function. Each item is rated on a five-point scale (04), with lower scores representing greater difficulty. Total scores can range from 0 to 80. Function is defined as follows: extreme difficulty or unable to perform activity (019points),quite a bit of difficulty (2039 points), moderate difficulty (4059 points), a little bit of difficulty (6079points),and no difficulty (80 points).(5)

After a mean follow-up of 2.4 (± 1.02) years, the quality of life and lower limb function were assessed with Lower Extremity Functional Scale (LEFS) questionnaires.(Figure 6) Many factors determine the management and outcome of this technique including the initial injury, patients' conditions, and meticulous treatment techniques. Some socioeconomic factors also take some part.(21) A study of 107 patients with fracture non-union shows Interestingly 11 out of 12 problem cases were in smokers(22) It may due to the nicotine which can cause delays in the process of healing by its negative effect on the mineralization of the regenerate.(23) In our patients the results of the LEFS questionnaire showed overall function of the lower extremity to be in the 'a little bit of difficulty' class for patients who underwent segmental bone transport and in 'moderate difficulty' for those who underwent Compression distraction. The functional results good or excellent in DO perform in deformity or Limp Length Discrepancy due to Road Traffic Crash (RTC) or Firearm injuries patients. Poor in osteomyelities or in correction of congenital deformities. The average LEFS score for injury due to RTC is 56 (Moderate difficulty), injury due to firearm without osteomyelities is 55.8(Moderate difficulty).These results indicate these patients, despite the severity of the initial injuries and prolonged treatment, have only moderate difficulties in daily activities including hobbies.

The complications included coronal axial malalignment, severe contracture or subluxation of the joints, fracture of the lengthened bone, severe pin-track infection at the site of the distraction, premature consolidation and non-unions or Delayed union. Foot equinus and Residual shortening also reported in some cases.(18, 19)In this case series, six out of sixteen patients developed a pin tract infection and treated

with antibiotics. The prevalence of pin tract infections in literature has been reported to be as high as 95%. Exposure to a series of radiographic examinations to follow the lengthening process and callus formation is a hazards to the patient. But a level II evidence suggest as the average patient's exposure during a limb-lengthening procedure is tolerable, but femur lengthening results in a higher cumulative organ dose.(20)

The limitation in interpreting our results is the small sample group (which is a case series).

Conclusion

In conclusion, functional outcome returned to near normal after post-traumatic distraction osteogenesis of the lower limb than DO for non traumatic conditions. Bone transport gave better functional outcome in bone transport than compression distraction.

Table-1 Demography and injury details

Case	Gender	Age at Distraction procedure	Aetiology of the bon defect/ Shortening	Site of injury	Smoker/ Nonsmoker	Duration of Nonunion (months)	No of Surgical procedure before DO	LLD	Bone defect(cm)
1	Male	50	Gunshot injury	Femur	Smoker	17.4	5	7	7.5
2	Male	22	RTA	Tibia	Smoker	1.2	1	12	12.2
3	Female	37	RTA	Femur	Non Smoker	144.2	3	8	9.6
4	Female	40	RTA	Femur	Non Smoker	16. 6	4	10	13.3
5	Male	62	Gunshot injury & chronic osteomyelities	Femur	Non Smoker	26	5	10	14.9
6	Male	34	RTA	Tibia	Non Smoker	31.4	1	6	8
7	Female	15	Congenital malformation	Tibia	Non Smoker	185.5	1	10	10.8
8	Male	13	Chondrodysplasia	Tibia	Non Smoker	44.2	1	14	10.4
9	Female	28	Polio myelities	Tibia	Non Smoker	309.2	0	4	11.2
10	Male	13	Blast injury	Femur	Non Smoker	146.5	3	9.5	10
11	Female	7	OsteogenesisImperfecta& fracture	Femur	Non Smoker	82.1	3	7	6.5
12	Male	31	Blast injury	Tibia	Smoker	10.4	2	10	9.4
12	Female	17	Pseudoarthrosis of tibia	Tibia	Non Smoker	174.2	6	14	11.6
14	Male	28	Blast injury	Femur	Non Smoker	11.1	2	11	15.9
15	Female	31	Blast injury	Femur	Non Smoker	11	5	5	5
16	Male	38	Blast injury	Tibia	Non Smoker	10.4	1	7	8

Table-2 Details of surgery

Case	Type	Mode	Duration of Fixator wear (month)	distraction Velocity	Bone Grafting at Docking Site	Length gain	Total Duration Of Treatment
1	Lengthening	transport Bone	39.23	1mm/day	Yes	7.5	40.23
2	Lengthening	transport	16.43	1mm/day	Not done	12.2	17.43
3	Lengthening	Compression Distraction	31.80	1mm/day	Not done	9.6	32.80
4	lengthening	Compression Distraction	12.10	1mm/day	Not done	13.3	13.10
5	Lengthening	Compression Distraction	10.60	1mm/day	Not done	14.9	11.60
6	Both lengthening & Axis correction	Compression Distraction	11.60	1mm/day	Not done	8	12.60
7	Both lengthening & Axis correction	Compression Distraction	9.23	1mm/day	Not done	10.8	10.23
8	Lengthening	Compression Distraction	7.13	1mm/day	Not done	10.4	8.13
9	Lengthening	Compression Distraction	12.23	1mm/day	Not done	11.2	13.23
10	Lengthening	Compression Distraction	9.00	1mm/day	Not done	10	10.00
11	Lengthening	Compression Distraction	9.67	1mm/day	Not done	6.5	10.67
12	Lengthening	Bone transport	8.37	1mm/day	Yes	9.4	9.37
13	Lengthening	Bone transport	10.63	1mm/day	Yes	11.6	11.63
14	Both lengthening & Axis correction	Compression Distraction	7.77	1mm/day	Not done	9.2	8.77
15	Lengthening	Compression Distraction	9.27	1mm/day	Not done	5.8	10.27
16	Lengthening	Bone transport	8.27	1mm/day	Yes	7	9.27

Table-3 Outcome of Distraction Osteogenesis

Case	Healing Index	External Fixator Index	Bony Result	Functional Result	LEFS
1	5.36	5.23	Excellent	Good	53
2	1.43	1.35	Excellent	Excellent	80
3	3.42	3.31	Excellent	Good	44
4	0.98	0.91	Excellent	Good	46
5	0.78	0.71	poor	Poor	9
6	1.58	1.45	Excellent	Good	56
7	0.95	0.85	Fair	Poor	10
8	0.78	0.69	Excellent	Excellent	51
9	1.18	1.09	Excellent	Fair	33
10	1.00	0.90	Excellent	Good	68
11	1.64	1.49	Excellent	Good	44
12	1.00	0.89	Excellent	excellent	56
13	1.00	0.92	Excellent	Good	61
14	0.95	0.84	Excellent	Excellent	65
15	1.77	1.60	Excellent	Excellent	70
16	1.32	1.18	Excellent	Excellent	70

Figure1 Aetiology of the bone defect/deformity

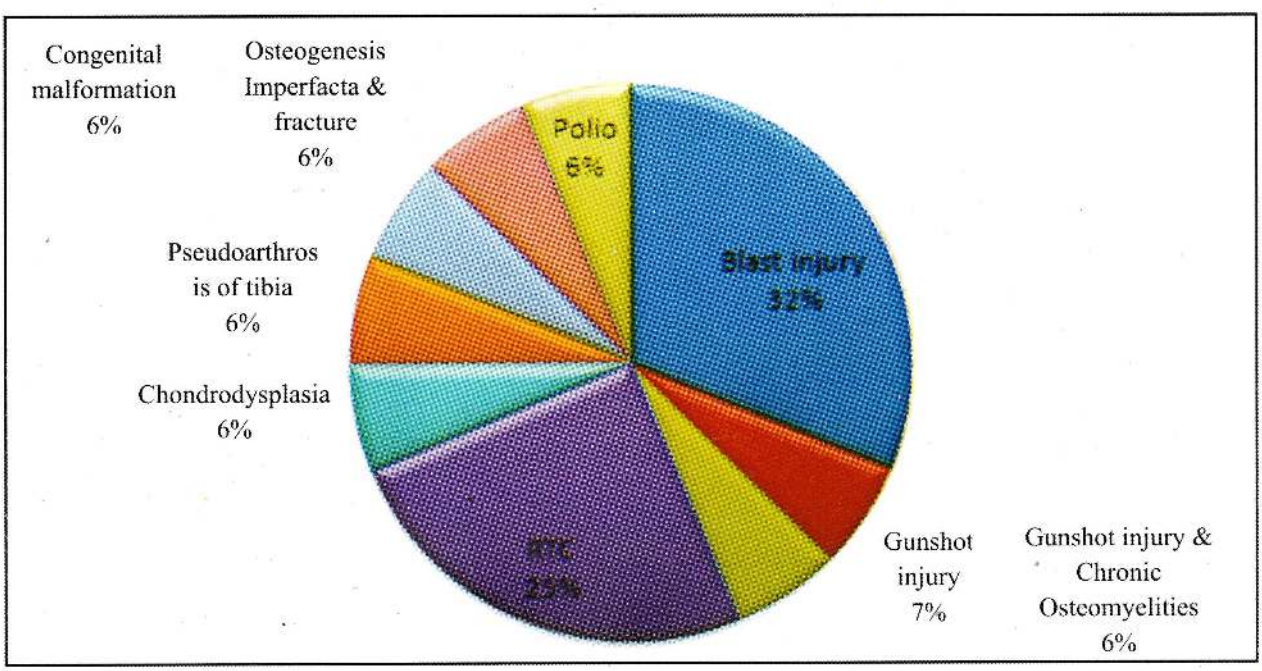


Figure 2 Bony results

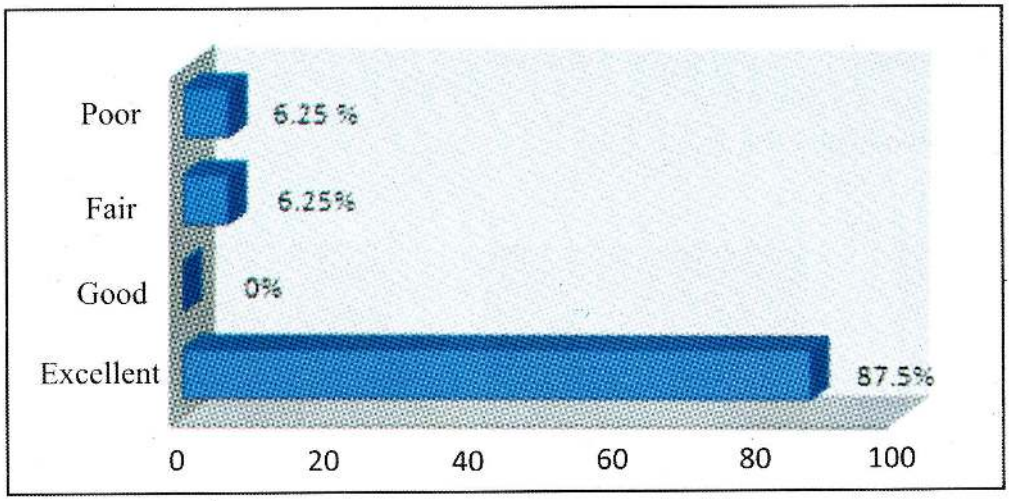


Figure 3 Functional results

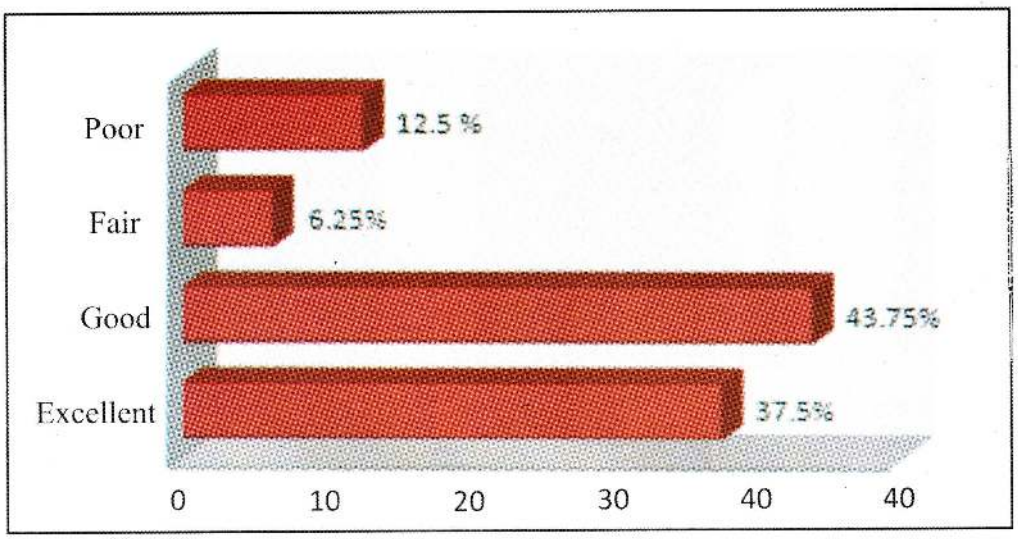


Figure 4 Phases of distraction osteogenesis

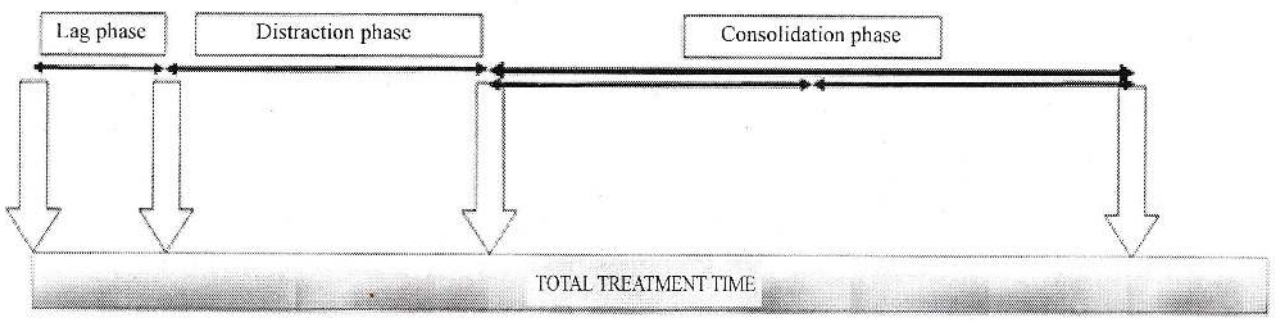


Figure 5 X-Rays of a patient

A. Pre operative X Ray - non union of femur after treatment for blast injury
B. On lag phase - Post operative day 2 after debridement, primary shortening & compression at the fracture site and osteotomy distally
C. On distraction phase - Post operative day 25
D. On consolidation phase -Post operative day 45
E. & F. On follow up

