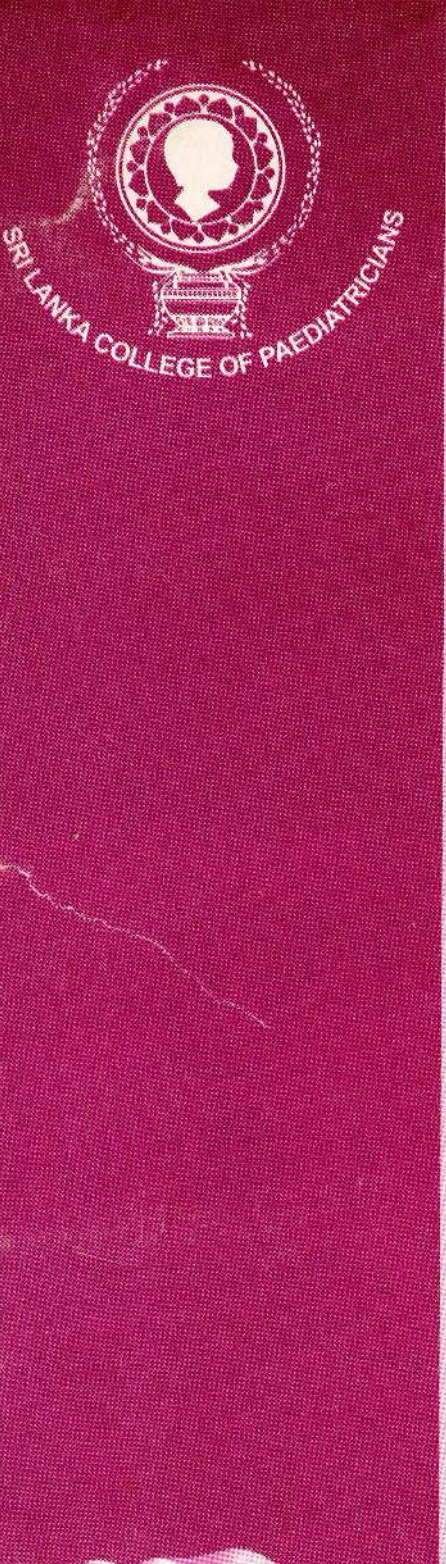

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CONTENTS Page

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Editorial

Accidental childhood poisoning and the environment

Sri Lanka Journal of Child Health, 2005; **34**:104-6

Accidental poisoning of a child is a complex interaction between the child, a hazardous substance and certain environmental situations[1].

The child

In all societies where statistics have been compiled, the 2 and 3 year old children account for over half of all reported poisonings, 80-90% of them being under the age of 5 years[2,3,4,5].

Characteristically children who are poisoned are more likely to be impulsive and overactive and are discipline problems for their parents. Not infrequently the parent-child relationship is disturbed[1]. Children are particularly prone to accidental poisoning when usual family patterns are interrupted, during such episodes as moving, pregnancy, illness, death, marital problems or visiting another home[1].

More than half the time, the child ingests a toxic agent that is in his clear view. A child tends to react to his environment impulsively, seeking what he wants when he wants it[1]. A toddler may not be innocent or ignorant when he secretively gobbles down a bottle of paracetamol syrup or samples granules of caustic detergent; however he is certainly ignorant of the consequences[1].

In many instances the possibility of poisoning is related to the developmental patterns of the child[1]. The 6 month old will put anything in his mouth. A 1–2 year old will empty cupboards, particularly low ones, and experimentally taste most things. The 3 year old child is adventurous and has virtual access to any unlocked drawer or cupboard in the home. All challenges are accepted in impulsive or ingenious ways[1]. Towards the end of the third year of life and by the age of 4, the number of accidental ingestions start to decline despite an increase in motor ability. The 4 year old child tends to be more selective in what he ingests, preferring those things that taste good including flavoured children's paracetamol, vitamins and candy coated tablets[1].

To prevent poisoning, programmes must be developed that will completely protect the child aged 3 and under. When he is approaching age 4 he will, if he is taught, understand simple safety rules and have enough good sense not to eat or sample everything that he comes across in the home. From the age of 4 on, self-control through education is the primary deterrent to poisoning[1].

Parents should strive for complete and instant obedience to rules of safety early in the child's life. Rules of obedience offer a form of guidance that a child must have to grow up free from serious injury. It is much better to go through life with a questionably scarred ego than a very real scarred oesophagus which will require a lifetime of repeated dilatations[1].

The hazardous substance

Most poisonings in children are from ingestion of toxic substances in the home, stored in accessible places[2,3,4,5]. In industrialized countries such as Germany, France, Italy, United States, England and Canada the average home contains over 30 containers of medications[6]. Thus, it is not surprising that in those countries the most common substances reported in accidental poisonings are medications. In New Zealand, agricultural chemicals are a prominent cause of accidental poisoning[1]. In India and Sri Lanka kerosene is a principal toxin[2,3]. In one study in India kerosene accounted for 60% of poisoning[2].

Kerosene oil ingestion was the leading cause of poisoning in Sri Lankan children, accounting for 35% of the total[3,7]. The frequency of kerosene oil poisoning can be attributed to several factors[7]. Firstly, kerosene oil is stored in almost every home. It is stored in bottle lamps, plastic cans, miscellaneous bottles, barrels, tins, cups and coconut shells. Secondly, kerosene oil containers are often kept in easily accessible places such as the kitchen floor, a low table or a low shelf. Thirdly, kerosene containers are hardly ever stoppered. Fourthly, due to its blue colour, children often mistake kerosene oil for bottled soft drinks[7].

In addition to kerosene oil there is a plethora of potential toxins in the home which are a ready source of potentially toxic material for ingestion by young children[2,3,4]. Agrochemicals especially are either stored within the house or in garages and are easily accessible to children [2,3].

Anticonvulsants and psychiatric drugs are usually prescribed on a fortnightly or monthly basis and as a result are available in bulk in the house. Furthermore, children see adults taking these drugs on a regular basis and tend to imitate them. Thus it is not surprising that these drugs are so frequently ingested by children[3]. People have a habit of throwing unused tablets on to the garden or road. Some children in my series were poisoned due to ingestion of these tablets[3]. It is not certain whether colouring and sugar coating play a decisive role in accidental poisoning. However, it is unwise to add attractions to tablets known to be harmful. For instance, in my series a toddler had ingested 20 paracetamol tablets of the lozenge type[3]. A sweet taste, in fact, may be more dangerous than colour. Drugs for local application such as surgical spirits, cetrimide lotion, camphor oil, methyl salicylate lotion and calamine lotion were often dispensed at outpatient departments of government hospitals into containers brought by parents and no labelling whatsoever had been done. At home these unlabelled containers were often kept alongside the baby's cough syrup or gripe water bottle on the same shelf or table[3]. It is thus not surprising that these substances were mistakenly administered to the child by an adult.

Although ingestion of poisonous plants accounted for only 10% of childhood poisoning overall, they accounted for 55% of poisoning by school children in my series[3]. Of the poisonous plants ingested the commonest were 'weta endaru' and 'beheth endaru', which together accounted for 43% of cases[8]. These plants along with yellow oleander are commonly found in roadside hedges and are thus freely accessible to children[8]. Plants of the Habarala variety are found in the home garden[8]. The close resemblance of Hondala fruit to passion fruit and the Niyangala tuber to sweet potato (bathala) is responsible for accidental poisoning among children[8]. Poisonous species of mushroom grow wherever edible varieties are to be found and bear some resemblance to each other making mistaken identification common[8].

Whilst emphasis in most reported accidental childhood poisoning is on ingestion of solids and liquids, inhalation of poisonous gases, fumes and smoke also produces poisoning[9]. The most common offender is carbon monoxide, which may arise from a defective auto exhaust system or a smouldering fire in a confined area. More than one child allowed to sleep on the back seat of an automobile have been poisoned from carbon monoxide seeping into a running automobile from a defective muffler[9].

Environmental factors

A number of environmental factors interrelate with the child and the hazardous substance to end up with poisoning. they include such things as time of day, relationship to meals, whether product is in or out of sight, recent experience with substance, family stress and parental attitudes toward the toxin[1]. There is a common belief among the general public and professionals that careless storage is a major factor in the causation of accidental poisoning, yet there is no clear cut evidence that this is true[1]. In a study reported by Sobel[10], of 400 families (122 families with a history of poisoning and 278 controls) no significance was found (1) in the degree of hazard in poisoned vs. non-poisoned homes; (2) in storage habits between poisoned and non-poisoned groups; or (3) in storage habits 1 year later even though there may have been a poisoning in the interim period.

How careful are physicians having young children with storage of medications in their own homes? One would think that paediatricians in particular would be quite sensitive to safe storage practices. However, a surprise survey carried out in the USA of medications in the homes of 12 paediatricians with young children disclosed that only one paediatrician had all his medications securely locked up[1]. In this sample there was a common disregard in the homes of practicing paediatricians of a basic principle of poison prevention extolled to their patients "Keep all medications out of reach of children". Yet it would seem pure heresy to recommend that parents disregard safe storage principles. In a similar study of 52 poisoned and 52 control families, Baltimore et al[11] found no significant differences in the poisoned and control groups in storage habits or the mother's knowledge regarding toxicity.

In all reported studies the vast majority of toxic substances in the home associated with accidental poisoning were accessible and packaged in containers easily opened by young children[2,3,4,5]. In this hostile home environment the child must develop self control early if he is to survive. However, we are far from achieving the goal of establishing self control in early childhood for all children. The alternative is to increase our protective efforts through elimination of useless toxins in the home, safer packaging and parent education while attempting to find better methods to control undesirable childhood impulses[1].

