

AMOEBAS THE QUIESCENT KILLER

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AT THE
OF THE


AMOEBAS THE QUIESCENT KILLER

CLASS NO.	M1
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PREFACE

I have neither an academic record of note, nor facilities to do extensive research, yet during my professional career as medical practitioner in the govt. service for more than quarter century. Noticed that the effect of amoeba in human life was not correctly understood. Hence I thought of printing this booklet of my experience and understanding of the effect of amoeba in human life. Which I presume may benefit humanity immensely. This book is very precise and people of medical knowledge that is academics and professionals may understand it better.

1993.

S. Ambigaipagan.

Fevers:

Fevers due to *Entamoeba Hystolytica* is much more than Hitherto Supposed. And its manifestations are mostly due to affliction of liver and intestine W. H. O. estimates 10% of population is infected with Amoeba.

Hepatic Amoebiasis or Amoebic Hepatitis

Besides the frank liver abscess there is another common liver condition called Amoebic Hepatitis or Hepatic Amoebiasis. It is established that Amoebic Hepatitis may be caused by attenuated infection of liver by Amoeba. This A-Hepatitis cause much of the misery in human life.

Clinically there is intermittent fever specially in the evenings. Muscular exercise and exposure to chilly weather aggravates this condition. Drenching night Sweats, Chills, Nausea Etc. are also seen. There may be hepatic enlargement with tenderness in (R) upper quadrant. This condition is common in winter. Response to chloro quin is also a diagnostic aid. Antibodies are also detected by immunofluorescence.

Treatment:

Metronidazole 800 mg t d s for 5—10 days. or
Tinidazole 2 g initial dose followed by 0.5 g b d
in addition

Chloroquin 600 mg base for 2 days followed by 150 mg base b d for 1 to 6 months depending on the severity of infection provided there is no side effects to Chloroquin.

Fevers due to intestinal Amoebiasis.

Now it is increasingly accepted that one of the common cause of F. U. O. is colitis and specially in tropical and sub tropical climate amoebic colitis form the main cause of P. U. O. or F. U. O. Amoeba establishes in the mucosal crypts of the intestine and then penetrates the submucosa of the colon thus producing the abscesses and this rupture into the lumen and develop into Ulcers. This ulcerative products or abscesses will cause fever until they are eliminated either by resolution or as Diarrhoea etc. and the fever may be high or low depending on the severity of infliction. Continuous fever may go on for even more than weeks if all the products of inflammation are not eliminated. If the elimination is incomplete mild fever will continue making confusion about diagnosis since in Typhoid also there is Diarrhoea after cold. Fever etc.

Clinically there may be continuous fever with or without tenderness along the line of colon with loss of Appetite nausea etc.

Investigatory diagnosis is beyond the scope of this book but we have to bear in mind that now it is established that several Non typhoidal strains have cross-reacting O & H antigen which will cause significant serum Antibody levels.

Antibody antigen Binding in such cross-reaction is weaker. So typhoid must be diagnosed if there is high titre or confirmed by blood culture etc.

So we can see the tendency to pass intestinal amoebiasis as typhoid etc. fulminating amoebic colitis may end up fatally.

Treatment:

From the above it is clear that unless amoebic abscess of the colon breaks down of its own the fever will continue or if its break down is partial there can be mild fever and ill health for several weeks. In the anatomical and pathological set up Hypertonic solution of Magnesium Sulphate (Mist Alba) given repeatedly appears to imbibe out the unwanted products. It is contraindicated in children. And it should be given only after detailed investigation to exclude Typhoid etc. In addition Metronidazole 800 mg tds for 5-10 days or Tinidazole 2 g. loading dose followed by 0.5 g bd for 5 days. If pyogenic infection too is suspected appropriate Antibiotic too is necessary.

Patients of Amoebic colitis. Amoeboma etc. require weeks of indoor treatment and further convalescence for one or two months.

In support of the view that fair proportion of the fevers were due to amoeba I wish to delve into the medical history.

For nearly 3 centuries dovers powder was in vogue for fevers. This dover's powder consists of (a) Ipecac (b) Lactose (c) Opium.