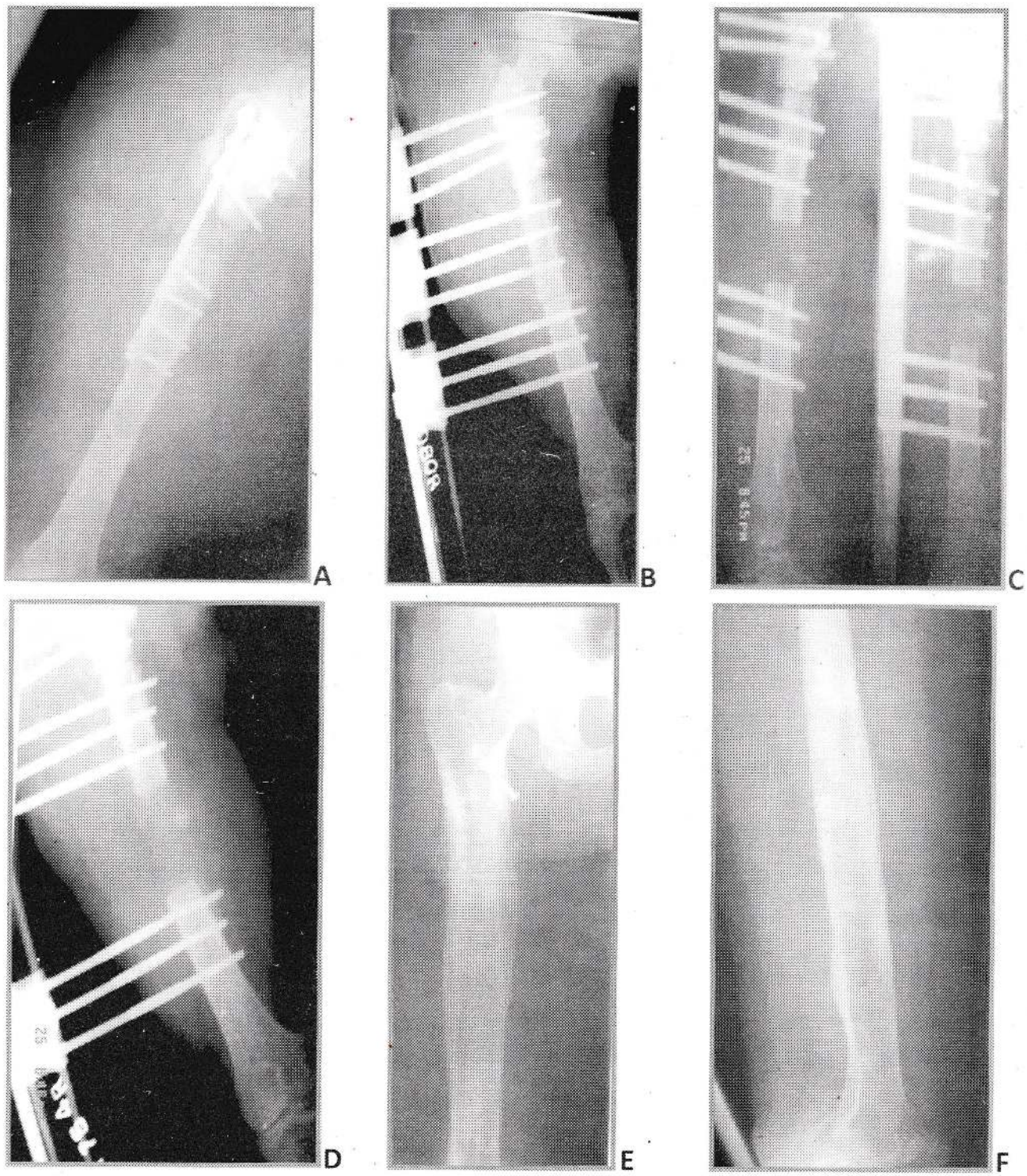
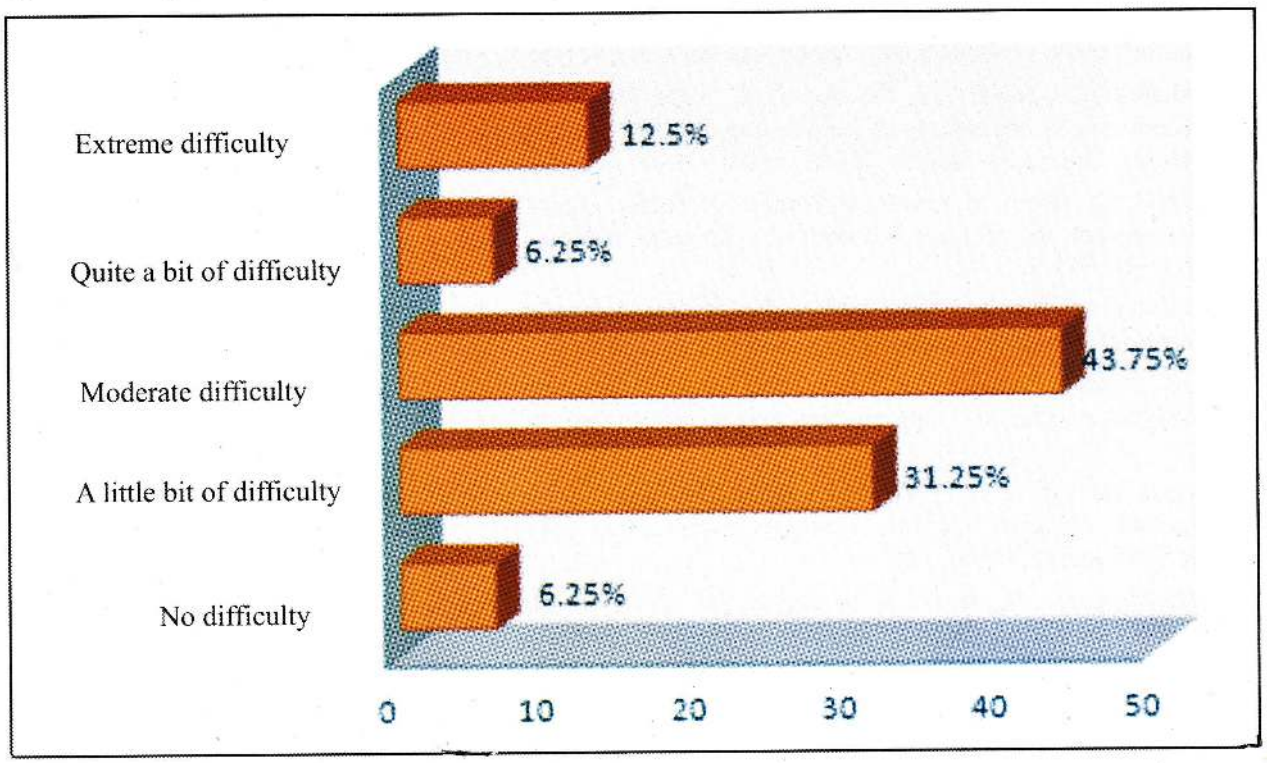


Figure 6 Long-term functional outcomes by Lower Extremity Functional Scale (LEFS).



References

1. Aronson J. Limb-lengthening, skeletal reconstruction, and bone transport with the Ilizarov method. J Bone Joint Surg Am. 1997 Aug;79(8):1243-58.
2. Paley D. Current techniques of limb lengthening. J Pediatr Orthop. 1988 Jan-Feb;8(1):73-92.
3. Bertram C, Nielander KH, Konig DP. [Pioneers in the lengthening of the extremities]. Chirurg. 1999 Nov;70(11):1374-8.
4. Kamegaya M, Shinohara Y, Shinada Y. Limb lengthening and correction of angulation deformity: immediate correction by using a unilateral fixator. J Pediatr Orthop. 1996 Jul-Aug;16(4):477-9.
5. Schep NW, van Lieshout EM, Patka P, Vogels LM. Long-term functional and quality of live assessment following post-traumatic distraction osteogenesis of the lower limb. Strategies Trauma Limb Reconstr. 2009 Nov 24.
6. Bhave A, Paley D, Herzenberg JE. Improvement in gait parameters after lengthening for the treatment of limb-length discrepancy. J Bone Joint Surg Am. 1999 Apr;81(4):529-34.
7. Paley D, Herzenberg JE, Paremain G, Bhave A. Femoral lengthening over an intramedullary nail. A matched-case comparison with Ilizarov femoral lengthening. J Bone Joint Surg Am. 1997 Oct;79(10):1464-80.
8. Jasiewicz B, Kacki W, Tesiorowski M, Potaczek T. [Results of femoral lengthening over an intramedullary nail and external fixator]. Chir Narzadow Ruchu Ortop Pol. 2008 May-Jun;73(3):177-83.
9. Oh CW, Song HR, Kim JW, Choi JW, Min WK, Park BC. Limb lengthening with a submuscular locking plate. J Bone Joint Surg Br. 2009 Oct;91(10):1394-9.
10. Oh CW, Shetty GM, Song HR, Kyung HS, Oh JK, Min WK, et al. Submuscular plating after distraction osteogenesis in children. J Pediatr Orthop B. 2008 Sep;17(5):265-9.
11. Potaczek T, Kacki W, Jasiewicz B, Tesiorowski M, Lipik E. [Femur lengthening with a telescopic intramedullary nail ISKD--method presentation and early clinical results]. Chir Narzadow Ruchu Ortop Pol. 2008 Jan-Feb;73(1):10-4.

12. De Bastiani G, Aldegheri R, Renzi-Brivio L, Trivella G. Limb lengthening by callus distraction (callotasis). J Pediatr Orthop. 1987 Mar-Apr;7(2):129-34.

13. Lu H, Yang Y, Zeng C, Zhou J, Hu J, Xu D, et al. [Effect of low-intensity pulsed ultrasound stimulation on maturation of regenerate bone]. Zhong Nan Da Xue Xue Bao Yi Xue Ban. 2009 Oct;34(10):984-90.

14. Venkatesh KP, Modi HN, Devmurari K, Yoon JY, Anupama BR, Song HR. Femoral lengthening in achondroplasia: magnitude of lengthening in relation to patterns of callus, stiffness of adjacent joints and fracture. J Bone Joint Surg Br. 2009 Dec;91(12):1612-7.

15. Kitoh H, Kawasumi M, Kaneko H, Ishiguro N. Differential effects of culture-expanded bone marrow cells on the regeneration of bone between the femoral and the tibial lengthenings. J Pediatr Orthop. 2009 Sep;29(6):643-9.

16. Kawasumi M, Kitoh H, Siwicka KA, Ishiguro N. The effect of the platelet concentration in platelet-rich plasma gel on the regeneration of bone. J Bone Joint Surg Br. 2008 Jul;90(7):966-72.

17. Zhao L, Fan Q, Venkatesh KP, Park MS, Song HR. Objective guidelines for removing an external fixator after tibial lengthening using pixel value ratio: a pilot study. Clin Orthop Relat Res. 2009 Dec;467(12):3321-6.