Family stress is an important factor in accidental ingestion of poisons in childhood[12]. It may make poisons readily available to children either because parents under stress are less careful or because medicines are being used during an illness. Another possibility is that the child's behaviour is altered when there is unhappiness in the home with disordered family relationships[12].

References

1. Scherz RG. Prevention of childhood poisoning: a community project. *Pediatric Clinics of North America* 1970; **17 (3)**: 713-27.

2. Buharwella RJ. Poisoning in children, a study of 303 cases. *Indian Pediatrics* 1969; **6**: 141-5.

3. Lucas GN. Acute childhood poisoning in Sri Lanka; a hospital based prospective study. *Ceylon Journal of Child Health* 1991; **20**: 4-12.

4. Moore EJ. Accidental poisonings in the home. *Canad J Pub Health* 1966; **57**: 126-8.

5. Varese LA. Accidental acute poisonings caused by medicinal substances in childhood. Minerva Pediatrica 1968; **20**: 1324.

6. Scherz RG, Latham GH, Stracener CE. Child-resistant containers can prevent poisoning. Pediatrics 1969; **43**: 84.

7. Lucas GN. Kerosene oil poisoning – the direful predicament of the Sri Lanka toddler. *Ceylon Journal of Child Health* 1992; **21**: 9-16.

8. Lucas GN. Plant poisoning; a hospital based prospective study in Sri Lanka. *Indian Journal of Pediatrics* 1992; **64**: 495-502.

9. Butler J. Perils of poisoning. *Postgraduate Medicine* 1968; **44**: 119-22.

10. Sobel R. Traditional safety measures and accidental childhood poisoning. Pediatrics 1969; **44**: 811.

11. Baltimore C, Meyer RJ. A study of storage, child behavioural traits and mother's knowledge of toxicology in 52 poisoned families and 52 comparison families. Pediatrics 1969; **44**: 291.

12. Sibert JR. Stress in families of children who have ingested poisons. *British Medical Journal* 1975; **3**: 87-9.

G N Lucas
Joint Editor

Leading Article

Functional, non-organic symptoms in children and young people

T L Chambers[1]

Sri Lanka Journal of Child Health, 2005;**34**:107-8

Chambers Dictionary defines functional as: "characterised by impairment of function, not of organs". Organic disease is one "accompanied by changes in structures involved" – presumably those structures might also be genetic, biochemical, immunological or organs which have been damaged by malfunction of those non-anatomical influences. Non-organic symptomatology – or semeiology, to be precise - is therefore unaccompanied by structural change. Such symptoms make up a large part of the work of doctors at all levels of care, may result in severe incapacity of sufferers and their families and may lead to serious misunderstanding and disharmony between the patient/carers and the health professionals. The 20th century specialist paediatrician's approach was often "rule out and discharge" – perhaps to a child psychiatrist. The child/young person's views may not have been taken account of and the parents felt there were no alternative options. 21st century medicine is about patient and carer empowerment, choice and the validity of the patient experience. It is time to recast our thinking about this group of conditions and recognise them as general medical rather than primarily psychological.

Common childhood* symptoms which present to primary care physicians, either together or singly include pain – head, chest, abdominal and limb -, tiredness, lightheadedness, sweatiness, nausea, double vision, sensitivity to light, noise and touch - and others. All of them are listed in the symptomatic diagnostic workup list for many organic diseases. The seasoned diagnostician relies heavily on a careful history concerning these and other complaints in formulating a provisional diagnosis. Likewise a thorough physical examination not only reassures the patient but also the doctor that it is unlikely that there is an organic condition present. Pointers to functional conditions include hyperasthesia to skin touch, and multifocal symptoms and pain sites with normal examination findings. From such a painstaking and methodical clinical examination a discerning choice of investigations may be made.Some adopt a rule-out approach and arrange a large number of laboratory and imaging tests "just in case". The drawback here – not including the cost of such a policy – is that tests may give equivocal results and then lead to further, more expensive, invasive and hazardous procedures. Tests should come as a result of asking precise clinical questions whose answers will influence management. Few would do none, but going beyond a blood count, acute phase protein, urinalysis and perhaps renal and liver function risks trouble for patient and doctor. Things may be more difficult if functional symptoms complicate a pre-existing classifiable disease. Although the general medical assessment should include mental and emotional health, in selected cases I explain to patients and carers that a psychological assessment is just as important a test as a scan because mental illness may have somatic symptoms. However I emphasise that – as with other tests - it is done with an open mind in order to assist in diagnosis.

It should be easy to make a diagnosis of a functional condition at a single consultation, particularly if the clinic organisation is "one stop". One may then engage the patient and carers in a discussion about the cause of the symptoms as soon as possible. This discussion may be troubled: is it good news to learn that symptoms which might have led to school non-attendance, family disruption and refocusing and an unhappy and bewildered young person do not have cause for which there is specific treatment? Some families take it as bad news. Much depends on how the physician handles that crucial consultation. The patient and family must not get the impression of professional disbelief in the symptoms, disparagement or disengagement. Just as with all other medical conditions a firm and positive diagnosis should be given, and explanation of why the symptoms occur and what needs to be done about them. My approach is to explain that much of what afflicts people does not have a serious or scary cause, for example needing a hospital stay or an operation – although functional symptoms resulting in prolonged school absence *are* serious. Moreover, the nervous system may experience unpleasant feelings both when there is identifiable disease present and when not – the phantom limb example is sometimes helpful. At this stage it is vital to get agreement from all that the diagnosis is understood, acceptable and one from which symptomatic treatment might follow. If this is not so then the doctor will have to explore why and how agreement might be achieved: this might mean a further test – perhaps to make sure a condition that causes

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specific anxiety is not present – or another opinion. If this is done with sympathy and authority (not authoritarian-ness) in partnership with patient and carers it is constructive. If it results from friction, professional hubris and the striking of attitudes, particularly between doctor and parents, no good will come, least of all to the patient.

Management of functional problems is by (re)habilitation. A team approach may be helpful: therapists to address the specific incapacities, including disinclination to eat (dietitian), contact with education so that the young person may remain in touch with school even if they cannot attend fully. In this and all other aspects of habilitation it is imperative to go at the patient's pace – indeed one might argue that this is a basic right of young people. It is duplicitous to tell a person one believes in their incapacities and then impose treatments which they cannot tolerate. Young people seldom experience gain from their problems and want to be back to their old selves. No harm comes from such an approach. In this and all other chronic paediatric conditions psychological support is important. This is provided by the family and the general paediatrician but may often be usefully supplemented by focused psychological or psychiatric input. One should remember that chronic illness may lead to depression which needs skilled treatment in its own right. Occasionally physical interventions are needed – splints, TENS machines for example. Pain should be managed proactively: if simple analgesics do not work then pain modifying drugs such as anticonvulsants service may be needed. If there is access to a paediatric pain and symptomatology team, so much the better. However the generalist should remain involved with the patient to co-ordinate services, review with an eye on complications and advocate on the patient's behalf. Of course many patients will not need the full service of a multidisciplinary team: they, the paediatrician and the parents will work with simple behavioural and pharmacological tools to achieve success. Complementary medicine and other interventions are used widely in the UK; my view is that if they are found to be beneficial, so be it – but do not reject the patient because they roam wider than your patch: be there to support them – and to put the internet into perspective.

A word about child protection. In some children functional symptoms may result from abuse, physical or emotional. Moreover, the attitude of some families to their child's symptoms might suggest factitious illness. Some might regard the parents of a functionally ill child who solicit multiple medical opinions as being *de facto* abusive (despite the professional collusion that it sometimes revealed). None of this can be denied but the magnitude of this problem seems small and it would be wrong and dangerous to assume there is abuse just because incapacitating symptoms present without any obvious cause. If there are well founded concerns local child protection policies should be followed.

Functional conditions are rewarding to work with: they challenge parts of paediatrics that organ oriented specialties do not reach and they are for the open minded and clinically ingenious physician. This is because medical history is littered with examples of functional conditions for which an organic explanation emerges: how many of Apley's "little belly-achers" had H Pylori gastritis or non-coeliac wheat intolerance? It is therefore also an important area for research, especially interdisciplinary. It lacks great evidence base; clinical pathways are not well trodden and the doctor may sometimes feel professional loneliness (but not as lonely as a parent of an unwell child who feels ignored or demeaned). However our medical paediatric calling expects that we should stick with and help these children and their families – they may have nobody else to turn to.

* I use the term child, children or childhood as shorthand for children and young people.

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A survey on febrile seizures at the Lady Ridgeway Hospital for Children

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(Key words: febrile seizures, children)

Abstract

Objectives To evaluate demographic, clinical and management aspects of febrile convulsions.

Method A prospective study was carried out on all children admitted to Lady Ridgeway Hospital with a diagnosis of febrile convulsions during June and July 2002. Children with a history of afebrile seizures and those with evidence of a neurodevelopmental deficit or central nervous system infection were excluded. Data was obtained from medical records and direct interview of parents/guardians of children with febrile convulsions using a pre-tested validated questionnaire.

Results 330 children were admitted with febrile convulsions. Male to female ratio was 3:2. The mean age was 22 months. Approximately 25% had a history of febrile convulsions in first degree relatives. 25% had complex febrile seizures. 80% of seizures occurred within 24 hours of the onset of fever. 24 (7%) children received long term prophylaxis for recurring febrile convulsions. Upper respiratory tract infection was the commonest trigger factor. 48% of the parents/guardians did not have a satisfactory knowledge of first aid.

Introduction

The International League Against Epilepsy (ILAE) defines a febrile seizure as "a seizure occurring in childhood after one month of age, associated with a febrile illness not caused by an infection of the central nervous system, without previous neonatal seizures or a previous unprovoked seizure, and not meeting criteria for other acute symptomatic seizures".