Neither Lactose nor opium has any Antipyretic properties of its own whereas essential Pharmacological product of ipecac is emetine and this emetine acts on amoeba and thus fair proportion of the fevers were

due to Amoeba. Even though Dover's Powder has diaphoretic effect we cannot say that all the fevers came down by mere sweating. Because Dr. Dover combined ipecac in emetic doses.

Steroids in high doses appear to have a seductive effect in amoebic hepatitis and thus many a tendency for amoebic hepatitis to pass off as viral fever (without identifying the virus).

Left ventricular failure:

Indirectly Amoebic hepatitis causes LVF by Hypertension etc. further it also causes direct effect in LVF so much so that a few days treatment for a hepatitis erases out LVF.

Physiopathological condition Not clearly understood. It may be that the abdominal aorta lies in front of the hard vertebra, expanded firm liver edge of amoebic hepatitis may press the aorta thus blocking easy flow of blood and the resultant L V F. Even inferior vena cava too may be squeezed.

Angina Pectoris

Coronary atheroma is mentioned as the cause of Angina. It is accepted that spasm increases the obstruction thus causing angina. And it is also observed that sometimes spasm occurs without atheroma.

This is to state that fair proportion of angina pectoris is caused by the constriction due to vasopressin which was not metabolised by the diseased liver mainly amoebic hepatitis or some other liver disease. And in addition the vasopressin released by the nicotine of smoking.

Treatment

On an average after one week course of amoebic hepaticide the pain starts to subside (provided there is no smoking) in fair amount of patients.

In still others even if amoebic hepaticide does not completely knock out pain on exertion the threshold for pain is increased.

The treatment has to be continued as on page. /

Atherosclerosis

The injury theory is increasingly accepted as the cause of Atherosclerosis. Here too the constrictive property of vasopressin plays a significant role.

Deposition of excessive atheroma at the bifurcation of arteries supports the above view.

Thus when amoebic hepatitis is treated vasopressin is metabolised and athero sclerosis prevented similarly when smoking is given up no vaso pressin.

Infarction:

Main risk factors of infarction can be prevented if Amoebic Hepatitis is treated and smoking stopped thus reducing the chances of infarction.

Risk factors are

- (1) Diabetes mellitus page. 13
- (2) Hypertension page. 6
- (3) Atherosclerosis page. 5
- (4) It is established that spasm cause the sudden contraction and this spasm is also caused by the vasopressin released in stressful situations.

Essential Hypertension:

The main universal cause of essential hypertension is amoebic hepatitis, other liver diseases too have their effect but other liver diseases end up in early fatality or the prodromal symptoms necessitate early treatment whereas amoebic hepatitis goes on quietly for years without producing any other symptoms, in essence when fully understood essential hypertension is a symptom of amoebic hepatitis.

Smoking has its own share in causing hypertension that is nicotine of smoking releases vasopressin. Nicotine mobilise catecholamine also. There is rise in pressure but we have to make sure whether it is due to vasopressin or catecholamines evidences support vasopressin because (1) Vasopressin was extracted in malignant hypertension of smokers No one has extracted catecholamines. (2) Catecholamines mutually antagonise each other (adrenalin or adrenaline).

Physiopathology:

Vasopressin is metabolised by liver and kidney when liver function is incapacitated by amoebic hepatitis or other liver diseases. The vasopressin accumulates causing constriction of smooth muscles of blood vessels and thus raising blood pressure.

In smoking nicotine is released which has a half life of 2 hours and this nicotine releases vasopressin which has a half life of 18 minutes. If an individual smokes 12 cigarettes a day there will be perpetual vasopressin even if the liver functions efficiently.

This hypothesis is further reinforced by the following facts.

1. Clinical trials.
2. Vasopressin was extracted by some researchers from malignant hypertension.
3. It is known that diuresis brings down pressure that means there is an anti diuretic principle that causes the hypertension that is vasopressin (ADH).

It is found that arterial walls of hypertensives contain more water and sodium than normal and this also may be a factor in increased peripheral resistance

4. Vaso dilative drugs bring down the pressure that is a constrictive principle is in action that is vasopressin
5. Essential hypertension is rare in children that is time lapse necessary for the amoebic hepatitis to develop.
6. It is observed that any measures which reduces Sympathetic tone and causes vasodilation will result in a substantial fall of blood pressure that is it follows that hypertension must first be due to vasoconstriction rather than narrowing of vessels that is constriction caused by vasopressin.
7. Now it is accepted that peripheral vascular resistance is the cause of essential hypertension and is also believed that there is a defect in the vascular smooth muscle That is vasopressin constricts the smooth muscles of blood vessels.