18. Noonan KJ, Leyes M, Forriol F, Canadell J. Distraction osteogenesis of the lower extremity with use of monolateral external fixation. A study of two hundred and sixty-one femora and tibiae. J Bone Joint Surg Am. 1998 Jun;80(6):793-806.

19. Antoci V, Ono CM, Antoci V, Jr., Raney EM. Pin-tract infection during limb lengthening using external fixation. Am J Orthop (Belle Mead NJ). 2008 Sep;37(9):E150-4.

20. Schiedel FM, Buller TC, Rodl R. Estimation of patient dose and associated radiogenic risks from limb lengthening. Clin Orthop Relat Res. 2009 Apr;467(4):1023-7.

21. Jochymek J, Gal P. Evaluation of bone healing in femurs lengthened via the gradual distraction method. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2007 Jun;151(1):137-41.

22. Hashmi MA, Ali A, Saleh M. Management of non-unions with mono-lateral external fixation. Injury. 2001 Dec;32 Suppl 4:SD30-4.

23. Kucukdeveci O, Sarisozen B, Atici T, Ozcan R. The effect of nicotine on distraction osteogenesis: an experimental study on rabbits. J Trauma. 2009 Dec;67(6):1376-83.

24. Fischgrund J, Paley D, Suter C. Variables affecting time to bone healing during limb lengthening. Clin Orthop Relat Res. 1994 Apr(301):31-7.

25. Li R, Saleh M, Yang L, Coulton L. Radiographic classification of osteogenesis during bone distraction. J Orthop Res. 2006 Mar;24(3):339-47.

26. Tesiorowski M, Potaczek T, Jasiewicz B, Kacki W, Lokas K. [Regeneration formation index--new method of quantitative evaluation of distraction osteogenesis]. Chir Narzadow Ruchu Ortop Pol. 2009 May-Jun;74(3):121-6.

Case report

Herpes Zoster in the first year of life following antetnatal exposure to Varicella Zoster virus

Herpes zoster in the first year of life is extremely rare and we present a case of Herpes Zoster in a 10 month old boy following antenatal exposure of varicella virus infection.

Case History:
10 month old male infant presented with a four day history of eruption of vesicular lesions on an erythematous base on the right side of the face. The rash was distributed in the ophthalmic branch of the trigeminal nerve. He did not have chickenpox previously neither did he give a history of household exposure to varicella. The mother gives a history of having chickenpox while she was pregnant with a period of amenorrhoea of 24 weeks. On initial physical examination clearly showed grouped vesicles with an erythematous base mainly in the right forehead, face, eyelid and upperlip compatible with the involvement of the ophthalmic branch of trigeminal nerve. There were no intra ocular lesions. The base and tip of the nose (distribution of naso ciliary branch of the ophthalmic) were spared. (Figure 1) He also had a normal slit lamp examination of the eyes.

Discussion:
Herpes zoster, a painful vesicular dermatomal eruption, is the result of reactivation of the varicella zoster virus (VZV) from infected sensory ganglia. Traditionally, it is considered to be a disease of adults, in contrast to primary infection with VZV, which tends to occur mainly in children

Generally, primary varicella tends to occur in childhood, whereas herpes zoster is a disease of adults, with most patients being older than 45 years. In the paediatric population, the incidence is the lowest in the group 0 to 5 years of. Antigen-specific T cells are believed to be the principal mechanism of latent VZV. Conditions in which cellular responses were lost or diminished by immunosuppression pose a risk for reactivation of VZV

Acquisition of herpes zoster in healthy immunocompetent children in early childhood or during intrauterine exposure has been attributed to the immaturity of the immune system. Maternal varicella during the first trimester is likely to produce congenital varicella syndrome; when women have the disease later in pregnancy, the foetus can develop asymptomatic congenital infection and subsequently present clinically with herpes zoster within the first year of life. This is the most likely cause in this child.

MG Sathiadas
Senior Lecturer in Paediatrics,
Faculty of Medicine, University of Jaffna
S Sukumaran,
Consultant Dermatologist, Teaching Hospital, Jaffna

A high index of suspicion should be aroused when vesicular lesions are noted to be in a dermatomal distribution. This is clearly demonstrated in our case with the ophthalmic branch of the trigeminal nerve involvement sparing the area supplied by the nasociliary branch. The most common differential diagnosis is impetigo, and bacterial culture can usually distinguish the 2 conditions, unless there is concurrent bacterial infection of the herpetic lesions. In our case the blood culture and culture of the lesions were negative.

Diagnostic test are Tzanck smear for multinucleated giant cells. Immunofluorescence and PCR techniques can be useful as well. Viral cultures are possible but viruses are difficult to scrape from the lesions.

The treatment of this condition is IV/oral Aciclovir, our patient received IV acyclovir for 7 days and the condition resolved without any further complications.

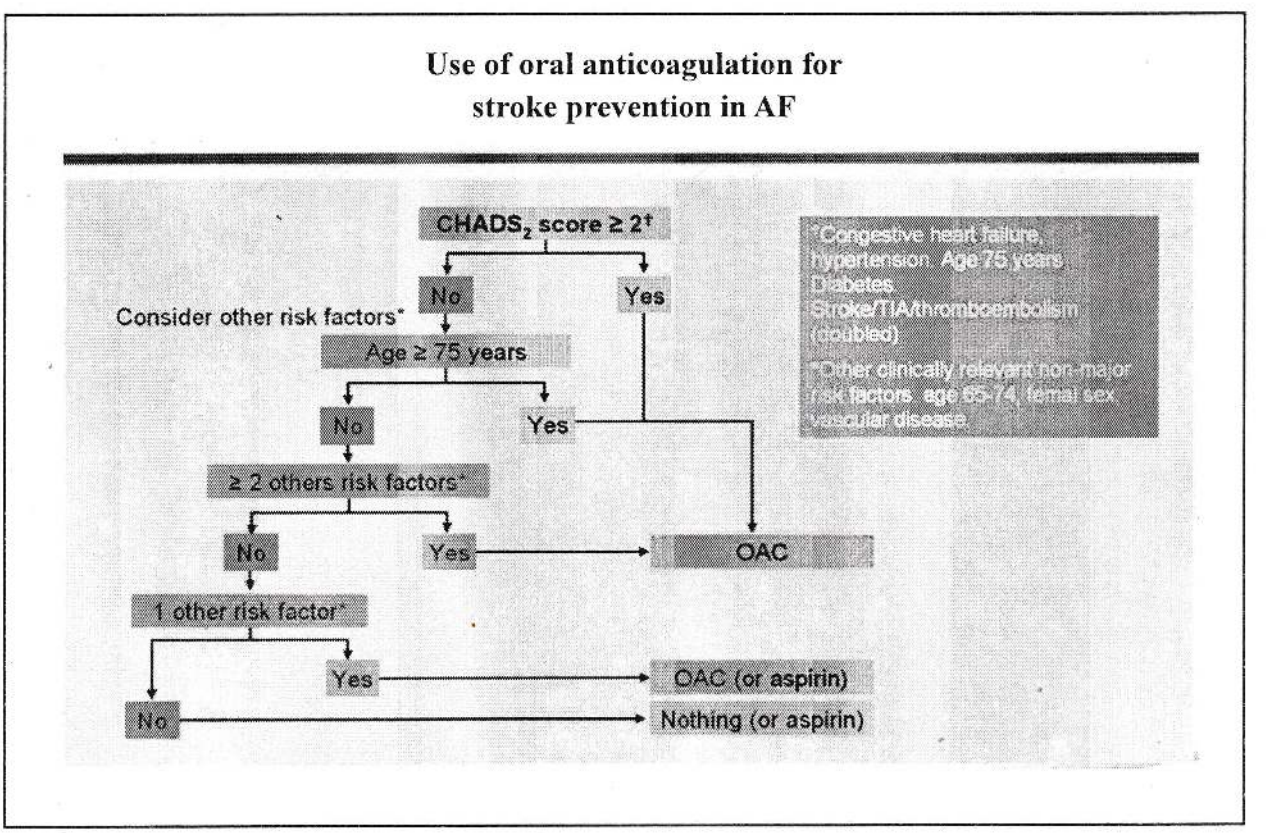
The classic feature associated is the Hutchison's sign in which skin lesions occur in the distribution of nasociliary branch (tip of the nose) of the trigeminal nerve. In regards to prognosis Hutchinson's sign was a powerful predictor of sight-threatening ocular complications in acute herpes zoster ophthalmicus [relative risks: 3.35 (CI 95%: 1.82-6.15)].(2) In our patient this was negative which explains the absence of eye involvement and the complete recovery.

Note: Prior permission was sought from the mother for publication

References

1. Julia G Kurlan, Beverly L Connely, Ann W Lucky. Herpes zoster in the first year of life following postnatal exposure to VZV. *Arch Dermatol. 2004;140:1268-1272*

2. Zaal MJ; Volker-Dieben HJ, D'Amaro J. Prognostic value of Hutchinson's sign in acute herpes zoster ophthalmicus. Graefs Arch Clin Exp Ophthalmol, 2003;241(3):187-91



View Point

Cancer Prevention and Control

Cancer is one of the most common causes of morbidity and mortality today with more than 10 million new cases and more than 6 million deaths each year worldwide. It is predicted that by 2020 cancer could kill 10.3 Million people unless we act promptly (Fig:1). The sad aspect of this is that most of the deaths would be in the developing countries than in developed countries (Fig:2). It is also estimated that around 43% of cancer deaths are due to tobacco use, unhealthy diets, alcohol consumption, inactive lifestyles and infection. Low income and disadvantaged groups are generally more exposed to avoidable risk factors such as environmental carcinogens, alcohol, infectious agents, and tobacco use. These groups also have less access to the health services and health education that would empower them to make decisions to protect and improve their own health.

In Sri Lanka, cancer incidence is steadily rising with more number of cancers reported among females (Fig:3). Among males Oro-pharyngeal cancer is the significant component of the cancer burden (Fig:4). Among females Breast and Cervical cancers are the leading cancers (Fig:5).

Prevention and screening of common cancers are important scientific and public health challenges to reduce the burden of cancer. Scientific researchers have demonstrated that cancer occur not as sudden catastrophic events but rather as a result of a complex and long evolving process called multi step carcinogenesis (Fig:6). The process of carcinogenesis can take decades to complete, providing time and opportunity for us to intervene, to stop or to reverse its progress either before the clinical appearance of cancer or a its earliest stage.

N.Jeyakumaran
Consultant Clinical Oncologist, TH Jaffna.

Betel nut chewing, tobacco use, alcohol consumption and poor oral hygiene are regarded as the major risk factors for the increased Oro-pharyngeal cancers among males. Tobacco use and alcohol consumption act synergistically to cause cancer of the oral cavity, pharynx, larynx and oesophagus. Oral cancer is preventable through risk factors intervention.

Breast and cervical cancers are most frequently reported and also present in late stages like ulcers over the breast. To prevent such a late presentation, it is advisable to conduct cancer awareness in large scale among the public. In addition, there are methods available for early detection of breast cancer such as screening by mammography, clinical breast examination and breast self-examination.

For control of cervical cancer too there are many effective programmes that are conducted worldwide like vaccination against Human Papilloma Virus infection especially against subtypes 16 and 18 and cytology-based screening using the Papanicolaou smear (Pap smear) programmes to detect early stages or even pre cancerous stages. Pap smear programme is very cost effective. In many low-income countries, however, cytology screening has proved difficult to sustain because of its reliance on highly trained cytotechnologists, good-quality laboratories and infrastructure to support up to three visits for screening, evaluation of cytologic abnormalities with colposcopy, and treatment. Two alternative screening approaches replace the Pap smear with simple visual screening methods, such as visual inspection after application of an acetic acid solution (VIA), or with HPV DNA testing.

Other cancers that add to the cancer burden among males are lung, oesophagus & colorectal cancers and among females are oesophgus, ovary & thyroid cancers. Some cancers like colorectal cancer that are

increasingly in developing countries may reflect the increasing Westernization of lifestyles, intake of high fat diet and longer life expectancy.

The World Health Organization (WHO) emphasizes that, when developing national strategies for controlling cancer, countries should consider the following four broad approaches.

1.Primary prevention.
The goal of primary prevention is to reduce or eliminate exposure to cancer causing factors, which include environmental carcinogens and lifestyle factors related to nutrition and physical activity. For many cancers, approaches to primary prevention like immunization against, or treatment of, infectious agents that cause certain cancers, tobacco control programs, reduction of excessive alcohol consumption, dietary intervention and pharmacological intervention can be implemented.

2.Early detection and secondary prevention.
The main objective of early detection or secondary prevention through population based screening programs is detection at a stage at which curative treatment is possible. Interventions for the early detection of cancer can help reduce mortality from cancer, only if they are part of a wider cancer control strategy that includes effective diagnostic follow-up procedures and treatment. For breast, cervical, and colorectal cancer, effective methods of early detection and treatment are available, but their implementation has been uneven.

3.Diagnosis and treatment.
The primary modalities of cancer treatment are surgery, radiotherapy and chemotherapy and these modalities may be used alone or in combination. There is increasing emphasis worldwide on the development of specialized cancer centers that apply evidence based multimodal therapies, including rehabilitation and palliative care.