Febrile seizures recur in 30-40%[1], risk of subsequent epilepsy is low[2] and the neurological outcome is excellent[1]. There appears to be a dearth of published studies on febrile convulsions in Sri Lanka. The aim of this study was to evaluate demographic, clinical and management aspects of febrile convulsions.

Method

All children admitted to Lady Ridgeway Hospital (LRH) with a diagnosis of febrile convulsions were prospectively studied during June and July 2002. Children with a past history of afebrile seizures and those with evidence of a neurodevelopmental deficit or central nervous system infection were excluded. Data was collected, using a pre-tested validated questionnaire, from medical records and direct interview of parent/guardian of children with febrile convulsions.

Simple febrile seizures were defined as generalised seizures lasting less than 15 minutes and which did not recur within a day. Complex febrile seizures were defined as focal or multiple seizures or seizures with duration >15 minutes or a combination of these.

Results

330 (3%) of all admissions to LRH were due to febrile convulsions. Male to female ratio was 3:2. Thirty nine percent of children with febrile seizures were less than one year of age. The mean age was 22 months. In 1.5% onset was after 5 years of age. In 2% febrile convulsions persisted beyond 5 years (Table 1). Approximately 25% had a history of febrile convulsions in first degree relatives. Only 4 (1.2%) children with febrile seizures had a positive family history of epilepsy in first-degree relatives (Table 1).

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75% had simple and 25% had complex febrile seizures. 29% of infants showed atypical seizures. Among children who had complex seizures 88% had multiple seizures, 5% had focal seizures and 17% had prolonged seizures (lasting more than 15 minutes). In 59% the first seizure was the longest. 80% of seizures occurred within 24 hours of the onset of fever (Table 2).

57 (17%) children had been given anti-convulsants prior to hospital admission either by a general practitioner or at the outpatient department and 4 of those children received diazepam intramuscularly (Table 3). Although 15 children had been prescribed short course diazepam prophylaxis, only 2 of them were given diazepam by their parents with the current febrile illness. 24 (6.7%) children received long term prophylaxis for recurring febrile convulsions, 67% with sodium valproate and 25% with carbamazepine (Table 3).

Upper respiratory tract infection was the commonest trigger factor, diagnosed in 36% of cases. Other trigger factors included lower respiratory tract infections, otitis media, acute gastro-enteritis and urinary tract infections (Table 4). Forty eight percent of the parents/guardians did not have satisfactory knowledge on first aid.

Discussion

Febrile convulsions occur in 2-4% of children[3]. Although the incidence in Sri Lanka is not known, it is evident that febrile convulsions account for a significant proportion of morbidity and hospital admissions. In this study, age range of the population was 2 months to 11 years. 129 (39%) were below one year of age. In 2% of children convulsions continued beyond 5 years of age while in 1.5% onset of febrile seizures was after 5 years of age. According to Webb *et al* only 10% developed epilepsy later on in this age category[4]. 25% had an affected first degree relative with febrile convulsions and it is comparable to the available information in the literature[5,6,7,8].

25% had atypical febrile convulsions. It is useful to follow up these children to assess the prognosis. According to Annergers *et al* atypical seizures are predictors of subsequent epilepsy and the risk ranged from 2.4% among children with simple febrile convulsions to 49% among children with all three complex features of febrile convulsions[2].

29% of infants had atypical febrile seizures. The most difficult part in evaluation is the decision regarding the need for a lumbar puncture in these infants. Because of the unreliability of the meningial signs in small children several studies give various cut-off points ranging from 6 months to 36 months for the age below which a lumbar puncture is recommended[9,10]. Some studies indicate that children with complex seizures[11] and children pre-treated with antibiotics have the highest risk of bacterial meningitis. Therefore, individual clinical decisions should guide the need for lumbar puncture rather than arbitrary cut off points for age.

In this study majority of children had a clinical diagnosis of viral infection. A minority were treated with antibiotics for lower respiratory tract infections, acute otitis media and urinary tract infections. These findings are compatible with available information[12].

It is noted that 4 children received diazepam intramuscularly in acute management highlighting the necessity for education of healthcare workers to avoid ineffective routes of diazepam administration.

24 children with recurrent convulsions were on long term prophylaxis. Majority (16) of them were on sodium valproate while 6 of them were on prophylactic carbamazepine. A meta-analysis of all published randomised, placebo-controlled trials of the preventive treatment of febrile seizures shows that valproate and phenorbarbitone are effective in prevention of recurrences, but both agents have known adverse effects and cannot be recommended for prevention of febrile seizures[13]. According to this analysis no difference in risk was found for recurrences between children receiving intermittent diazepam and placebo[13]. Many studies have shown that phenytoin and carbamazepine have no place in prophylaxis [13,14].

Almost half of the parents did not have adequate knowledge on febrile convulsions highlighting the need for parental health education. There is evidence that, parents' poor knowledge, negative attitudes, anxiety, and inadequate first-aid measures toward febrile convulsions can be effectively improved by parent education[15].

Conclusions

- Febrile seizure disorder is an important health problem in Sri Lanka. Often it accounts for parental anxiety and childhood morbidity.

- ❑ According to many randomised controlled trials long-term prophylaxis is not recommended due to the side effects and should only be used in ighlyselected cases, if at all. Sodium valproate and phenorbarbitone are the drugs of choice. Intermittent diazepam prophylaxis has no proven effect[13,14].

- ❑ Parents need to be counselled and educated at the time of the seizure. A leaflet with important details can be useful to reduce the workload of the busy healthcare worker.

- ❑ Further studies are recommended to evaluate prognosis of atypical febrile seizures and febrile seizures with onset after 5 years of age. It is also important to look further at children less than one year with fever and convulsions, since there are difficulties in diagnosis and management.

References

1. Nelson K B, Ellenberg J H. Prognosis in children with febrile seizures. *Pediatrics* 1978; **61**:720-7.

2. Annegers J F, Hauser W A, Shirts S B. Factors prognostic of unprovoked seizures after febrile convulsions. *New England Journal of Medicine* 1987; **316**:493-8.

3. Schumann S H, Miller L J. Febrile convulsions in families: findings in an epidemiological survey. *Clinical Pediatrics (Philadelphia)* 1966;**5**:604-8.

4. Webb D W, Jones R R, Manzur A Y, Farrell K. Retrospective study of late febrile seizures. *Paediatric Neurology* 1999; **20**: 270-3.

5. Degen R, Degen H E, Hans K. A contribution to the genetics of febrile seizures: Waking and sleep EEG in siblings. *Epilepsia* 1991; **32**: 515-22.

6. Corey L A, Berg K, Pellock J M, Solaas M H et al. The occurrence of epilepsy and febrile seizures in Virginian and Norwegian twins. *Neurology* 1991; **41**:1433-6.

7. Frantzen E, Lennox-Buchtal M A, Nygaard A, Stene J. A genetic study of febrile convulsions. *Neurology* 1970 ; **20** : 909-17.

8. Tsuboi T. Genetic aspects of febrile convulsions. *Hum Genet* 1977 ; **38** : 169-73.

9. Wolf S: Laboratory evaluation of the child with a febrile convulsion. *Pediatrics* 1978 ; **62**:1074-6.

10. Ratcliffe J C, Wolf S M. Febrile convulsions caused by meningitis in young children. *Annals of Neurology* 1977 ; **1** : 285-6.

11. Green S M, Rothrock S G. Can seizures be the sole manifestation of meningitis in febrile children? *Pediatrics* 1993 ; **92** :527-34.

12. Lewis H, Parry J, Parry R. Role of viruses in febrile convulsions. *Archives of Diseases in Childhood* 1979; **54** : 869-76.

13. Rantala H, Tarkka R, Uhari M. A meta-analytic review of the preventive treatment of recurrences of febrile seizures, *Journal of Paediatrics*, 1997 ; **131(6)** : 922-5.

14. Knudsen F U. Febrile convulsions, treatment and prognosis. *Ugeskrift For Laeger* 2001;**163**: 1098-102.

15. Mei-Chin Huang, Ching-Chuan Liu, Chao-Ching Huang. Effects of an educational program on parents with febrile convulsive children. *Paediatric Neurolog* 1998 ; **18 (2)** : 150-5.

Table 1
Demographic data

Data	Number (%)
Age distribution (current episode)	
<12 months	129 (39)
13-60 months	194 (59)
> 61 months	07 (02)
Sex distribution	
male	198 (60)
female	132 (40)
Family history (first degree)	
Family history of febrile convulsions	82 (25)
Family history of epilepsy	04 (01)
Past history (total=135)	
1 febrile convulsions	65 (48)
2 febrile convulsions	35 (26)
3 febrile convulsions	16 (12)
4 febrile convulsions	12 (09)
>=5 febrile convulsions	07 (05)

Table 2
Seizure characteristics

Characteristics	Number (%)
Atypical seizures (n=83)	
multiple	73 (88)
focal	04 (05)
prolonged	14 (17)
Onset of seizure	
Within 24 hr of fever onset	66 (20)
After 24 hrs	264 (80)
Longest seizure	
first	194 (59)
second	92 (28)
third	30 (09)
fourth	07 (02)
>fifth	07 (02)

Teble 3
Treatment

Management	Number
Acute management before admission(n=60)	
Diazepam	
Per rectal	51
Intramuscular	04
Paraldehyde	
Intramuscular	04
Phenobarbitone	
Intramuscular	01
Prophylaxis (n=39)	
Diazepam (oral)	15
Sodium valproate	16
Carbamazepine	06
Phenorbarbitone	02

Table 4
Aetiology of fever

Aetiology	Number(%)
Upper respiratory infection	118 (36)
Viral fever	110 (33)
Lower resperatory Infection	59 (18)
Acute gastro-enteritis	26 (08)
Acute otitis media	06 (02)
Urinary tret infection	04 (01)
Other	07 (02)

Sociodemographic and health aspects of mothers in a paediatric ward

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Sri Lanka Journal of Child Health, 2005; **34** :114-9

(Key words: study of mothers, paediatric ward, physical health, psychological profile, reaction to stress)

Abstract

Objectives To study a group of mothers in a paediatric ward and describe sociodemographic profile, presence of existing disease, pulse and blood pressure (BP) profiles during hospital stay, psychological profile prior to admission and changes demonstrated during period of stay.