8. As the age advances even in non hypertensives patchy distribution of narrowing may be seen. That is narrowing of blood vessels is not the mere cause of hypertension.

Treatment:

Treatment here is really meant to knockout amoebic hepatitis such that liver will regain the functional capacity to metabolise vasopressin hence there are individual variations depending on the intensity of infection etc. Flagyl 800 mg. t d s for 5 to 10 days coupled with Chloroquin 150 mg b. d at least for 1 month. Mild to moderate hypertension of short duration in the young comes down within 3, 4 weeks of chloroquin in the middle aged on an average it takes two months of Chloroquin therapy.

In the elderly despite the involutionary changes that takes place due to aging in fair proportion pressure come down in 2 months of intensive therapy In some it takes even 6 months of treatment of Chloroquin (provided there are no side effects) or even more.

In some cases specially in elderly it is observed that in the first 2 weeks of Chloroquin therapy there was a fluctuation in b. p.

That is sometimes it is increased (which we can control temporarily by diuretics etc.) but subsequently it comes down.

Malignant hypertension is unpredictable specially when other complications has taken place. Even after B. P. is brought down we have to keep in mind of reinfection which is common. If there is reinfection very often it is brought down by short course of therapy.

In smokers nicotine is released and this nicotine release vasopressin and this may raise the pressure for which prevention is the best management.

If there are other liver diseases they need the appropriate treatment for example schistosomiasis etc.

Bronchial Asthma:

Specially in tropics fair proportion of 'Extrinsic', (Atopic or IgE mediated) asthma as well as intrinsic or late onset asthma are due to amoeba.

Amoeba causes B. Asthma in two ways

- (1) Directly IgE mediated
- (2) Liver of amoebic hepatitis does not metabolise vasopressin readily and this vasopressin causes hyper sensitivity.

The first category is found in all age groups where as the latter category is common in late onset since time lapse is necessary for amoebic hepatitis to develop. The above factors may or may not cause overt B. Asthmatic symptoms. But when there are trigger factors like cold air, viral respiratory infection exercise etc. They may appear in episodic forms. In tropics even fair proportion of chronic B. Asthma answers amoebicidal treatment. (both hepatic and tissue has to be treated)

But there is no response if there is tissue damage due to chronicity.

Treatment refer page 1

we have to prevent or treat the trigger factors also.

Perennial Sneezing: (Allergic Rhinitis)

Perennial sneezing, Allergic rhinitis or even unexplained allergy are caused mainly by amoeba specially in tropics.

And this hyper sensitivity usually goes off within one week course of Amoebicide Occasionally we may have to give amoebic hepaticide also.

Primary (Idiopathic) Nephrotic Syndrome:

The cause of minimal change nephropathy is amoeba.

The above contention is supported by the following.

1. By clinical trials
2. More than sixty percent of the above condition is found in tropics where amoebic infection is predominant.
3. Some researchers have said that there is high levels of circulating immune complexes.
4. Since the condition abates to steroids it must be immunologically related.

Maximum lesion of acute nephritis produced by very virulent streptococcus. Whereas minimum lesion nephrotic syndrome is produced by less virulent amoeba.

Treatment refer page *FLAGY*

If the treatment is commenced early, after one week every thing subsides.

Even in adults if the lesion is minimal response is good.

In any case, treatment has to be commenced early, otherwise it may not respond.

Relapse (reinfestation) is common, treatment will have to be repeated.

Abdominal Pain

One of the common causes of Abdominal Pain (specially in tropics) is due to amoeba. One is the infection and invasion of colon. Very often tenderness can be elicited along the line of colon.

The other condition is the pain experienced when the liver expands due to hepatitis. This is common in children too.

At times, it is observed that children squeals with pain when the liver expands. In tropics pain due to amoebae is more common than due to worms.

Treatment refer pages. *1 + 3*

Haemetemeses:

In tropics almost eighty percent of haemetemeses is due to oesophageal varices caused by transient portal hypertension caused by A-Hepatitis

Schistosomiasis too may cause a fair proportion where it is endemic.