4.Palliative care.
The scope of palliative care has been expanded in recent years to encompass the alleviation of symptoms and treatment during all phases of disease, starting from diagnosis till or even after death and to address matters related to the psychological and quality of life aspects of disease, as well as the physiological aspects. Furthermore, palliative care has been expanded to include consideration for the well being of the patient's family members as well as for the patient.

In Jaffna, due to the overuse of fertilizers and pesticides, the underground water nitrate levels are high. Vegetables may contain pesticide residues as most of the farmers use pesticides and they do not adhere to the concept of pre harvest interval after the last spray of chemicals. These chemicals in the food and drinking water could be an attributing factor for cancer causation.

Early detection, which comprises screening of asymptomatic populations for common cancers and creating awareness of early symptoms of cancers among the public, increases the probability of cure. Awareness of early signs and symptoms is particularly relevant for cancers of the breast, cervix, mouth, larynx, colon and rectum. However, it requires the facilities to confirm diagnosis and provide treatment, and availability of resources to serve the population in need.

Fig: 1 **Global Projections for Selected Causes for Deaths : 2004 - 2030**

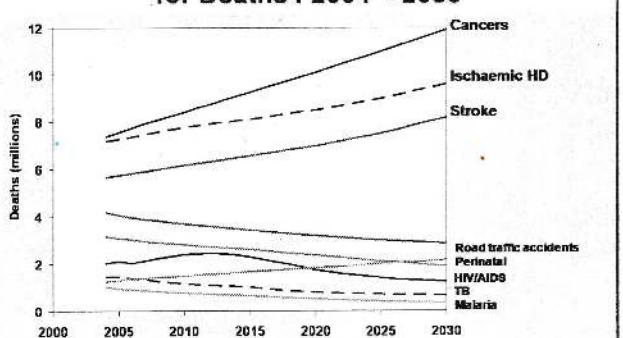
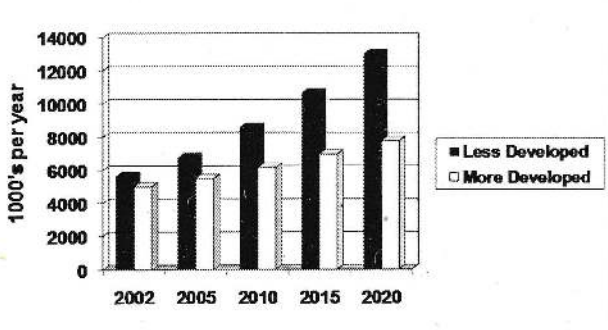


Fig: 2 **Predicted Cancer Deaths**



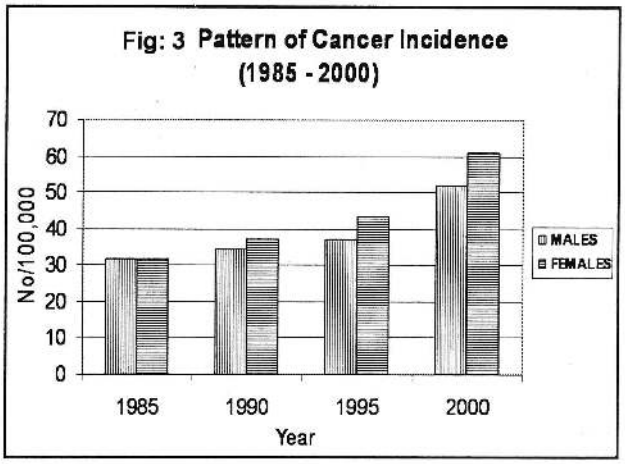


Fig: 3 Pattern of Cancer Incidence
(1985 - 2000)
No/100,000
70
60
50
40
30
20
10
0
1985
1990
1995
2000
Year
MALES
FEMALES

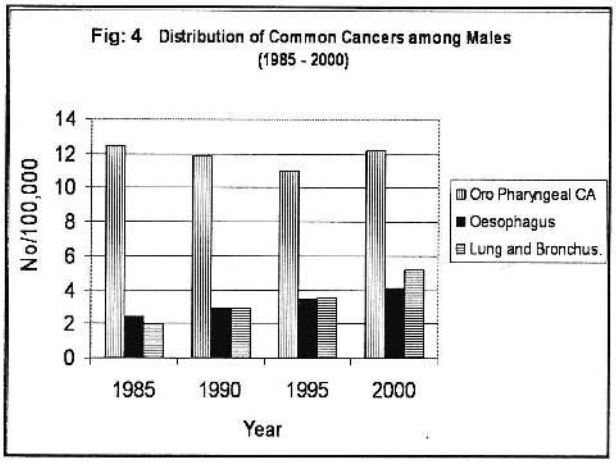


Fig: 4 Distribution of Common Cancers among Males
(1985 - 2000)
No/100,000
14
12
10
8
6
4
2
0
1985
1990
1995
2000
Year
Oro Pharyngeal CA
Oesophagus
Lung and Bronchus.

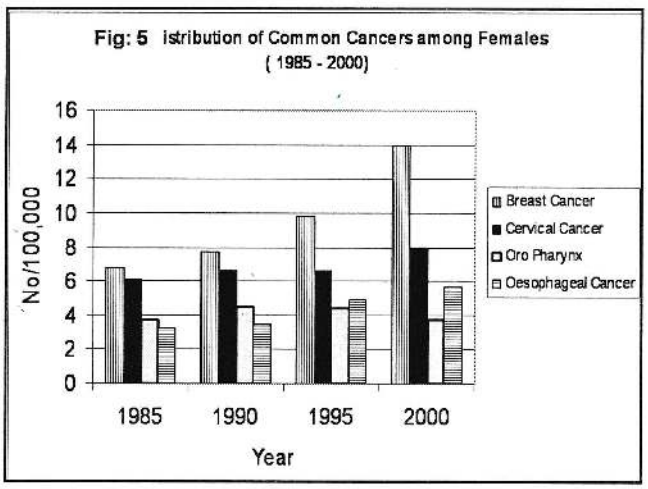


Fig: 5 Istribution of Common Cancers among Females
(1985 - 2000)
No/100,000
16
14
12
10
8
6
4
2
0
1985
1990
1995
2000
Year
Breast Cancer
Cervical Cancer
Oro Pharynx
Oesophageal Cancer

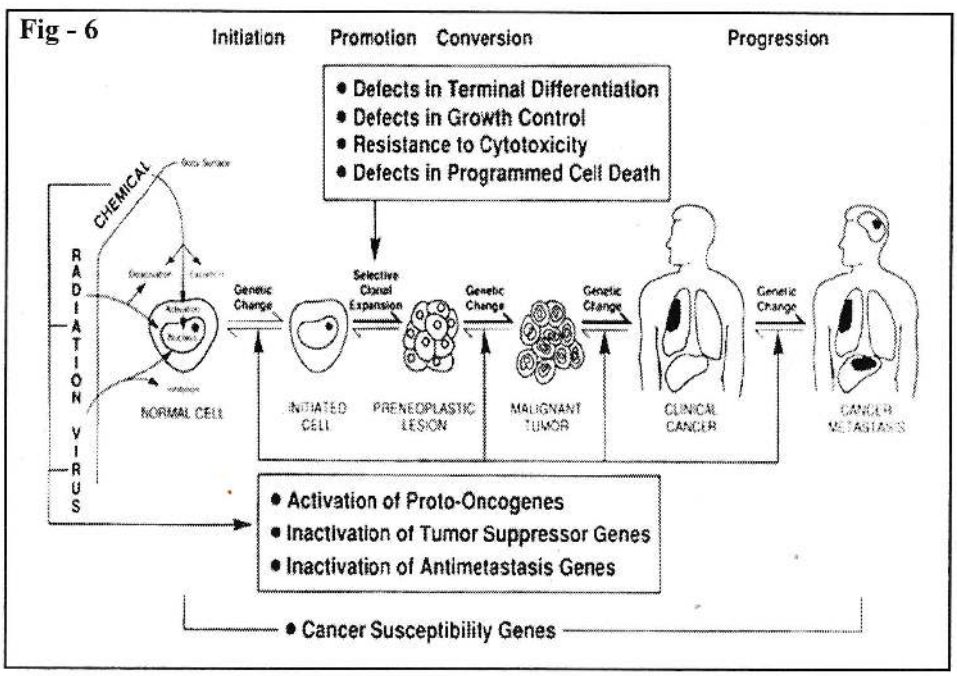


Fig - 6
Initiation
Promotion
Conversion
Progression
• Defects in Terminal Differentiation
• Defects in Growth Control
• Resistance to Cytotoxicity
• Defects in Programmed Cell Death
CHEMICAL
RADIATION
VIRUS
Genetic Change
Selective Clonal Expansion
Genetic Change
Genetic Change
Genetic Change
NORMAL CELL
INITIATED CELL
PRENEOPLASTIC LESION
MALIGNANT TUMOR
CLINICAL CANCER
CANCER METASTASIS
• Activation of Proto-Oncogenes
• Inactivation of Tumor Suppressor Genes
• Inactivation of Antimetastasis Genes
• Cancer Susceptibility Genes

Case report

Unusual Presentation of Adult Myelodysplastic Syndrome

Introduction

Myelodysplastic syndrome (MDS) is a clonal hematopoietic stem cell disorder characterized by peripheral cytopenia(s), bone marrow dysplasia and susceptibility to acute leukemia (1). MDS is rare in adulthood,(2). The disease seems to be more frequent in patients who previously received radiotherapy and chemotherapy for a first malignancy (3). Bone Marrow cellularity is normal or increased in MDS with hypocellular marrow being a rare feature (4). This case highlights a rare feature of MDS with hypocellular bone marrow in a adult.

Case report

A 68 year old gentleman presented with low grade fever, easy fatigability and dizziness for three weeks duration. General physical examination was unremarkable except for mild pallor with no hepatosplenomegaly or lymphadenopathy. Past history was non contributory with no prior exposure to cytotoxic drugs, radiation or other mutagens. Laboratory picture showed pancytopenia, with hemoglobin 7.2 g/dl, hematocrit 21.9%, mean corpuscular volume (MCV) 104.8 fl, Mean corpuscular concentration (MCH) 34.4pg,Mean corpuscular hemoglobin concentration (MCHC) 32.9 g/dl, total leukocyte count (TLC) 3.4 X 109 /l, with neutrophils 52%, lymphocytes 46%, platelet count 80 X 109 /l . Morphological diagnosis was based on analysis of peripheral blood smear (PBS), bone marrow aspirate (BMA) and bone marrow biopsy (BMB) with cytochemical staining with periodic acid Schiff (PAS) and Perl's stains. PBS showed severe pancytopenia. BMA showed no blast cells. Dyserythropoiesis was characterized by nuclear-cytoplasmic asynchrony, nuclear budding and megaloblastic nuclei. Dysgranulopoiesis was evident principally by nuclear hyper segmentation and abnormal granulation. Megakaryocytes were markedly reduced, even occasionally are seen with separated nuclei and monolobated forms. Iron stain showed increased iron stores (4+) and no ringed sideroblasts. BMB indicated a hypocellular bone marrow for that age with characteristic trilineage dysplasia; areas of marked hypocellularity and patches of mildly hypercelluar marrows spaces composed mild erythoid hyperplasia with dysplasia including megaloblastic and normoblastic maturation, active myelopioesis with mild dysplasia including maturation arrest at late precursor stage and active megakaryopioesis including monoblated forms. There was no blast proliferation or infiltration. The cytogenetic analysis revealed no numerical or structural abnormalities (Karyotype; 46, XY). Bone marrow for Tuberculosis culture and PCR were negative. Serum protein electrophoresis has no monoclonal bands. The case was diagnosed as MDS, Refractory Cytopenia with Multiline age Dysplasia (RCMD) in accordance with the WHO criteria; Intermediate 1 risk category according to International Prognostic Scoring System IPSS). The patient received supportive treatment consisting of folate and B12 and blood transfusions. The patient could be followed up with routine blood tests to evaluate the hematological outcome of the disease. Trail of recombinant Human erythropoietin combined with tranfusional support was commenced, even though hypothylating agents (Azacytadine/Decitabin-which are not available in Sri Lanka) are recommended. The patient did not transform to a more aggressive form of MDS or leukemia and followed up in a satisfactory clinical condition.