Method A descriptive, cross-sectional study was done in a ward at Lady Ridgeway Hospital for 6 weeks from May 2004. 500 mothers staying with their sick children were interviewed using a questionnaire dealing with various aspects. Mothers of children with chronic diseases needing regular admissions and those transferred to and from ward were excluded. Pulse rate and blood pressure (BP) of mothers were recorded on admission, within 24 hours and on discharge. Psychological profile was assessed as psychological score, describing behaviour prior to admission, and acute stress score, analysing changes demonstrated during present admission. This was done using a scoring system on a standard questionnaire with 23 items and a modified one with 5 items where higher scores identified mothers reacting more to stressful situations.

Results 476 (95.2%) questionnaires were analysed. Mean age of mothers was 32.1 years with a range of 17 to 52 years. 442 (92.9%) mothers had received secondary or higher education but 8 had never been to school. Monthly income was <Rs.10,000 in 296 (62.2%) families and 385 (80.9%) children were from middle and lower social classes. 117 (24.6%) mothers had preschool children at home when they were in hospital, majority of whom were looked after by grandparents. 65 (13.7%) mothers had long standing illnesses needing daily medication but only 15 (23.1%) took the drugs while in hospital. 54 (11.3%) mothers did not have anyone to confide in when they had a problem. Mean pulse rate and systolic and diastolic BP on discharge were significantly lower than the mean values on admission. Highest mean psychological score of 26.4 was found in the 21-30 year age group. Significantly higher acute stress scores were found in mothers below 30 years and in those who did not have anyone to confide in.

Conclusions This study demonstrates that mothers who play a vital role in management of their sick children are under immense stress and have their own health issues and family commitments that are mostly not appreciated or addressed. A social worker assigned to paediatric wards could provide invaluable help to mothers to cope with the stressful situation of hospitalisation of a sick child.

Introduction

Mothers are an integral part of paediatric practice. Information from them and their continued presence and support is vital for care of the sick child. Although we depend on them we know hardly anything about them. Hence this study was done to gather information about mothers in a paediatric ward.

Method

Study was done at the University Unit of Lady Ridgeway Hospital (LRH) for 6 weeks from May 2004 following approval from the Ethics Committee of University of Colombo. Informed verbal consent was obtained from mothers and 500 consecutive admissions were assessed using an interviewer administered questionnaire. Children with chronic illnesses and transfers to and from ward were excluded. Pulse rate and blood pressure (BP) of mothers were recorded on admission, within 24 hours and on discharge and presence of pallor was documented.

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The pre-tested questionnaire dealt with information regarding patient's sociodemographic data, physical health and psychological profile of the mothers and details of preschool children at home. Two aspects were considered for psychological profile, namely their behaviour prior to present admission and changes demonstrated during this admission.

We used 'Mothers' mental health questionnaire', prepared by Rutter in his Isle of White studies and modified for local use by A. Nikapotha[1], to study their psychosocial profile. A scoring system was used in the 23 item questionnaire. Each positive answer was given 2 marks and each negative answer 1 mark with a maximum of 46 for questionnaire. Disturbances in sleep, changes in appetite and bowel habits, feelings of sadness and fear, bodily aches and pains, were some of the questions asked. Reaction to acute stress was assessed using 5 items from this questionnaire, each positive answer getting 1 mark and each negative answer 0, with a maximum score of 5. The questions were on disturbances in sleep, appetite and bowel habits, palpitations and feelings of sadness. A higher score in the 23 item questionnaire (overall psychological score) identified mothers who were prone to react more to stressful situations and the 5 specific items in questionnaire (acute stress score) identified those who reacted more to the acute stressful situation.

Results

476 (95.2%) questionnaires were analysed. There were patients from all parts of the country but 244 (51.3%) patients were from Colombo and 162 (34%) came from Gampaha. Their ages ranged from 2 days to 12 years with a mean age of 2 years and 11 months and their duration of hospital stay was from 1 to 20 days with a mean of 2.88 days. Commonest cause of admission was viral fever, which affected 135 (28.4%) children. Acute gastro-enteritis/dysentery was the reason for admission in 110 (23.1%) patients and 20 children (4.2%) had dengue fever.

Sociodemographic description of the mothers is given in Table 1. Mean age of mothers was 32.1 years with a range of 17 to 52 years. Majority of mothers (92.9%) had secondary education or more but 8 had never been to school. 89 (18.7%) were gainfully employed. Monthly income was less than Rs 10, 000 in 296 (62.2%) families and 385 (80.9%) children were from middle and lower social classes [2].

Admission of a child to hospital may seem a trivial occurrence to hospital staff caring for the child. But it is an event that causes immense stress to family members especially mother. We found that 117 (24.6%) mothers in our study had left behind pre-school children at home. Seventy (59.8%) were looked after by grandparents and 25 (5.3%) by fathers while their mothers were in hospital. Of the mothers, 446 (93.7%) had no one who could relieve them even for a few hours and 9 (1.9%) had no visitors during hospital stay.

In a busy paediatric ward attention is focussed on the sick child and caregivers tend to forget that some mothers may have their own health problems. We found that 65 (13.7%) mothers had long standing illnesses such as hypertension, type II diabetes mellitus, bronchial asthma and epilepsy. They needed daily medication but only 15 (23.1%) mothers took the drugs while in hospital. Pallor was detected in 66 (13.9%) mothers. We also found that 264 (55.5%) mothers confided in their husbands when they had a problem and varying numbers in their parents, sisters and friends. But it was interesting to note that 54 (11.3%) did not confide in anyone.

Mean values for pulse rate and BP (systolic and diastolic) on admission, within 24 hours and on discharge were available in 206 mothers and are summarised in Table 2a. Mean values on discharge of all 3 parameters were significantly lower than mean values recorded on admission (Table 2b).

Analysis of psychological profile is shown in Table 3. Highest mean psychological score of 26.4 was found in the 21–30 year age group. Acute stress scores showed a progressive decline with advancing age with the highest score of 3 seen in mothers <20 years. For comparison of psychological profiles mothers were further divided into two groups – young mothers less than 30yrs of age and above. Psychological profiles were also compared in mothers who had a person to confide in and those who did not. This data is summarised in Tables 4a and 4b. Mothers below 30yrs had significantly higher mean acute stress scores than the others. Those who did not confide in anyone had significantly higher psychological scores as well as acute stress scores.

Discussion

The bond that exists between a mother and her child becomes more pronounced when child falls sick. Studies have been done on various aspects of illnesses of a child but very few have targeted the mother. One such study has described the coping strategies among parents in an Intensive Care Unit (ICU) setting[3] and another the impact of environmental stressors on parents of children in ICUs[4]. A previous study done in the same Unit at LRH demonstrated that inspite of the meagre

facilities available for mothers[5,6] they were satisfied with the overall care of the sick child[6].

The timing of this study was at the start of the dengue epidemic. This must undoubtedly have contributed to enhance the stress brought on by the child's illness. This may also have been the reason for some mothers to forget to take their own medications.

Parameters of physical reaction to stress that we studied in mothers were pulse rate and BP. These showed a significant reduction on discharge from hospital demonstrating how stressful the hospital admission was to them and confirming the need for some form of support while in hospital. We also found that young mothers found it harder to cope and reacted more to acute stress. A person who confides in others is less likely to react adversely to stressful situations[7,8]. We demonstrated that mothers who did not have good confiding relationships reacted more to the acute stressful situation of child being ill.

When we concentrate on the sick child, we often forget that mothers are under immense stress and that they have their own health issues that are mostly not appreciated or addressed. We believe that a trained social worker assigned to paediatric wards would be able to spend more time with mothers, be their confidante and help to make the stressful situation more manageable. In the absence of social workers this responsibility would lie with the medical and nursing staff. However, time available for such a task is limited. At least showing empathy towards mothers trying to answer their questions during some time of the day could be tried out by all of us. Having a less stressful mother would indirectly influence the well being of the sick child to speed up the recovery process.

References

1 Rutter M, Tizard J, Yule M, Graham PJ, Whitmore K. Research Report: Isle of White studies 1964-1974. *Psychological Medicine* 1976; 6:313-32.

2 Abramson JH. Survey Methods in Community Medicine. 4th ed. London: Churchill Livingstone, 1974; 101-7.

3 Carnevale FA. A description of stressors and coping strategies among parents of critically ill children – a preliminary study. *Intensive Care Nursing* 1990; 6(1):4-11.

4 Carter MC, Miles MS. The Parental Stressor Scale: Pediatric Intensive Care Unit. *Maternal-Child Nursing Journal* 1989;**18**(3):187-98.

5 de Silva S, Dharmage SC. Plight of mothers in a paediatric ward. Proceedings of the 30th Annual Scientific Congress of the Sri Lanka Paediatric Association, 1995.

6 de Silva KSH, Dharmage SC. Assessment of client satisfaction in a paediatric ward. *Ceylon Medical Journal* 1996; **41**(4):148-50.