Discussion

MDS is a rare hematological disorder in adults. The symptoms are non specific and the diagnosis

S. Pirasath, Medical officer,
K.T.Sundaresan, Consultant Physician
Teaching Hospital, Batticaloa.

may be difficult to make. Prognosis is extremely variable depending on the type of MDS. The pre leukemic phase is usually short in adults [12 18 months] and the disease rapidly evolves into overt leukemia. This case highlights uncommon feature: unusual finding is the hypocellularity of the bone marrow for the age of the patient. Bone marrow hypocellularity is a rare feature in MDS (5), Hypocellularity on BMB is defined when it is below the normal range value adjusted for the age on a semi-quantitative evaluation (6). Bone marrow cellularity is the critical determinant for the recognition of hypocellular MDS (7). According to the WHO classification (8), MDS patients are classified into three groups: refractory cytopenia (RC), refractory anemia with excess of blasts (RAEB) and RAEB in transformation (RAEB-T). In accordance with the WHO criteria for detecting MDS, this patient fulfilled as MDS, Refractory Cytopenia with Multiline age Dysplasia (RCMD); Intermediate 1 risk category according to International Prognostic Scoring System IPSS); suggested by Hasley et al (9): sustained unexplained cytopenia and/or at least bilineage morphological myelodysplasia and/or acquired clonal cytogenetic abnormality in hematopoietic cells and/or increased blasts > 5%. The differential diagnosis considered in this case was hypocellular acute myeloid leukemia (AML) with low blast count, aplastic anemia and several congenital bone marrow failure syndromes. The diagnosis was established by a combination of clinical and laboratory parameters along with BMA and BMB pictures which showed trilineage dysplasia.The diagnosis of hypocellular MDS without excess blasts is to be made with caution especially in the absence of cytogenetics. The sparse number of cells available for evaluation and the subjective grading of qualitative abnormalities account for the diagnostic difficulties. It would have been difficult to diagnose if a monolineage dysplasia was present.

Conclusion

MDS should be considered in the differential diagnosis of all cytopenic disorders in adults. It is important to diagnose MDS at an early stage by critical evaluation of dysplasia as preleukemic phase is usually short in adults and the disease rapidly evolves into overt leukemia.

Acknowledgements

We thank Dr Sanaja De Silva, Consultant Hematologist, Dr.Thanuja Dissanayakae, Consultant Hematologist, Dr Bhaddika Jayaratne, Consultant Hematologist, Dr.Vajirah H W Dissanayakae, Medical Geneticist, Dr.Vasantha Ariyarathne, Consultant Clinical Oncologist and Dr. Krishanthy Rajasuria, Consultant Oncologist for their contribution in diagnosis , multidisciplinary management and follow up of this gentleman.

References:

1.Chen B, Zhao W-L, Jin J *et al.* Clinical and cytogenetic features of 508 Chinese patients with Myelodysplastic syndrome and comparison with those in Western countries. *Leukemia* 2005; 19:767-5 .

2.Hasle H, Wadsworth LD, Massing BG, *et al.* A population based study of childhood Myelodysplastic syndrome in British, Columbia, Canada. *Br J Haematol* 1999; 106:1027-32 .

3.Koeffler HP. Myelodysplastic syndromes (preleukemia). *Semin Hematol* 1986; 23:284-9.

4.Luna-Fineman S, Shannon KM, Atwater SK, *et al.* Myelodysplastic and myeloproliferative disorders in childhood: a study of 167 patients. *Blood* 1999; 93:459-66

5.Barnard DR, Kalousek DK, Wiersma SR, *et al.* Morphologic, immunologic, and cytogenetic classification of acute myeloid leukemia and Myelodysplastic syndrome in childhood: a report from the Children's Cancer group. *Leukemia* 1996; 10:5-12

6.Foucar K, Bone marrow pathology, Chicago: American Society of Clinical Pathology (ASCP) Press; 2001:13

7.Tuzuner N, Christopher C, Jacob M, Rowe, Wtarous D and Bennett JM. Hypocellular Myelodysplastic syndromes (MDS): new proposals. *Br J Haematology* 1995:91:612-17 .

8.Hassle H, Niemeyer CM, Chessels JM, *et al.* A Pediatric approach to the WHO Classification of Myelodysplastic and myeloproliferative diseases. *Leukemia* 2003; 17:277-82.

Literature review

Arteriovenous fistula for chronic haemodialysis

An arterio venous (AV) fistula is defined as a communication between an artery and vein and implies all gradation of orificial size.[1]The success of chronic haemodialysis in terminal renal failure depends on repeated access to blood vessels that will provide a continuous flow of blood in a required level (up to 250-300ml/min).[1] Previously, permanent implantation of cannula into an artery and vein of the forearm was performed to achieve the portal for the dialysis. The prosthesis carried several disadvantages and caused morbidity in patients undergoing chronic dialysis. Prompted by these experiences with external shunts, consideration regarding the surgical creation of arteriovenous fistula in between radial artery and any available vein in the forearm was utilized.[1]

Timing of AVF

Arteriovenous fistula should be planned at least 3-4 months before scheduled beginning of dialysis to establish vascular access when serum creatinine concentration become more than 4mg/dl and estimated GFR $\leq$25ml/min.[2]

Sites for AVF formation

Though the radiocephalic AV fistula is the commonly performed procedure, than, alternative sites for AV fistula include anatomic snuff box fistula, high radiocephalic forearm AV fistula, elbow AV fistula, transposed basilic vein AV fistula.[3]

Initial evaluation

Venipuncture and central venous catheter, insertion of arterial lines, upper extremity, neck, and chest surgery and the history of previous thrombosis should be included in the history.[4]

Rajendra S
Consultant surgeon, Teaching hospital Jaffna.

Kalaventhan P
Demonstrator, Department of Surgery,
Faculty of Medicine, University of Jaffna.

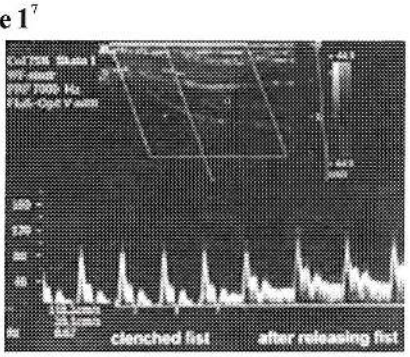
In physical examination, size, symmetry, colour, texture of skin and the presence of the oedema and collateral circulation should be noted.[4] Radial, ulna, brachial pulses, and the pulse volumes must be palpated on each arm and Allen test will be performed to find out the dominant artery of the hand. Following that, physical examination should include venous examination to see for any obstruction in cephalic, basilic, antecubital area, and forearm, by using tourniquet to assess.[5] Ultra sound scan/Duplex sonography also can be incorporated to the initial assessment to exclude any obstruction in the vein, artery.[6]

Arterial examination by Ultra sound scan/Duplex sonography

The lumen of the radial artery and the vein should be measured. It is well known that a large proportion of the blood flow in the fistula is provided by the ulnar artery by the palmar arch in upto one third of patients. The vasodilatory capacity of the palmar arch should therefore be assessed by calculating the resistance index from post ischaemic diastolic blood flow after fist clenching. This is an index of vascular reactivity to shear stress and of vascular reserve. Improvement of the outcome of the fistula has been documented by Silva MB *et al*[5]

$$\text{Resistance index(RI)} = \frac{\text{Maximal systolic flow velocity-Diastolic flow velocity}^{7}}{\text{Maximal systolic flow velocity}}$$

Figure 1[7]



Preoperative imaging of the radial artery by color flow duplex sonography. Doppler frequency spectrum shows a high-resistance flow with a clenched fist (typical tricyclic flow; left) and a low-resistance flow after release.[7]

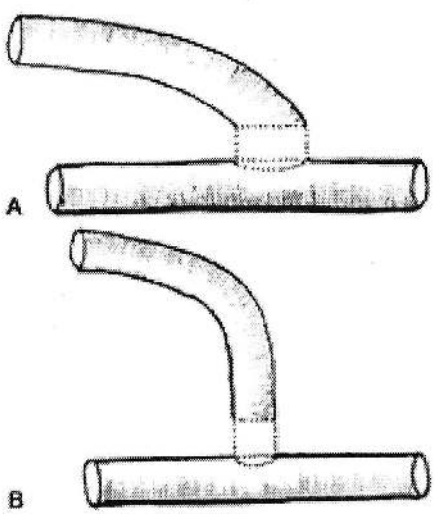
Technique of radial - cephalic fistula creation

After positioning and draping, the course of the cephalic vein out lined with the help of the tourniquet. A longitudinal incision will be made after infiltrating the skin with local anaesthetic solution. The cephalic vein is mobilised first to assess its calibre. The cephalic vein should be handled with care to avoid vasospasm.[7]

Following exposure of the artery, the vein is divided and the proximal end is flushed with heparinized saline. An arteriotomy is made by number 11 blade and straight scissors. Anastomosis is started with double armed 6/0 or 7/0 prolene suture. The anastomosis should be started from proximal end distal end. Before completing the anatomosis, each vessel is irrigated through the tubing with heparinized tubing with heparinized saline. Skin closure is usually done by subcuucular 4/0 vicryl and the good thrill should be felt over the fistula and over the vein above the level of the fistula.[3]

Technical errors in radial - cephalic fistula

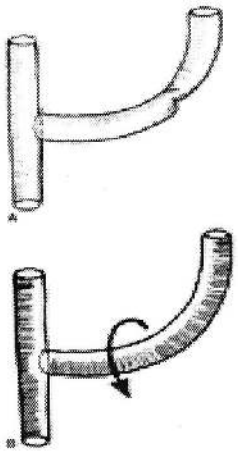
Figure 2[7]



(Panel A) The venous stump is cut in a direction parallel to the artery. If an acute angle is created between the artery and the vein, the result will be a long arteriotomy, exceeding by far the diameter of both artery and vein. (Panel B) If the vein approaches the artery at an angle of 90 degrees, the length of the anastomosis will equal the diameter of the vein.[7]

If a distant vein is mobilized and the venous stump is made to meet the artery without any adaptation, there is a high risk that the vein undergoes torsion, which is often responsible for early failure and thrombosis.[7]

Figure 3[8]



Haemodynamics of arterio-venous fistula

Blood flow in a normal peripheral artery will be around 20-30 ml/min. In immediate post operative period blood flow dramatically increased upto 200-300 ml/min from the above blood flow rate due to the decreased peripheral resistance. But, can exceed 1000ml/min in a well functioning arterio venous fistula in the long run. Venous remodeling ("venous arterialization" or arterialization of the vein) occurs due to increased blood flow through vein and it depends on arterial dilatation. Anything impeding the increased blood flow and the vascular remodeling will endanger the final outcome of a wellfunctioning arterio venous fistula.[7]

Post operative care

During immediate post operative period, vital signs should be checked and systolic blood pressure should be maintained more than 100mmHg, diastolic blood pressure should be maintained more than 60mmHg, pulse rate should be maintained more than 60/minute and less than 110/minute and temperature should be kept within 38°C. During examination in post operative period, thrill and the bruit will be felt to ascertain the function of the arteriovenous fistula. A Stable patient can be discharged 4 hours after surgery.[7]

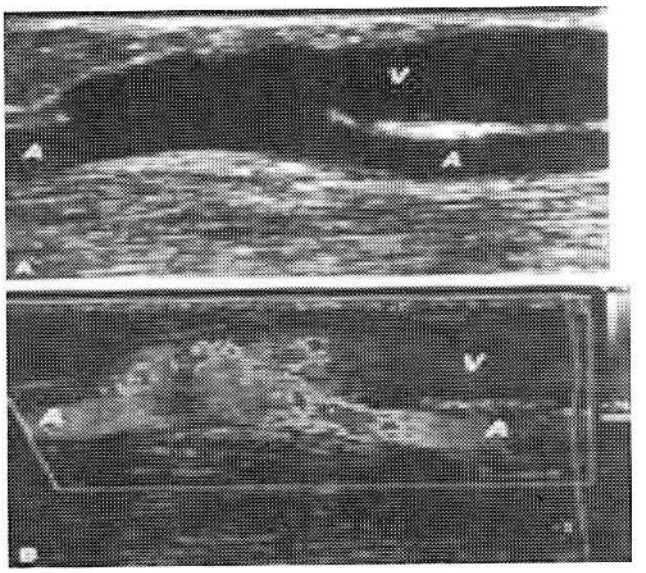
Follow up care

Upper extremity will be kept elevated and patient should be informed about the "fistula care" pre-operatively. Patients should be told to wear loose sleeved garments, avoid lying on fistula bearing

extremity, avoid measuring blood pressure, and blood drawing from that extremity. They should do hand exercise regularly by squeezing a squash ball.[3]

Post operative care

Figure 4[7]



(Panel A) B-mode sonogram showing a longitudinal section of an arteriovenous fistula created by anastomosing the cephalic vein (V) to the radial artery (A) at the wrist in a side-to-end fashion. (Panel B) With the same sectional view, Color Doppler sonography shows turbulent flow at the arteriovenous anastomosis. The radial artery (A) is feeding the shunt vein (V).[7]

Complications of AVF

Early failure (upto 29%) of arteriovenous fistula due to errors in surgical technique. Thrombosis is the commonest cause for the arterio venous fistula failure. Other causes are systemic clotting disorders, hypotensive episodes especially in haemodialysis sessions may contribute in the arteriovenous fistula failure.[8]

Steal phenomina is one of the complications of arteriovenous fistula, where retrograde blood flow from the distal artery of the fistula to the low resistance venous system causing hypoperfusion to the palm and fingers.[9]

Heart failure (High Output) can be a complication. But, it is rare with brachiocephalic/transposed basilic vein access.[8]

Stenosis of central veins (Paget Schroetter Syndrome) may clinically asymptomatic before creating the vascular access and become symptomatic after when the flow is increased.[9]

Aneurysm of arterio venous fistula due to repeated puncture and occur after the dialysis sessions.[9]

Post operative blood flow measurements

Post operative blood flow measurement can predict the outcome of the arteriovenous fistula. Radial artery cross sectional area of >8.5mm^2& venous flow of >425ml/min have positive predictive value of 0.95 and 0.97 for outcome of radio-cephalic fistula. Low flow rate & velocities in arteriovenous fistula within first 2 weeks will cause fistula failure.[10] Post operative blood flow measurements can reveal delayed maturation. USS examination of AVF of delayed maturation at 1 month can be referred to surgery/interventional radiology.[11]

Patients' factors and surgeons' factors and their influence on the patency of the arteriovenous fistula

Burt CJ *et al* concluded that, age over 60 years not influence on the patency of radio cephalic fistulae and which remains as the haemodialysis access procedure of choice at all age.[12]

Diabetes has no effect on radio cephalic fistula failure. But, significantly shortened the mean patency time.[13]

Surgical trainees can perform primary AVF surgery without significantly reducing fistula outcomes.[14]

References

1. Brescia MJ, Cimino JE, Appel K, Hurwich B. Chronic haemodialysis using venepuncture and a surgically created arteriovenous fistula .The New England journal of medicine.1966 ;275:1089-92.
2. NKF-K/DQQI. Clinical practice guidelines for vascular access: New update 2000. Guideline 1: Patient evaluation prior to access placement. American journal of Kidney disease .2001; 37(1): 141-9.

3. Hakim NS. How to perform an arteriovenous fistula. British journal of hospital medicine [Internet]. 2005 Dec; 60(12): M94-95.
4. De marchi S, Falleti E, Giacomello R, Cecchin E, Seplacci G, Bartolotti N, Zanello F, Gonano F, Bartoli E. Risk factors for vascular disease and arteriovenous fistula dysfunction in haemodialysis patients. J Am Soc Nephrol.1996;7: 1169-1177.
5. Silva MB, Hobson RW, Pappas PJ. Vein transposition in the forearm for autogenous haemodialysis access. J vasc surgery.1997;26(6):981-6.
6. Malovrh M. Native arterio venous fistula:Pre operative evaluation. AM J Kidney Dis. 2002;39: 1218-25.
7. Konner K. A primer on the arteriovenous fistula-achilles heel, but also Cinderella of haemodialysis. Nephrol Dial transplant. 1999;14: 2094-98.
8. Palder SB, Kirkman RL, Whittemore AD, Hakim RM, Lazarus JM, Tilney NL. Vascular access for haemodialysis: Patency rates results of revision. Annals of surgery. 1985; 202: 235-9.
9. Konner K, Daniel BN, Ritz E. The arteriovenous fistula. J Am Soc Nephrol. 2003 June 1 ;14(6): 1669-80.
10. Lin SL, Chen HS, Huang CH, Yen TS. Predicting the outcome of haemodialysis arteriovenous fistulae using duplex ultrasonography. J formos Med association. 1997; 96:864-68.
11. Robin ML, Lockart ME, Allon M. Haemodialysis arteriovenous fistula maturity: Ultrasound scan evaluation [Letter]. Radilogy. 2003 June; 227:906-907.
12. Burt CG, Little JA, Mosquera DA. The effect of age on radio-cephalic fistula patency. Journal of vascular access [Internet]. 2001;2(3): 110-3.
13. Fernstrom A, Hylander B, Olofsson P, Sweedenborg J. Long and short term patency of radiocephalic arteriovenous fistulas. Acta chir scand 1988;154:257-9.[Medline]
14. Weale AR, Barwell J, Chant H, Lear PA, Mitchell DC. The impact of training on outcomes in primary vascular access surgery. Annals of the Royal college of surgeons of England. 2004;86(4): 275-80.

Stages of Chronic Kidney Disease

Stage 1 CKD (GFR > 90 ml/min/1.73m²) Normal or increased GFR, with other evidence of kidney damage

Stage 2 CKD (GFR 60–89 ml/min/1.73m²) Slight decrease in GFR, with other evidence of kidney damage

Stage 3 CKD (GFR 30–59 ml/min/1.73m²) Moderate decrease in GFR, with or without other evidence of kidney damage. Stage 3a is GFR 45–59 ml/min/1.73m² and stage 3b is GFR 30–44 ml/min/1.73m²

Stage 4 CKD (GFR 15–29 ml/min/1.73m²) Severe decrease in GFR, with or without other evidence of kidney damage

Stage 5 CKD (GFR < 15 ml/min/1.73m²) Established renal failure

Case report

A Non-Invasive Papillary Carcinoma arising in a Thyroglossal Duct Cyst

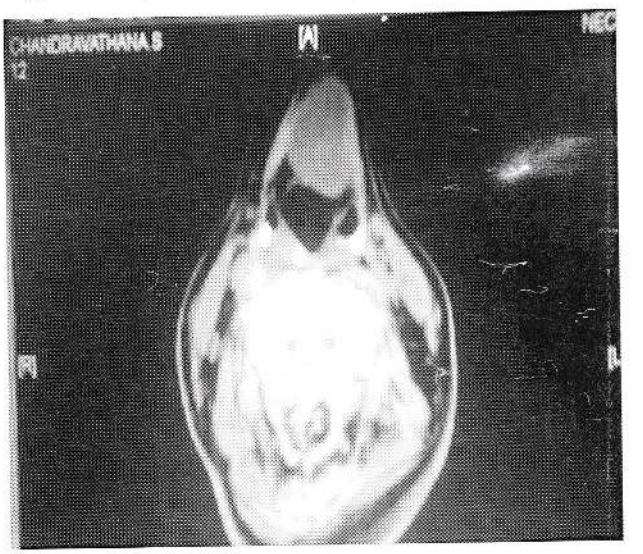
Introduction

The thyroglossal duct cyst (TDCs) is the most common anomaly in the development of the thyroid gland. Seventy percent of thyroglossal duct cysts are diagnosed during childhood and 7% are diagnosed in adulthood. Only 1% of thyroid carcinomas evolve from a thyroglossal duct cyst. (1) Hence, since the first description by Bretano in 1911 until the present, only approximately 250 cases of thyroglossal duct carcinoma have been reported in the relevant literature. (2) Carcinomas arising from a thyroglossal duct cyst are rare and characterized by relatively non-aggressive behaviour and rare lymphatic spread. They are also diagnosed mostly during the third and fourth decades of life. Among repoted cases,papillary carcinoma is the majority (80%)and other form are :mixed papillary-follicular carcinoma , squamous cell carcinoma, follicularcarcinoma, malignant struma, epidermoid carcinoma and anaplastic carcinoma(3)

Case presentation

58year old Sri Lankan women presented with an anterior midline neck lump that gradually appeared without pain over one month duration. The physical examination of the patient revealed a 3cm×2cm mass that was painless, smooth and moving with swallowing. The mass was located in the anterior aspect of the neck at subhyoid region. There was no any lymph node enlargement in anterior or posterior triangle of the neck. Her thyroid gland was looking normal. She was investigated in the view of midline neck lump Ultrasound scan of neck was done that reveal a midline cystic lesion with hyper echoic particle, no lymph node enlargement seen and

Figer01 computerized tomography of neck of the patient

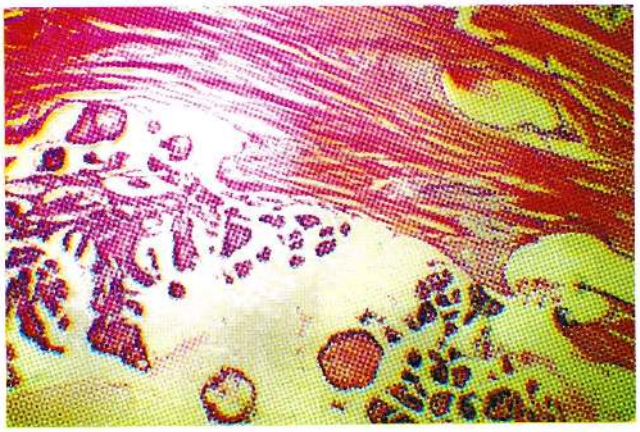


eventually impressed as dermoid cyst in the neck and suggest non contrast computerized tomography which reveal a cystic mass seen in the subhyoid mid line with the foci of calcification, airway not obstructed, no infiltration into surrounding and no any masses seen in the neck including thyroid. (Fig 01)

Elective surgery was done and operative finding was thyroglossal cyst. The cyst has been excised using Sistrunk'sprocedure and sent for histopathology which reveals that a cystic mass measuring 30×20×15 mm, with bony fragment. A small whitish nodule 7mm in diameter is seen in one focus of the cyst containing papillary structures lined by cuboidal cells with clear overlapping nuclei consistent with papillary carcinoma of thyroid.(Fig02) Cyst wall contain normal thyroid follicle.

Then she underwent total thyroidectomy after discussion with oncologist and histopathologist. Her post operative period was uneventful. She has no symptom & signs of hypocalcaemia and her serum

R.Vimalarajan[1], R.Dhanraj[2], S.Raviraj[3],
[1]Registrar, [2]Senior registrar, [3]Consultant surgeon,
Teaching Hospital Jaffna



calcium level was within normal limit. Then she referred to oncologist for further management.

Discussion

Thyroglossal duct cysts usually present as aysmptomatic, soft, firm, or hard masses in the midline of the anterior neck, and are nontender and generally move with the swallowing. Malignant form present in the same manner but Carcinoma should be suspected if it is hard, fixed with adjutant structure and which has undergone any recent change. A history of irradiation treatment during childhood should also arouse suspicion of carcinoma.

Malignant tumours developing from the thyroglossal duct have two origins: thyrogenic carcinoma arising from thyroembrionic remnants in the duct or a cyst, and squamous cell carcinoma arising from metaplastic columnar cells that line the duct. Malignant struma, epidermoid carcinoma and anaplastic carcinoma also reported in some cases. More than 250 cases of thyroglossal duct carcinomas have been reported, in which papillary carcinoma accounts for 80% of cases, with the rest mixed papillary-follicular carcinoma,squamous cell carcinoma and follicular carcinoma. Medullary carcinoma can be excluded, because which arises from parafollicular cells embryologically unrelated to the thyroid. (3)

The main difficulty encountered with a cancer evolving from a thyroglossal duct cyst is that the diagnosis is usually made during surgery or from definitive pathological samples. Because the frequency of cancer of the thyroglossal duct cyst is very low, the surgeons often does not consider an oncologic diagnosis. A second difficulty lies in terms of what approach should be taken during and after surgery when dealing with a preoperatively diagnosed thyroglossal cyst; that is, how extensive should the surgery be and what type of adjuvant therapy should be used To be able to respond to these two issues, the procedure used for thyroglossal cyst surgery must be standarized.

When a thyroglossal duct cyst has been excised using Sis trunk's procedure and when the definitive histological analysis reports malignancy, the thyroid gland must be studied with radiological and scintigraphic examinations and the extension of surgery must be handled according to the criteria established for differentiated thyroid cancer. In our case, radiologically thyroid gland &regional lymphnode were looking normal and we did total thyroidectomy and referred to oncological management with thyroid supression.(5)

The common surgical procedure used for a thyroglossal duct cyst is Sistrunk's procedure, consisting of excision of the thyroglossal duct cyst, the central portion of the body of the hyoid bone, and a core of tissue around the thyroglossal tract to open into the oral cavity at the foramen cecum. In case of malignancy, additional steps consist of thyroidectomy, radioactive iodine and thyroid supression, as is the case for differentiated thyroid cancers. But in some centres they reported as well-differentiated thyroid cancer that is confined to a thyroglossal duct cyst in a patient at low risk for aggressive disease can be adequately treated by a modified Sistrunk procedure.(2)

Conclusion

Malignancy within a thyroglossal duct cyst is very rare but should be included in the differential diagnosis of a neck mass. This condition is seldom diagnosed preoperatively. Once diagnosed, therapy includes surgery, radioactive iodine and thyroid suppression, as is the case for differentiated thyroid cancers.

Consent

Written informed patient consent was obtained for publication.