7 Mirza I, Jenkins R. Risk factors, prevalence and treatment of anxiety and depressive disorders in Pakistan: systematic review. *British Medical Journal* 2004; **328**:794.

8 Stress in the workplace. Publication by BUPA's Health Information Team, 2004.

Table 1
Sociodemographic description of the mothers

Sociodemographic feature		Number (%)
Age (years)		
	≤ 20	7 (01.5)
	21 - 30	198 (41.6)
	31 – 40	216 (45.4)
	≥ 41	55 (11.6)
Level of education (Grade)		
	No education	8 (1.7)
	1 – 5	26 (5.5)
	6 – 10	279 (58.6)
	11 – 12	147 (30.9)
	Higher education	16 (3.4)
Social class*	I	31(06.5)
	II	60 (12.6)
	III	128 (26.9)
	IV	189 (39.7)
	V	68 (14.3)
Monthly family income (Rs)		
	< 5000	75 (15.8)
	> 5000 – 10 000	221 (46.4)
	> 10 000 – 20 000	121 (25.4)
	> 20 000	59 (12.4)

* According to fathers' occupation[2]

Table 2a
Pulse rates and BP profiles of the mothers

Timing	Pulse rate/minute	SBP* (mmHg)	DBP** (mmHg)
	mean (SD)	mean (SD)	mean (SD)
On admission n = 206	82.26 (8.78)	113.64 (8.89)	71.14 (8.15)
Within 24 hours n = 206	80.34 (10.06)	107.91 (10.91)	67.38 (9.44)
On discharge n = 206	78.05 (8.12)	108.52 (9.35)	68.25 (8.24)

* Systolic blood pressure
** Diastolic blood pressure

Table 2b
Comparison of the mean pulse rate and BP values of the mothers on admission and on discharge

	On admission mean (SD)	On discharge mean (SD)	Statistical significance
Pulse rate/minute n = 206	82.26 (8.78)	78.05 (8.12)	$p < 0.0001$
SBP (mmHg) n = 206	113.64 (8.89)	108.52 (9.35)	$p < 0.0001$
DBP (mmHg) n = 206	71.14 (8.15)	68.25 (8.24)	$p < 0.0005$

Table 3
Psychological profile of mothers in the different age groups

Age (years)		Psychological score* mean (SD)	Acute stress score** mean (SD)
< 20	n=7	25.86 (3.24)	3.00 (0.82)
21-30	n=198	26.41 (3.36)	2.48 (1.14)
31-40	n=216	26.08 (2.99)	2.02 (1.31)
> 41	n=55	26.31 (3.07)	1.71 (1.33)

* Highest score possible = 46
** Highest score possible = 5

Table 4a
Psychological profile in the two groups of mothers

	≤ 30yrs n=205	> 31yrs n=271	Statistical significance
Psychological score mean (SD)	26.40 (3.34)	26.09 (3.00)	$p > 0.05$
Acute stress score mean (SD)	2.50 (1.14)	1.96 (1.32)	$p < 0.0001$

Table 4b
Psychological profile in relation to confiding in others

	Confide + n=422	Confide - n=54	Statistical significance
Psychological score mean (SD)	25.82 (2.79)	29.39 (3.96)	$p < 0.0001$
Acute stress score mean (SD)	2.14 (1.26)	2.57 (1.34)	$p < 0.0001$

Headache in children

Duminda Pathirana[1]

Sri Lanka Journal of Child Health, 2005; **34**:120-3

Introduction

Headache is a common childhood complaint. Population-based studies have shown that 66% of all children between 5 and 15 years of age had at least one episode of headache during a 1-year period and in 22% the headache was severe enough to interfere with daily activities[1].

Categorization of headache

There are three main categories of headache in childhood viz. vascular, psychogenic and organic[2,3]. By careful history taking and examination one could differentiate these categories to a certain extent (Table 1). Headaches can also be categorized as acute, recurrent or chronic in nature.

Many children present with acute headaches. These do not usually require further investigation. The causes of acute headache in 150 unselected children attending an accident and emergency department were: upper respiratory tract infections (57%), migraine without aura (18%), viral meningitis (9%), brain tumour (2.6%), ventriculo-peritoneal shunt dysfunction (2%), intracranial haemorrhage (1.3%), postictal headache (1.3%), post-concussion (1.3%) and undetermined causes (7%)[4].

In chronic headache children present with at least 3 months' history of constant headache or a headache with a fluctuating intensity, but with no periods of complete recovery. These are rare in children but may be the presenting clinical manifestation of intracranial tumours[5].

In recurrent headache a history of at least 6 months of recurrent episodes is common. The attacks of headache are clearly separated by periods of complete normality. In a population-based study, migraine (with or without aura) was the most common cause of recurrent headache in school children accounting for 77.2% of cases[1]. Other causes were: tension headache in 11.7%, non-specific headache in 9.7% and headache associated with specific illnesses such as asthma, hay fever, allergy and constipation in 1.5%[1].

Evaluation of headache

The following features in the history and examination may help the paediatrician to decide on further investigations[6].

1. Headache - recent onset, increasing severity and frequency, morning/nocturnal occurrence, awakening the patient from deep sleep, constant daily, lack of relieving and triggering factors, lack of family history of migraine, occipital or strictly unilateral location, association with projectile vomiting, made worse by straining, sneezing, coughing, exacerbated or improved by change in position.
2. Vomiting – increased severity and frequency without nausea.
3. Psychological or behavioural signs - drowsiness, irritability, change in eating habits, tantrums, anxiety, mood swings, poor concentration, recent deterioration in school performance.
4. Other suspected features – Neurological abnormalities/decreased visual acuity/seizures associated with headache / focal neurological signs developing during headache / visual problems occurring at peak of headache rather than aura./ deceleration of linear growth /age less than 6 years.

Investigations

Investigations are only required in the minority of children with chronic or recurrent headache. *Lumbar puncture* may be needed at times to rule out entities such as pseudotumor cerebri or if there is some concern about an infectious process[7]. *Electroencephalography* should be reserved for patients with alteration of mental status, loss of consciousness or entities suggestive of the epilepsy syndrome[7,8]. *Computed tomography* (CT) and *magnetic resonance imaging* (MRI) are safe, rapid and accurate methods for evaluating intracranial content if and when an intracranial disorder is suspected[7,9]. For acute evaluation of headaches, CT is performed easily

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and rules out most intracranial pathology. Abnormalities of the posterior fossa can however be missed on CT scan[7]. MRI is recommended for patients whose history is suggestive of a vascular event, a space-occupying lesion or posterior fossa abnormality[7,10].

Migraine

Migraine is the commonest basis of recurring headache in childhood. It is a neuromuscular syndrome that leads to a generalized vasomotor instability and vulnerability to multiple extraneous factors[11]. It is estimated that around 1 in 10 school children suffer from migraine[1]. The aetiology of migraine is not known but it has a familial tendency. Both migraine without aura and migraine with aura are inherited disorders[12]. The International Headache Society (IHS) classified migraine into 2 major forms: migraine without aura and migraine with aura[2]. Less common forms such as abdominal migraine and cyclical vomiting syndrome were also recognized and defined[2]. Criteria have been established by the IHS for the diagnosis of the various forms of migraine[2]. 75-85% of children suffer from migraine without aura[1]. Both major forms of migraine may be present in the same patient[1]. Boys and girls under the age of 12 years are almost equally affected with migraine but in children older than 12 years migraine is commoner in girls than in boys[1].

Migraine headache is typically recurrent in nature with complete recovery between attacks. Stress and anxiety are the most commonly identified trigger factors[11]. Only 10-15% of patients can identify a certain type of food, such as cheese, chocolates and caffeine-containing drinks, as a trigger factor[11]. Aura symptoms, if present, precede the onset of headache and are commonly visual in nature (blurred vision, tunnel vision, blind spots [scotomata] or zigzag coloured lines in front of the eyes)[11]. Rarely, the aura symptoms are sensory (tingling or numbness), motor (hemiplegia or speech disturbances), autonomic or non-specific[11]. During the attack of migraine the child is pale, quiet and wants to be left alone. He refuses food and drink, feels nauseated and may vomit. Light, noise, smell and exercise may aggravate pain. Dizziness, abdominal pain, visual disturbances and sensory or motor deficits may be associated[11]. Some patients describe unusual visual hallucinations or distortion of images or both called the *Alice in wonderland* syndrome[13].

Complicated migraine

- *Basilar migraine* – clinical features of migraine are dominated by transient symptoms of cerebellar and brainstem dysfunction such as vertigo, ataxia, visual field defects, motor deficits, dysphasia and confusion. These features are attributed to vascular constriction of the basilar artery[11].

- *Confusional migraine*- attacks of migraine triggered by minor head injury. Clinical features include an aura, followed by headache, drowsiness, irritability, agitation, disturbed speech, aggressive behaviour and amnesia[11].

- *Ophthalmoplegic migraine*- migraine attacks complicated by paralysis/paresis of the extraocular muscles, ptosis and pupillary dilatation but with no associated confusion or loss of consciousness[11].

- *Hemiplegic migraine* – attacks of migraine complicated by unilateral weakness, impaired speech or unilateral sensory loss[11].

Differentiation of epilepsy and migraine[8]

This is shown in Table 2.

Treatment of migraine

1. *Non medical treatment* – includes reassurance of child and parents about the benign nature of the disorder and education concerning its natural course of remissions and relapses. Children should be encouraged to identify their own trigger and relieving factors and explore their own strategies of treatment.