Reference

1. Geok Chin T, Mohd Sidik Sh, Manickam S, Seng Phang K, Clarence-Ko CH, Jeevanan J:Papillary carcinoma of the thyroglossal duct cyst in a 15-year-old girl.Int J Otorhinolaryngology 2007, 2:732-735.
2. Berni Canani F, Dall'Olio D, Chiarini V, Casadei GP, Papini E, Papillary carcinoma of a thyroglossal duct cyst in a patient with thyroid hemiagenesis: effectiveness of conservative surgical treatment. Endocri Pract 2008, May-Jun; 14(4):465-9.
3. Weiss SD, Orlich CC: Primary papillary carcinoma of thyroglossal duct cyst report of a case and review literature.Br J Surg 1992, 79:1248-1249.
4. Martin-Peres E, Larranaga E, Marron C: Primary papillary carcinoma arising in thyroglossal duct cyst.Eur J Surg 1997, 163:143-145. PubMed Abstract
5. Luna Ortiz K, Hurtado-Lopez LM, Valderrama-Landaeta JL, Ruiz-Vega A: Thyroglossal duct cyst with papillary carcinoma: What must be done? Thyroid 2004, 14:363-366. PubMed Abstract | Publisher Full Text

Sites of Hip Fractures

Hip fractures (or proximal femoral fractures) Fractures occurring between the edge of the femoral head and 5 cm below the lesser trochanter.

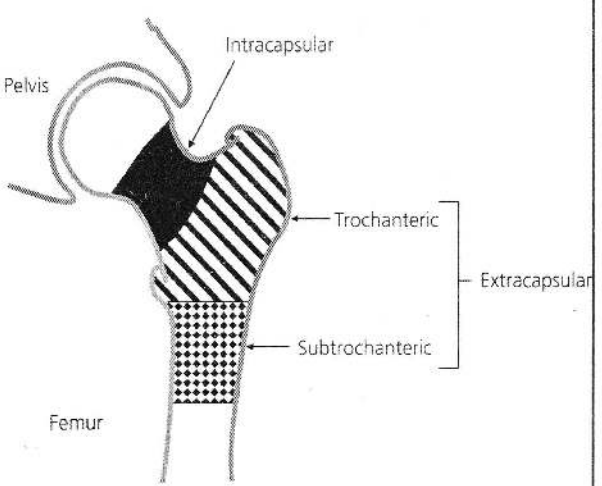
Intracapsular fractures Fractures between the edge of the femoral head and insertion of the capsule of the hip joint. Also known as femoral neck fractures.

Extracapsular fractures Fractures between the insertion of the capsule of the hip joint and a line 5 cm below the lesser trochanter.

Trochanteric fractures A subgroup of the extracapsular group that includes inter- or pertrochanteric and reverse oblique fractures.

Subtrochanteric fractures A subgroup of the extracapsular group where the fracture occurs below the lesser trochanter.

The regions where hip fractures occur



Review

Erectile Dysfunction in Diabetes Mellitus

The prevalence of diabetes is increasing rapidly in Sri Lanka and rest of the developing world, the age of onset is getting lower with more individuals being diagnosed with diabetes in their third and fourth decade[1]. Of these 90 to 95 % are considered to have type 2 diabetes which is strongly associated with changing life styles. Furthermore, childhood type 2 diabetes is rising with the increasing prevalence of childhood obesity[1] so more and more young men are at risk of developing Erectile Dysfunction (ED) as a complication of diabetes. Globally, diabetes affects 5.9% of the adult population but the prevalence is much higher in some ethnic groups for example diabetes is three times commoner among Afro-Caribbeans and five times commoner in people of South Asian origin compared with the white European population living in the UK[2] . The prevalence of diabetes in Sri Lanka is high as 16.4% in urban populations and 8.7% in rural populations[3]. The prevalence of undiagnosed diabetes makes the situation even more bleak: for example in a study involving nine European countries, more than half of the individuals below 50 years with diabetes remained undiagnosed[4]. Furthermore, approximately 200 million people in the world have impaired glucose tolerance, a precursor of diabetes, and this figure is expected to rise to 420 million by 2025[5].

Erectile dysfunction (ED) is a distressing complication of diabetes. The prevalence of ED among diabetic men varies from 35% to 90%[2,6].

Lasantha Malavige
Specialist in Sexual Medicine -Asiri Surgical Hospital
Visiting Academic- Oxford Centre for Diabetes, Endocrinology and Metabolism, University of Oxford, UK
Senior Honorary Research Fellow-
Genetech Research Institute

ED in men with diabetes is more severe and associated with a poorer quality of life[7] and is less responsive to oral treatment[8] than non-diabetic men with ED. In some patients and often among elderly patients, ED can be the presenting symptom of diabetes[9].

The majority of prevalence studies have not distinguished between type 1 and type 2 diabetes, therefore limited data is available to determine the differences between the two forms of diabetes. Bacon et al and Kalter-Leibovici et al reported a similar likelihood of developing ED among men with Type 1 and Type 2 diabetes after adjusting for age[10, 11] unlike a previous report of greater likelihood of ED among men with Type 1 diabetes[12]

Three longitudinal studies have estimated incidence rates of ED in diabetic men. None of the studies exclusively studied type 2 diabetics. The Massachusetts Male Ageing study reported ED incidence of 51/1000 population years[13] and while an Italian study reported incidence of 68/1000 person years among diabetic patients[14]. The smaller third study reported that out of 278 potent at study entry 28% had developed ED at the five year follow up assessment[15].

Risk factors and associations

Advancing age and longer duration of diabetes[16] have consistently been shown to increase the risk of ED. Some cross sectional studies have shown an association between poor glycaemic control and ED[17-19] while others did not find such an association [20]. Poor socioeconomic conditions[21] and sedentary life style[22] have all been shown to increase the likelihood and severity of ED.

Hyperlipidemia[12], hypertension [23] and obesity[24] are conditions that often coexist with diabetes as well as being independent risk factors that have been shown to further increase the risk of ED among diabetic men. Diabetic microvascular complications which include neuropathy, nephropathy and retinopathy[22] and macrovascular complications presented as cardiovascular disease [12] are associated with ED among diabetic men. Various medications, including several antihypertensive agents such as beta blockers and thiazide diuretic, unrelated risk factors such as smoking[12] and excessive alcohol intake have shown to have an additive deleterious effect.

Some studies have found an association between a moderate consumption of alcohol and better erectile function in both general populations [25] and among men with diabetes [10], while others have shown a slightly higher prevalence of ED among alcohol consumers [26]. It is possible that moderate alcohol consumption exerts a protective effect as has been found in ischemic heart disease. Most of these studies were cross sectional and should be interpreted with caution, and prospective studies would be useful to elucidate the true nature of the effect of alcohol on erectile function.

The Pathophysiology of ED in diabetes

The actiology of ED in diabetes is considered to be multifactorial. Multiple physical factors are thought to play a major role but psychological and relationship issues often coexist. Pathophysiologic changes associated with diabetes can broadly be classified as vasculopathy, neuropathy, hypogonadism and local pathological factors. These variables are closely interrelated and their relative significance is not clear.

Psychological and relationship issues

Men with diabetes can have psychosexual and relationship issues predisposing them to develop ED. Diabetes and diabetic complications are associated with depression, a known predictor of ED. The attitude of the diabetic men and their partners towards chronic illness and its complications can also lead to sexual inhibition and arousal problems. Furthermore, the reduction in erectile function due to physical changes associated with diabetes can generate anxiety, making the condition worse.

Organic factors

Vasculopathy

Diabetic vasculopathy consists of macrovasculopathy, microvasculopathy and endothelial dysfunction, all of which play a role in pathophysiology of ED.

Macrovascular disease, a known complication of diabetes, and other co-morbidities commonly associated with diabetes, is thought to play an important role in the pathophysiology of ED by limiting the blood flow to the penis. Lack of NO due to endothelial dysfunction leads to insufficient relaxation of the vascular smooth muscle of the corpora cavernosa resulting in ED.

Peripheral and Autonomic neuropathy

Diabetes is associated with both peripheral and autonomic neuropathy and both of these can contribute to ED. The mechanism for ED in autonomic neuropathy is due to the reduced or absent parasympathetic activity needed for relaxation of the smooth muscle of the corpus cavernosum[27].

Diabetes-associated peripheral neuropathy leads to impairment of sensory impulses from the shaft and the glans of the penis to the reflexogenic erectile centre and impairment of motor nerve impulses to the bulbocavernous and ischiocavenous muscles.

Hypogonadism

Hypogonadism is often associated with type 2 diabetes[28]. Kapoor et al reported that 20% of diabetic men with ED had frank hypogonadism with a total testosterone level below 8 nmol/l and 31% had borderline low total testosterone levels between 8 and 12 nmol/l. In the same study, 50% had free testosterone levels below the recent European Association of Urology guidelines[29]. However, there are uncertainties regarding the level of testosterone needed for proper erectile function.

Peyronie's disease, fibrosis, balanitis and phimosis

Peyronie's disease is common in diabetes mellitus. The underlying mechanism of Peyronie's disease in diabetes is not well known. Further structural changes associated with diabetes include the progressive loss

of normal cavernosal endothelium and smooth muscle cells from corpus cavernosum leading to fibrosis [30].

Balanitis is commonly associated with diabetes. The irritation, pain and discharge associated with fungal balanitis could have both physical and psychological effects on erectile function and intercourse.

Phimosis is common in men with diabetes. A recent study found that 32% of the men presenting to urology clinic with phimosis had diabetes[31]. Phimosis can make intercourse painful and difficult both physically and psychologically.

Management of Diabetic men with ED

Evaluation

The management of diabetic ED can be complex and challenging. Proper sexual, medical and psychosocial evaluation should be carried out in all patients. The presence of premature ejaculation and reduced libido should also be identified, as these three conditions are strongly interrelated[6]. A full drug history should be taken. Diabetic complications, other conditions of co-morbidity and independent risk factors should be identified. The cardiovascular system should be assessed. The assessment of glycaemic control, lipid levels, a full blood count, renal function tests, and serum testosterone are recommended. If the total testosterone level is lower than expected, gonadotrophin levels should be assessed. If the gonadotrophin levels are inappropriately low, for the low testosterone level pituitary function tests should be carried out.

Psychosexual management

Psychosexual and relationship issues should be identified and addressed in all patients. . For many diabetic men with ED, a combined approach is thought to be the most effective, as both organic and psychological factors often coexist. High failure and drop out rates from purely medical management could be improved by combining medical and psychosexual therapy[32].

Risk factor modification

Control of blood glucose and other risk factors which include dyslipidaemia, hypertension and weight is essential in all patients. A combination of medical management and life style changes have been proven to be useful in risk factor modification. Physical exercise, healthy eating, the cessation of smoking should be recommended.

Antihypertensive drugs such as beta blockers and thiazides should be replaced with drugs less associated with ED such as angiotensin converting enzyme inhibitors (ACE inhibitors), angiotension -II receptor antagonists and alpha-adrenoceptor blocking drugs.

Phosphodiesterase -5 inhibitors: Sildenafil, Tadalafil and Vardenafil

All three PDE5 inhibitors have been evaluated for ED in diabetic patients and shown to have similar levels of efficacy, though there have been no head-to-head comparative studies. All PDE5 inhibitors are less efficacious in diabetic men[33-35]. Generally, diabetic patients require the maximum dose of each agent, i.e. sildenafil 100mg, vardenafil 20mg and tadalafil 20mg.

Vasoactive substances

The transurethral application or intracavernousal injection of vasoactive substances can be used to treat ED. The current therapies include papaverine (non specific phospodiesterase inhibitor), phentolamine (alpha adrenorecepetor blocker) and alprostadil (prostaglandin E1). Prostaglandin E1(PGE1) is the most commonly used vasoactive agent in the rest of the world(not yet available in Sri Lanka), which increases the concentration of cyclic adenosine mono phosphate (cAMP) and causes smooth muscle relaxation.

Diabetic patients, particularly those who are on insulin therapy, accept intracavernosal injection therapy more easily and compliance is better compared to non-diabetic men[36].

Trimix, a combination of PGE1, phentolamine and paperverine, has been used when PGE1 alone has failed to induce an erection.

Testosterone supplementation

Recent guidelines recommend testosterone supplementation for symptomatic individuals with 9.00 am total testosterone below 8nmol/l[37]. A trial of testosterone may be considered in symptomatic men with total testosterone levels between 8 and 12 nmol/l [37]Several studies among men with ED of different actiologies[38, 39] and one study in type 2 diabetic

patients[40], all having concomitant hypoganadism, have shown beneficial effects of testosterone supplementation as an additional treatment when PDE5 alone has failed.

Vacuum erection devices and penile implants

This mechanism can be successful with correct technique. The constriction band should not be worn for more than 30 mins. Penile implants are the last resort in ED treatment. Diabetic men are at a slightly higher risk of infection of the prosthesis. The choice of device depends on patient's preference and his ability to manipulate the device. However, among diabetic men malleable implants should be avoided due to high risk of erosion.