2. *Treatment of acute attack*- Children should be allowed to rest and lie down in a quiet environment. Early administration of analgesics is commonly associated with good results. Paracetamol and ibuprofen are the first line of treatment[11]. Domperidone, metoclopramide or prochloperazine may alleviate nausea and vomiting during migraine attacks. Concerns about possible dystonic reactions may preclude the use of metoclopramide and prochloperazine[11]. Ergotamine preparations could be used in children but can cause adverse effects such as prolonged vasoconstriction. Propranalol is not. used for acute attacks in children. Sumatriptan, a selective 5 HT agonist, is effective in adults but its efficacy in children is not yet proven[7].

3. *Interval treatment* - is only indicated if acute treatment is unsuccessful and migraine attacks are frequent (more than 2 attacks per month). Propanolol

was shown to reduce the frequency of migraine attacks in a double-blind placebo-controlled trial[14]. However, propanolol should not be used in children with a history of wheezing.

References

1. Abu-Arafeh I, Russel G. Prevalence and causes of headache in school children. *BMJ* 1994; **309**: 765-9.

2. Headache Classification Committee of International Headache Society. Classification and diagnostic criteria for headache disorders, cranial neuralgia and facial pain. *Cephalgia* 1988; **89(suppl 7)**: 1-96.

3. Abu-Arafeh I, editor. Childhood Headache. London; Mac Keith Press, 2002.

4. Lewis DW, Quereshi FA. Acute headache in the paediatric emergency department. *Headache* 2000; **40**: 200-3.

5. Vasquez-Barquero A, Ibanez FJ, Herrera S. Isolated headache as the presenting clinical manifestation of intracranial tu mours: a prospective study. *Cephalgia* 1994; **14**: 270-2.

6. Miltenbutg G, Louw DF, Sutherland GR. Epidemiology of childhood brain tumours. Can J Neurol Sci 1996; **23**: 118-22.

7. Linder SL, Winner P. Paediatric headache. Medical Clinics of North America 2001;**85**: 1037-53.

8. Andermann F, Lugaresi E. editors, Migraine and epilepsy. Boston: Butterworths, 1987; 281-91.

9. Maytal J, Bienkowski RS, Patel M. The value of brain imaging in children with headache. *Pediatrics* 1995; **98**: 413-6.

10. Wober-Bingol C, Wober C, Prayer D. Magnetic resonance imaging for recurrent headache in childhood and adolescents. *Headache* 1996; **36**: 83-90.

11. Bille B. Migraine in children and its prognosis. *Cephalalgia* 1981; **1**: 71-5.

12. Russell MB, Iselius L, Olesen J. Migraine without aura and migraine with aura are inherited disorders. *Cephalalgia* 1996; **16**: 305-9.

13. Golden GS. The Alice-in-wonderland syndrome in juvenile migraine. *Pediatrics* 1979; **65**: 517-9.

14. Ludvigsson J. Propanolol used in prophylaxis of migraine in children. *Acta Neurol Scand* 1974;**50**: 109-15.

Table 1

Categorization of headache in childhood

	Vascular	Psychogenic	Organic
Occurrence	Periodic	Continuous	Periodic or continuous Nocturnal/early morning
Quality	Throbbing	Pressure, aching, tightness	Pressure, throbbing, aching, tightness. Sometimes localized
Associated symptoms	Gastrointestinal. –anorexia, nausea, vomiting, abdominal pain. Visual-photophobia, blurred vision, scotoma Others-vertigo, syncope Convulsions-rare Fever- rare	Anxiety Depression	May include any symptoms listed under vascular or psychogenic headings. Evidence of infection as in sinusitis.
Family history	Yes, 90% of children	Variable	Variable
Associated signs	Pallor, visual field defects, confusion, amnesia, hemisyndromes, aphasia, ophthalmic sympathoparesis, 3rd nerve palsy (brain stem, basilar)	None	Transient or persistent neurological signs of elevated pressure. Neurological abnormalities found in 95% of tumors in the first 4-6 months of headache.

Table 2

Differentiation of epilepsy and migraine

	Migraine	Epilepsy
Paroxysmal expression	Primary vascular	Neuronal
Principal manifestation	Headache, nausea, vomiting, pallor	Seizures
State of consciousness	Typically preserved	Typically altered
Duration	Portion of hour or more	Seconds or minutes
Aura	Typically visual	Wide range of neurological phenomena
Duration of aura	Minutes	Seconds
Post-ictal sleep	Occasional	Common
EEG abnormalities	Low incidence	High incidence discharge
Family History	90%	Low
Onset	Characteristically gradual	Sudden
Influenced by emotion	High frequency	Low
Recognizable triggers	Moderate frequency	Low

Snippets from the world wide web

Sri Lanka Journal of Child Health, 2005; **34**:124-5

Reduced Carbohydrate Intake May Lower Cardiovascular Risk

In a healthful diet, partial substitution of carbohydrate with either protein or monounsaturated fat may further lower blood pressure, improve lipid levels, and reduce cardiac risk.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4rD0Em

Fish Oil Supplements Improve Lipid Risk Factors in Obese Children

In a small, randomized study, triglyceride levels decreased and HDL cholesterol levels increased; total cholesterol/HDL and total cholesterol/LDL ratios also improved.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4rR0E1

High-Resolution CT Scans Reveal Airway Remodelling in Severe Asthma

High-resolution CT scans can quantify airway remodelling in children with severe asthma, according to a report in the October Journal of Allergy and Clinical Immunology.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4rz0Eg

Hypertension Is Not a "Silent" Disease in Children

This study showed that elevated blood pressure was associated with frequent headaches and sleep trouble in youngsters.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4rK0Et

Chorioamnionitis Tied to Intraventricular Hemorrhage in Premature Infants

Very low birth weight infants with chorioamnionitis appear to be at increased risk of intraventricular hemorrhage and retinopathy of prematurity, but not developmental impairment, researchers report in the November issue of the Archives of Pediatrics and Adolescent Medicine.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4rT0E3

MRI Corresponds With Neurodevelopmental Outcome in Preterms

The results of a study published in the November issue of the Archives of Diseases in Childhood-Fetal and Neonatal Edition, suggest that a normal neonatal cranial ultrasound excludes severe lesions on magnetic resonance imaging in most infants born preterm. MRI is more strongly correlated with mean IQ and median total impairment score than is ultrasound at school age.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4u50Ea

Transdermal Fentanyl Effective for Pediatric Pain Patients

A study showed that a transdermal patch delivering the opioid fentanyl is a safe and effective way to control moderate to severe chronic pain in children aged 2 and older with previous exposure to opioid therapy.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4uZ0ED

IgE Antibodies to Inhaled Allergens Predict Wheeze in Young Children

Serum levels of IgE antibodies to common allergens can predict the likelihood of persistent wheeze and reduced lung function among preschool-aged children, a new study shows.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4r20EU

Risk of Birth Defects With Intracytoplasmic Sperm Injection Warrants Study

Children conceived by intracytoplasmic sperm injection do not have delayed or abnormal neurodevelopment, a study shows, but they do appear to be at higher risk for congenital malformations.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4v20EY

Tamiflu Link Probed in Deaths of Two Japanese Youths

Japan's Chugai Pharmaceutical Co. said it has told the government that 2 teenage boys exhibited abnormal behavior that led to their deaths after taking the anti-flu drug Tamiflu, made by Chugai's Swiss parent Roche Holding AG.

http://mp.medscape.com/cgi-bin1/DM/y/evSP0EIZ1O0DzQ0G4tj0ES

A New Method May Predict Adult Height of Adolescents

The technique was a valid, nonintrusive, inexpensive, and simple method of predicting adult height in adolescent children, at least in white people who are free of growth-limiting diseases.

http://mp.medscape.com/cgi-bin1/DM/y/eub20EIZ1O0DzQ0G1m80EC

Pneumonia in Older Adults May Have Decreased Because of Conjugate Pneumococcal Vaccine Use in Children

A study suggested that use of conjugate vaccine in children has substantially benefited older adults, more so in healthier persons than in those with certain comorbid conditions.

http://mp.medscape.com/cgi-bin1/DM/y/eub20EIZ1O0DzQ0G1nz0EJ

Cochrane Review Refutes Link Between MMR Vaccination and Serious Illness

An extensive Cochrane Collaboration review of studies examining outcomes after immunization with the measles-mumps-rubella vaccine has turned up no credible evidence that the vaccine is associated with autism or Crohn's disease or other serious illnesses.

http://mp.medscape.com/cgi-bin1/DM/y/eub20EIZ1O0DzQ0G1mj0E1

C-Section Not Tied to Childhood Asthma

The mode of delivery at birth, whether by cesarean section or vaginally, appears to have no bearing on the subsequent development of asthma, according to researchers.

http://mp.medscape.com/cgi-bin1/DM/y/eub20EIZ1O0DzQ0G1kl0E1

Boosters May Not Be Needed 10 Years After Childhood Vaccination for HBV

A study suggested that strong immunologic memory persists more than 10 years after immunization of infants and adolescents with a primary course of vaccination.

http://mp.medscape.com/cgi-bin1/DM/y/euMz0EIZ1O0DzQ0Gzez0EE

Inhaled Insulin May Improve Glycemic Control Without Altering Pulmonary Function

An open-label, randomized trial suggested that inhaled insulin improved glycemic control when added to oral agents in type 2 diabetes without altering pulmonary function.

http://mp.medscape.com/cgi-bin1/DM/y/euMz0EIZ1O0DzQ0Gzdy0EC

B J C Perera
Joint Editor

CaseReports

A girl with Rett Syndrome

S P Abeywardena[1], AC D De Alwis[1], G W J Keerthiwansa[2]

Sri Lanka Journal of Child Health, 2005; **34**:126-7

Introduction

Although Rett syndrome (RS) was first described by Andreas Rett in 1966, this disorder became internationally recognized only after the report of Hagberg et al. in 1983[1]. It is a neurodevelopmental disorder which affects almost exclusively girls and is associated with deceleration of head growth, typical stereotyped hand movements, severe mental deficiency, cortical and extrapyramidal dysfunction including gait disturbance and truncal ataxia as well as loss of purposeful use of the hands[2,3].