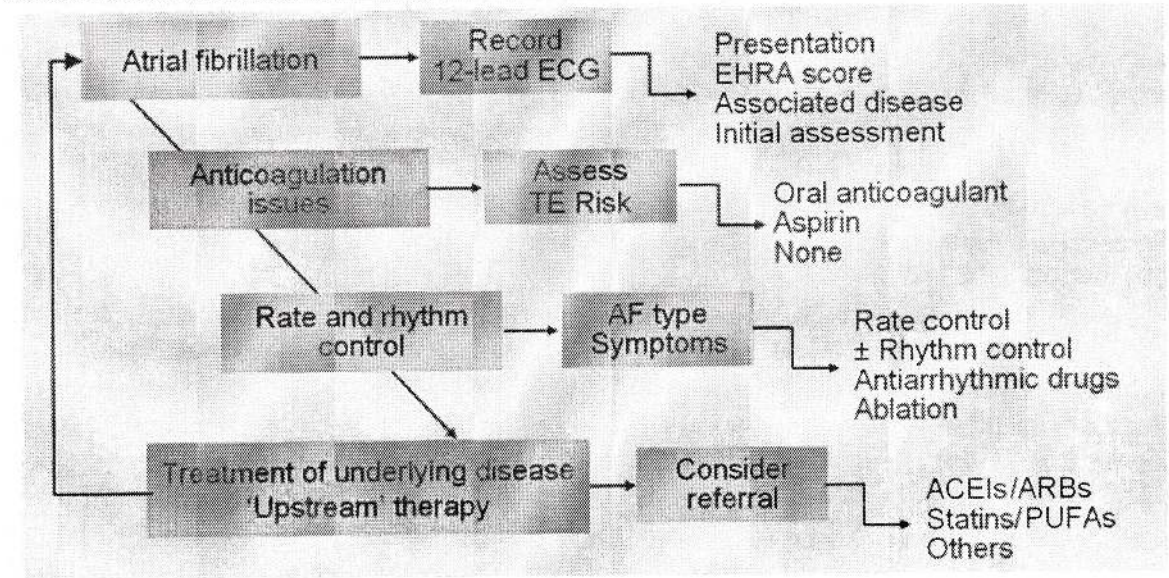
References

1. Alberti G, Zimmet P, Shaw J, Bloomgarden Z, Kaufman F, Silink M. Type 2 diabetes in the young: the evolving epidemic: the international diabetes federation consensus workshop. *Diabetes Care*. 2004;**27**: 1798-811.
2. Malavige LS, Levy JC. Erectile dysfunction in diabetes mellitus. *J Sex Med*. 2009;**6**: 1232-47.
3. Katulanda P, Constantine GR, Mahesh JG, Sheriff R, Seneviratne RD, Wijeratne S, Wijesuriya M, McCarthy MI, Adler AI, Matthews DR. Prevalence and projections of diabetes and pre-diabetes in adults in Sri Lanka--Sri Lanka Diabetes, Cardiovascular Study (SLDCS). *Diabet Med*. 2008;**25**: 1062-9.
4. Age- and sex-specific prevalences of diabetes and impaired glucose regulation in 13 European cohorts. *Diabetes Care*. 2003;**26**: 61-9.
5. Hossain P, Kawar B, El Nahas M. Obesity and diabetes in the developing world--a growing challenge. *N Engl J Med*. 2007;**356**: 213-5.
6. Malavige LS, Jayaratne SD, Kathriarachchi ST, Sivayogan S, Fernando DJ, Levy JC. Erectile dysfunction among men with diabetes is strongly associated with premature ejaculation and reduced libido. *J Sex Med*. 2008;**5**: 2125-34.
7. Penson DF, Latini DM, Lubeck DP, Wallace KL, Henning JM, Lue TF. Do impotent men with diabetes have more severe erectile dysfunction and worse quality of life than the general population of impotent patients? Results from the Exploratory Comprehensive Evaluation of Erectile Dysfunction (ExCEED) database. *Diabetes Care*. 2003;**26**: 1093-9.
8. Goldstein I, Lue TF, Padma-Nathan H, Rosen RC, Steers WD, Wicker PA. Oral sildenafil in the treatment of erectile dysfunction. Sildenafil Study Group. *N Engl J Med*. 1998;**338**: 1397-404.
9. Lewis RW. Epidemiology of erectile dysfunction. *Urol Clin North Am*. 2001;**28**: 209-16, vii.
10. Kalter-Leibovici O, Wainstein J, Ziv A, Harman-Bohem I, Murad H, Raz I. Clinical, socioeconomic, and lifestyle parameters associated with erectile dysfunction among diabetic men. *Diabetes Care*. 2005;**28**: 1739-44.
11. Bacon CG, Hu FB, Giovannucci E, Glasser DB, Mittleman MA, Rimm EB. Association of type and duration of diabetes with erectile dysfunction in a large cohort of men. *Diabetes Care*. 2002;**25**: 1458-63.
12. Fedele D, Coscelli C, Santeusanio F, Bortolotti A, Chatenoud L, Colli E, Landoni M, Parazzini F. Erectile dysfunction in diabetic subjects in Italy. Gruppo Italiano Studio Deficit Erettile nei Diabetici. *Diabetes Care*. 1998;**21**: 1973-7.
13. Johannes CB, Araujo AB, Feldman HA, Derby CA, Kleinman KP, McKinlay JB. Incidence of erectile dysfunction in men 40 to 69 years old: longitudinal results from the Massachusetts male aging study. *J Urol*. 2000;**163**: 460-3.
14. Fedele D, Bortolotti A, Coscelli C, Santeusanio F, Chatenoud L, Colli E, Lavezzari M, Landoni M, Parazzini F. Erectile dysfunction in type 1 and type 2 diabetics in Italy. On behalf of Gruppo Italiano Studio Deficit Erettile nei Diabetici. *Int J Epidemiol*. 2000;**29**: 524-31.
15. McCulloch DK, Young RJ, Prescott RJ, Campbell IW, Clarke BF. The natural history of impotence in diabetic men. *Diabetologia*. 1984;**26**: 437-40.

16. McCulloch DK, Campbell IW, Wu FC, Prescott RJ, Clarke BF. The prevalence of diabetic impotence. *Diabetologia*. 1980;**18**: 279-83.
17. Roth A, Kalter-Leibovici O, Kerbis Y, Tenenbaum-Koren E, Chen J, Sobol T, Raz I. Prevalence and risk factors for erectile dysfunction in men with diabetes, hypertension, or both diseases: a community survey among 1,412 Israeli men. *Clin Cardiol*. 2003;**26**: 25-30.
18. Romeo JH, Seftel AD, Madhun ZT, Aron DC. Sexual function in men with diabetes type 2: association with glycemic control. *J Urol*. 2000;**163**: 788-91.
19. Matfin G, Jawa A, Fonseca VA. Erectile dysfunction: interrelationship with the metabolic syndrome. *Curr Diab Rep*. 2005;5: 64-9.
20. Siu SC, Lo SK, Wong KW, Ip KM, Wong YS. Prevalence of and risk factors for erectile dysfunction in Hong Kong diabetic patients. *Diabet Med*. 2001;18: 732-8.
21. Malavige LS JS, Sivaogan S,Kathriarachchi S,Fernando DJ,Levy JC. Prevalance,clinical and socioeconomic assocaitaions of erectile dysfunction among Type 2 diabetic patients in Sri Lanka. *Daibetes UK,Annual Professional conference* Birmingham,UK: Diabetic Medicine; 2006.
22. Zheng H, Fan W, Li G, Tam T. Predictors for erectile dysfunction among diabetics. *Diabetes Res Clin Pract*. 2006;71: 313-9.
23. De Berardis G, Pellegrini F, Franciosi M, Belfiglio M, Di Nardo B, Greenfield S, Kaplan SH, Rossi MC, Sacco M, Tognoni G, Valentini M, Nicolucci A. Identifying patients with type 2 diabetes with a higher likelihood of erectile dysfunction: the role of the interaction between clinical and psychological factors. *J Urol*. 2003;169: 1422-8.
24. El-Sakka AI, Tayeb KA. Erectile dysfunction risk factors in noninsulin dependent diabetic Saudi patients. *J Urol*. 2003;169: 1043-7.
25. Ahn TY, Park JK, Lee SW, Hong JH, Park NC, Kim JJ, Park K, Park H, Hyun JS. Prevalence and risk factors for erectile dysfunction in Korean men: results of an epidemiological study. *J Sex Med*. 2007;4: 1269-76.
26. Derby CA, Mohr BA, Goldstein I, Feldman HA, Johannes CB, McKinlay JB. Modifiable risk factors and erectile dysfunction: can lifestyle changes modify risk? *Urology*. 2000;56: 302-6.
27. Pegge NC, Twomey AM, Vaughton K, Gravenor MB, Ramsey MW, Price DE. The role of endothelial dysfunction in the pathophysiology of erectile dysfunction in diabetes and in determining response to treatment. *Diabet Med*. 2006;23: 873-8.
28. Corona G, Mannucci E, Mansani R, Petrone L, Bartolini M, Giommi R, Forti G, Maggi M. Organic, relational and psychological factors in erectile dysfunction in men with diabetes mellitus. *Eur Urol*. 2004;46: 222-8.
29. Kapoor D, Aldred H, Clark S, Channer KS, Jones TH. Clinical and biochemical assessment of hypogonadism in men with type 2 diabetes: correlations with bioavailable testosterone and visceral adiposity. *Diabetes Care*. 2007;30: 911-7.
30. Jevtich MJ, Khawand NY, Vidic B. Clinical significance of ultrastructural findings in the corpora cavernosa of normal and impotent men. *J Urol*. 1990;143: 289-93.
31. Bromage SJ, Crump A, Pearce I. Phimosis as a presenting feature of diabetes. *BJU Int*. 2008;101: 338-40.
32. Malavige LS LJ. Erectile Dysfunction in Diabetic Men- Medical and Psychosexual Review. *Institute of Psychosexual Medicine Journal*. 2008;47: 10-15.
33. Ng KK, Lim HC, Ng FC, Li MK, Consigliere D, Chia SJ, Moorthy P, Munisamy M. The use of sildenafil in patients with erectile dysfunction in relation to diabetes mellitus--a study of 1,511 patients. *Singapore Med J*. 2002;43: 387-90.
34. Padma-Nathan H. Efficacy and tolerability of tadalafil, a novel phosphodiesterase 5 inhibitor, in treatment of erectile dysfunction. *Am J Cardiol*. 2003;92: 19M-25M.
35. Goldstein I, Young JM, Fischer J, Bangerter K, Segerson T, Taylor T. Vardenafil, a new phosphodiesterase type 5 inhibitor, in the treatment of erectile dysfunction in men with diabetes: a multicenter double-blind placebo-controlled fixed-dose study. *Diabetes Care*. 2003;26: 777-83.

36. Perimenis P, Gyftopoulos K, Athanasopoulos A, Barbalias G. Diabetic impotence treated by intracavernosal injections: high treatment compliance and increasing dosage of vaso-active drugs. *Eur Urol.* 2001;40: 398-402; discussion 03.
37. Nieschlag E, Swerdloff R, Behre HM, Gooren LJ, Kaufman JM, Legros JJ, Lunenfeld B, Morley JE, Schulman C, Wang C, Weidner W, Wu FC. Investigation, treatment and monitoring of late-onset hypogonadism in males. ISA, ISSAM, and EAU recommendations. *Eur Urol.* 2005;48: 1-4.
38. Shabsigh R, Rajfer J, Aversa A, Traish AM, Yassin A, Kalinchenko SY, Buvat J. The evolving role of testosterone in the treatment of erectile dysfunction. *Int J Clin Pract.* 2006;60: 1087-92.
39. Aversa A, Isidori AM, Spera G, Lenzi A, Fabbri A. Androgens improve cavernous vasodilation and response to sildenafil in patients with erectile dysfunction. *Clin Endocrinol (Oxf).* 2003;58: 632-8.
40. Kalinchenko SY, Kozlov GI, Gontcharov NP, Katsiya GV. Oral testosterone undecanoate reverses erectile dysfunction associated with diabetes mellitus in patients failing on sildenafil citrate therapy alone. *Aging Male.* 2003;6: 94-9.

The Management cascade for patients with AF



Guide to Authors

Papers are accepted on the understanding that they have not been and will not be published elsewhere, as they become the copyright of the journal if accepted and cannot be produced elsewhere without permission. Three copies of the manuscript and its electronic version should be submitted. Papers should be sent to :

The Editor
The Jaffna Medical Journal
JMA Library
Teaching Hospital Jaffna
email: plakshman@fastmail.fm

Title page should include title of paper , Author (s) name (s) with Initials , qualifications, place of work , place where the work was carried out , address of author who will deal with correspondence .

Text should preferably have summary of not more that 150 words. 3-6 key words at the end of the abstract, introduction, materials and methods, statistics (details of method if used), results and discussion, acknowledgments.

Tables: Each table should be double spaced and typed on separate sheets of paper. Tables should be numbered in arabic numerals, consecutively, in the order of their first citation in the text and supply a brief title for each. Each column should be given an abbreviated heading.

References: Should be numbered in arabic numerals in the order they are first cited in the text. Use the Vancouver style.

Disclaimer : Neither the editor nor the publishers accept responsibility for the views of the author (s) expressed in their contribution.

Address all correspondence to

Editor,
The Jaffna Medical Journal
JMA Library,
Teaching Hospital Jaffna.
email: plakshman@fastmail.fm

Mega Pharma (Pvt) Ltd.

A mega commitment to Medicine