Case report

A 4 1/2 year old girl presented to General Hospital Ratnapura with a history of reduced speech, abnormal hand movements and reduced awareness of surroundings of one year duration (Figure 1).

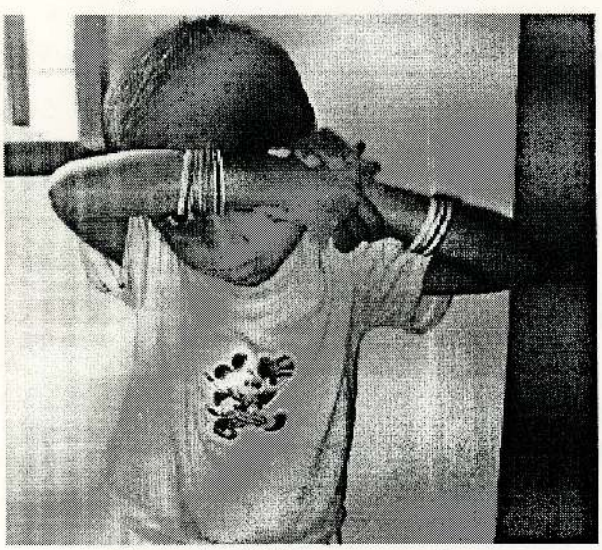

Figure 1 *Child on presentation*

She had normal speech development until the age of 3 1/2 years; she could speak four word sentences, relate a short story and sing songs. Her speech gradually deteriorated over one year and now she can only speak three single words. At 3 1/2 years she could draw a circle although now she cannot scribble due to wringing movements of both hands, more on the left. Child's grasp is abnormal and she is unable to hold a plate or a pen properly. Her social development was age appropriate till 3 1/2 years. Now she avoids eye contact and interaction with peers and plays on her own. Her gross motor development is normal for the age. Mother recollected 4-5 episodes of sighing breathing during the last 4 months. Child is grinding her teeth at night. The sleep pattern is normal. Parents sought indigenous treatment for one year. As there was no response to indigenous treatment she was brought for western medical advice.

She never had seizures or fainting attacks. There was no history of head injury or measles in infancy. Except for three hospital admissions for minor ailments like viral fever and diarrhoea, child was well previously. Child is the younger of two girls, product of a non-consanguineous marriage. Birth weight was 3 kgs. There were no perinatal complications and no neonatal jaundice. Elder sister is 6 1/2 years old and is in good health. There are no relatives with epilepsy, psychiatric disorders, mental subnormality or liver disease such as Wilson disease.

On examination, child's weight was 9 kgs, well below the 3rd centile for her age; height was 95cms, at 3rd centile; occipito frontal circumference was 46 cms well below 3rd centile. Child was active, conscious, shy, covering half of the face with one hand (Figure 1); there were no dysmorphic features; child was not pale or icteric; hand wringing movements were present, more on left side; she holds left with right hand. There were numerous carious teeth. Dry skin and icthyosis were present. There was no scoliosis. The cardiovascular and respiratory systems and abdominal examination revealed no abnormality. Central nervous system i.e. higher functions, cranial nerves, including fundi, were normal. Tone and reflexes were normal. Power-grasp was poor but difficult to assess. Gait was normal.

Blood counts, liver function tests and renal function tests were normal. ECG was normal. Epileptiform changes were present in EEG.

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As this girl has a regression disorder with apparently normal development until 3 1/2 years, normal head circumference at birth followed by slowing, repetitive hand movements, reduced speech and growth retardation, a diagnosis of Rett syndrome was made.

Discussion

Prevalence of Rett syndrome is estimated between 1:10,000 and 1:15,000 girls[4]. Until 1999, there was no known biochemical, morphological or genetic marker for RS and diagnosis was established on clinical criteria[5]. Recently, Amir et al. have identified mutations on the gene MECP2, which is located on Xq28 and encodes methyl-CpG-binding protein 2 (MeCP2) in patients with RS[6]. Mutations have been found in about 80% of RS patients[7].

References

1. Hagberg B, Aicardi J, Dias K, Ramos O. A progressive syndrome of autism, dementia, ataxia and loss of purposeful hand use in girls: Rett syndrome: report of 35 cases. *Ann Neurol* 1983; 14: 471-9.

2. Armstrong DD. Review of Rett syndrome. *J Neuropathol Exp Neurol* 1997; **56**: 843-9.

3. Hagberg B. Rett syndrome: clinical peculiarities and biological mysteries. *Acta Paediatr* 1995; **84**: 971-6.

4. Kozinetz CA, Skender ML, Macnaughton N. Epidemiology of Rett syndrome: a population-based registry. *Pediatrics* 1993; **91**: 445-50.

5. Diagnostic criteria for Rett syndrome. The Rett Syndrome Diagnostic Criteria Work Group. *Ann Neurol* 1988; **23**: 425-8.

6. Amir RE, Van Den Veyver IB, Wan M, Tran CQ et al. Rett syndrome is caused by mutations in X-linked MECP2, encoding methyl-CpG-binding protein 2. *Nat Genet* 1999; **23**: 185-8.

7. Webb T, Latif F. Rett syndrome and the MECP2 gene. *J Med Genet* 2001; **38**: 217-23.

Child with cerebrospinal fluid rhinorhoea complicated by recurrent meningitis

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Introduction

Recurrent meningitis in children may be due to immune deficiency, presence of indwelling devices in the ventricular system or breakage of the mucocutaneous barrier between skin and cerebrospinal fluid (CSF). We report a child who presented with recurrent meningitis and CSF rhinorrhoea due to presence of a defect in the cribriform plate.

Case report

A seven year old boy presented with the seventh episode of pyogenic meningitis in July 2003. He was the second child of non consanguineous parents and was born at term by elective caesarean section with a weight of 2.5 kg. He had multiple dysmorphic features mainly in the craniofacial region (Figure1) and bilateral congenital sensorineural deafness. The dysmorphic features consisted of bilateral aniridia, microphthalmia and low set ears. There was marked delay in speech but his motor and social functions were age appropriate. Despite aniridia he had useful vision and attended a special school. He had no history of head trauma.

Figure 1. *Facial dysmorphic features.*

He had initially presented at ten weeks of age with features suggestive of meningitis which were confirmed by CSF analysis. Subsequently almost every year he presented with episodes of pyogenic meningitis which were treated successfully with appropriate antibiotics. Excessive watery discharge from the left nostril had been noted since the neonatal period by his parents. This was confirmed as left sided CSF rfinorrhoea only during the third episode of meningitis at the age of four years when he was extensively investigated for recurrent meningitis. Screening for immune deficiency, including HIV screening, immunoglobulin and complement levels, was normal.

During the first recurrence he was confirmed to have *Streptococcal pneumoniae* meningitis on CSF culture. Since he was detected to have a CSF leak with microbiologically confirmed streptococcal growth during the fourth episode of meningitis he was started on long term three weekly IM benzathine penicillin prophylaxis with good compliance. He was immunized with 23 valent unconjugated pneumococcal vaccine which was repeated after three years. At the same time he was immunised against meningitis due to Haemophilus influenzae type B and Neisseria meningitidis.

Despite these prophylactic measures he has had a total of nine recurrences. *Streptococcal pneumoniae* was confirmed on five occasions, including last four episodes, as the causative organism. Since the sixth recurrence these organisms have developed resistance to penicillin resulting in treatment with intravenous vancomicin.

During the fourth recurrence he underwent imaging studies to detect an identifiable anatomical defect with a computed tomography (CT) scan which proved negative. An intrathecal contrast enhanced helical CT scan was done during the sixth recurrence; it consisted of 0.5mm coronal sections through the anterior

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fossa and the paranasal region. Pre and post contrast films were obtained after confirming CSF rhinorrhoea during the procedure. This CT scan was performed following a discussion with a multi-disciplinary team consisting of a neurosurgeon, an ENT surgeon and a team of radiologists. Cranial defects in the cribriform plate were confirmed on CT scan by this team of consultants at a joint meeting (Figure 2).

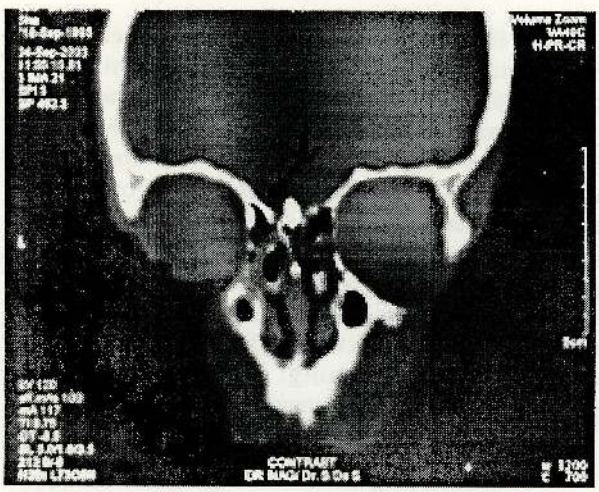

Figure 2. *CT scan showing defect in cribriform plate.*

The patient underwent an endo-nasal repair of the identified defects in two stages. However he had 2 further episodes of meningitis since then. He continues to have CSF rhinorrhoea with a remarkable reduction in volume. He was further investigated with a magnetic resonance imaging (MRI) scan to detect residual defects without any success.

Discussion

Congenital CSF leaks across a mucocutaneous barrier, such as cranial or midline facial defects across the cribriform plate or inner ear fistulae, are associated with increased risk of recurrent pneumococcal meningitis[1]. These leaks are under diagnosed, difficult to locate and carry a potentially lethal cumulative long term risk due to recurrent meningitis. Sensorineural hearing loss and craniofacial anomalies are associated with congenital inner ear anatomical abnormalities. Mondini dysplasia[2], consisting of peri lymph fistulae connecting the defective inner ear through temporal bone to paranasal sinuses resulting in CSF rhinorrhoea, is one such example. There were no defects detected in the inner ear or temporal bone in our patient.

Nasal endoscopy with intra-thecal fluorescein dye enables location of most of these CSF fistulae and correction with minimal morbidity[3]. An endoscopic technique was used to seal the defect in our patient though intrathecal injection of fluorescein dye was not feasible due to lack of resources. Therefore it is important to maintain possible prophylactic measures to prevent recurrences in this child. Unconjugated pneumococcal vaccine confers capsular specific immunity against 23 sero types in immunologically competent individuals and is the current international recommendation to prevent invasive pneumococcal infection in children more than 5 years of age[4]. Life-long benzathine penicillin prophylaxis at three weekly intervals to achieve higher drug levels is recommended to prevent invasive infection.

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References

1. Wen HY, Chou ML, Lin KL, Kao PF, Chen JF. Recurrence of pneumococcal meningitis due to primary spontaneous cerebrospinal fluid fistulas. *Chan Gung Medical Journal* 2001; **11**: 724-8.

2. Tullu MS, Khanna SS, Kamat JR, Kirtane MV. Mondini Dysplasia and pyogenic meningitis. *Indian Journal of Paediatrics* 2004; **71 (7)** : 655-7.

3. Swift AC, Foy P. Advances in the management of CSF rhinorrhoea. *Hospital Medicine* 2002; **63**: 28-32.

4. Morbidity and mortality weekly report (MMWR) recommendations and reports. Preventing pneumococcal disease among infants and young children. Recommendations of the advisory committee on immunisation practices 2000; 49 (RR-9) : 1-35.

Picture story

A female with dyskeratosis congenita

T S D Amarasena[1], G K Hapuarachchi[2], M H A D dc Silva[3]

Sri Lanka.Journal of Child Health. 2005: 34:130-1

Introduction

Dyskeratosis congenita is an inherited bone marrow failure syndrome, with multisystem involvement. Incidence is approximately I case per million population. 225 individuals have been reported in the literature[1].

Case report

An 11 year old girl, born to unrelated parents, was admitted with fever and gum bleeding. She was apparently well up to this presentation. Both parents and siblings (all females) were healthy. She has had learning difficulty in the school.

She was severely pale with no lymphadenopathy or organomegaly. There was reticulated hyperpigmentation prominent over the face. neck, chest and limbs, including palms and soles, suggestive of poikilodcrma (Figure I).

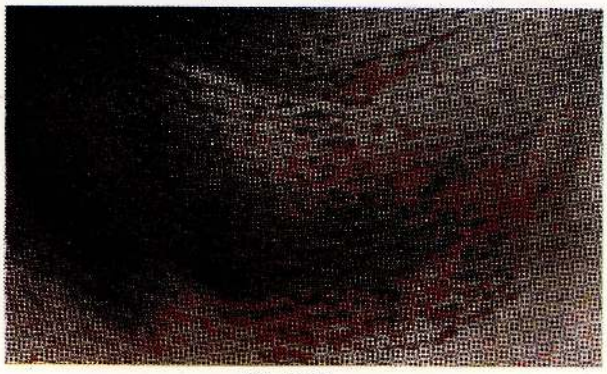

Figure I

This was later confirmed by a dermatologist. She also had thin sparse hair and an atrophic tongue. Nails were dystrophic with longitudinal ridging (Figure 2).

The haemoglobin was 5.5 g/dl and the platelet count was 28 x 10^9/L. Blood picture showed normocytic normochromic anaemia with low platelets. There was neutropenia but no atypical or immature cells. Trephine marrow biopy revealed moderately hypoplastic erythropoietic series and absent megakaryocytes in the bone marrow. A diagnosis of hypoplastic anaemia was made. Ham's test was negative and antmuclear antibodies were absent. A diagnosis of dyskeratosis congenita was made.

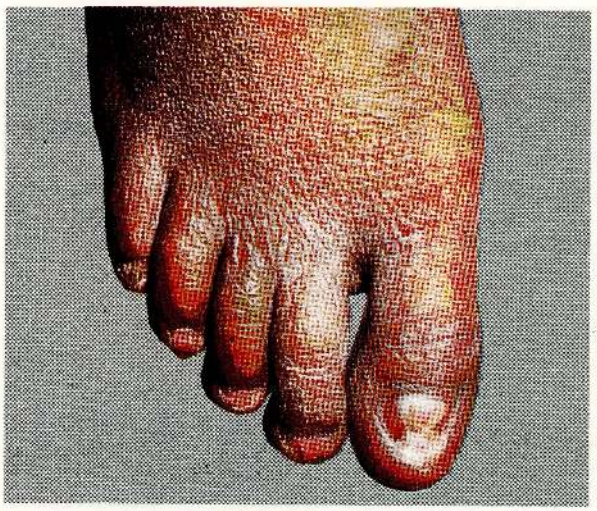

Figure 2

Initially she was given blood and platelet transfusions. She was treated with prednisolone 2mg / kg / 24 hrs. Six weeks later she developed avascular necrosis of the left femoral head and prednisolone had to be tailed off. She needs regular blood transfusions every 2 - 3 months now.

Discussion

Dvskeratosis congcnita is a rare disease which has X linked recessive, autosomal recessive or autosomal dominant forms of inheritance. X linked recessive is the most common form of inheritance. Male: Female ratio is 10: 1[2] . The female carriers may have subtle clinical features. Mutant gene is DKC 1. located at Xq28. It encodes a protein. called dyskerin. which has widespread tissue distribution[2]. This is involved in the regulation of the proliferative capacity of the cell

Diagnosis is based on cutaneous and mucosal findings with bone marrow abnormalities The cutaneous findings include reticulated or mottled pigmentation of the skin, hyperhidrosis of palms and soles, hyperkeratosis of palms and soles and adermatoglyphia. Mucosal findings include leukoplakia mainly on the buccal mucosa. but other mucosal sites can get involved. These mucosal findings may not appear until the second

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or the third decade and on occasions, not until after the onset of bone marrow failure. Incidence of malignant neoplasms, particularly squamous cell carcinomas, is increased in these patients. There is an increased incidence of dental caries and early tooth loss. Progressive nail dystrophy with ridging and longitudinal fissures occur with progressive atrophy. Mild to moderate mental retardation is found in *21%* of patients.

Median age of diagnosis of initial hacmalological disease is about 16 years. First haematological manifestation is pancytopenia. Bone marrow shows hypoplastic anaemia which would develop in approximately 50% of the patients. Hb F is increased[1]. Chromosome breakages have been observed in 10% of patients[1]. Predicted median age of survival is 33 years[1] . Majority of the patients die of bone marrow failure. The\ are also susceptible to squamous cell carcinomas and gut malignancies.

Treatment

50% of the patients show a transient response to androgens[1] but relapses are common. Oxymetholone was not available for this patient. The only curative therapy to date has been bone marrow transplantation. However, the preparative regimens generally used during the bone marrow transplantation can adversely impact the susceptibility to malignancy. Steroids. granulocyte macrophage colony stimulating factors and erythropoietin may be helpful transiently. Treatment with multiple cytokines (IL-3, IL-6) may offer additional benefits[1] anti-thymocyte globulin (ATG) is not recommended for these patients. Gene therapy may become a feasible consideration.

Once an index case has been identified. genetic counselling is important. The parents must be oriented to the pattern of inheritance and the prospect of prenatal diagnosis. This will allow early harvest and storage of their bone marrow for use after anticipated marrow failure.

References

1. D'Andrea AD. The Constitutional Pancytopaenias. In Behrmann RE, Kliegman RM, Arvin AW, editors, Nelson Textbook of Paediatrics, 17th ed. Philadelphia: W B Saunders, 2004; 1642-4.

2. Chan EF. Dyskeratosis congenita: available from http://www.emedicine.com/derm/topic111.htm (accessed on 01/07/2005)

3. Bolton-Maggs P, Thomas A. Disorders of the blood and bone marrow In: McIntosh N, Helms PJ, Smith RL, editors. Forfar and Arneil Textbook of Paediatrics, 6th ed. London; Churchill Livingstone, 2003: 1065-1066.

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Organization as author

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Both personal authors and an organization as author

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Volume with supplement

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Issue with supplement

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No volume or issue

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Books and other monographs

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Editor(s), compiler(s) as author(s)

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Author(s) and editor(s)

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Organization(s) as author

Royal Adelaide Hospital; University of Adelaide, Department of Clinical Nursing. Compendium of nursing practice development, 1999-2000. Adelaide (Australia): Adelaide University; 2001.

Chapter in a book

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Conference proceedings

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Conference paper

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Dictionary and similar references

Dorland's illustrated medical dictionary. 29th ed. Philadelphia: W B Saunders; 2000. Filamin; p. 675.

Unpublished material

In press

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Journal article on the Internet

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Monograph on the Internet

Foley KM, Gelband H, editors. Improving palliative care for cancer [monograph on the Internet]. Washington Academy Press; 2001 [cited 2002 Jul 9]. Available from: http://www.nap.edu/books/0309074029/html/.

Homepage/Web site

Cancer-Pain.org [homepage on the Internet]. New York: Association of Cancer Online Resources, Inc.; c2001-2002 May 16; cited 2002 Jul 9].Available from: http://www.cancer-pain.org/.

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