Treatment refer page. |

By 3rd or 4th day of treatment condition subsides but treatment has to be continued.

EPISTAXIS

Amoebic hepatitis is the major cause of epistaxes (in tropics) specially in the young ones. May be because the above diseased liver synthesise reduced Amount of factors II, VII, IX, X and fibrinogen Or plasminogen activator may be cleared slowly etc.

Treatment refer page |

Usually on the fourth day of treatment condition improves.

Bleeding P. R.

(? Haemorrhoids or rectal varices)

In tropical condition more than seventy percent of bleeding P. R. are due to portal hypertension caused by venous pressure rising transiently in amoebic hepatitis due to sinusoidal compression by swollen hepato cytes. Other infections like schisto somiasis or even Malaria can cause the same condition if untreated.

In clinical trials it is observed even 1st degree haemorrhoids to subside for amoebic hepaticidal treatment in certain percentage of cases. So it may be that Idiopathic haemorrhoids may Co-exist with rectal varices etc.

Treatment refer page. |

Usually condition starts to subside by the 5th day of treatment. Reinfection common and hence recurrence common.

Diabetes Mellitus

It is accepted that liver disease can cause diabetes mellitus (secondary) now it remains to be convinced that amoebic hepatitis or rather hepatic amoebiasis is a liver disease. And this increased blood sugar (secondary diabetes mellitus) due to the undiagnosed amoebic hepatitis in the long run destroys the betacells (Auto immunity) thus producing the diabetes mellitus proper. (or rather primary diabetes mellitus)

• In the ~~Idm~~ if unchecked, increased blood sugar completely destroys beta cells. Whereas in NIDDM initial destruction of beta cells may increase the deposition of amyloid amylin which may to certain extent protect further destruction.

Even other liver diseases or infections like schistosomiasis malaria etc., can produce the same condition but the prodromal symptoms of these diseases necessitate the patient to go for early treatment or the patient may succumb to the disease early. Whereas undiagnosed amoebic hepatitis can go on for long time and perhaps increased blood sugar may be one of its symptoms than a disease (in the initial stages)

• In obese people we have to consider another factor. That is in obesity there is fatty infiltration of liver cells, depending on the degree of infiltration it

may impair the depot capacity of the liver, for further deposition of glycogen or fat etc. Thus forcing excess food taken to remain as excess blood sugar thus destroying the (beta) cells and so producing the chain of events.

Genetics and other pathology may contribute to few percentage of diabetic Mellitus.

This hypothesis is further supported by the following

1. Clinical trials

2. Diabetes mellitus is more in tropics than in temperate climate and there is no diabetes in eskimos. Because in tropics 20 — 50% of population is infected with amoeba in temperate climate amoebic infection is about 5 — 20% whereas in eskimos it is only a trace.

3. It is claimed that at the initial stage of diabetes mellitus there was enough insulin but it is said to be ineffective. It may be ineffective because the enzyme glucokinase glycogen synthase etc. may not have been adequately produced by the diseased liver. Since insulin can act only in the presence of these enzymes.

Later when the beta cells are destroyed there wont be insulin or less insulin.

4. Primary diabetes mellitus is rare in children. That is time lapse necessary for the amoeba to infect the liver.

5. Physiologically it is established that excess blood sugar can destroy beta cells that in initial excess blood sugar due to A-Hepatitis will destroy beta cells thus producing primary diabetes mellitus. Further there is evidence that IDDM is a slow auto immune disease. Islet cell cytoplasmic antibodies demonstrated in 60—85% of patients with IDDM.

TREATMENT

We know diabetic symptoms manifests when fair proportion of beta islet cells are damaged so the best management is to prevent the sequence of events. That is we have to treat A-hepatitis in advance. Never the less if symptoms or increased blood sugar are noticed in the initial stages, to some extent we can reduce blood sugar thus permitting the the labelled diabetic to lead an average life with moderate food.

Even in Juvenile diabetics it is observed to respond well for early treatment.

Amoebic hepatitis treatment refer page. /

Rheumatoid Arthritis

Though definite conclusions cannot be made at this stage yet following evidences tend to support that fair proportion of rheumatoid arthritis is due to amoebic hepatitis.

- (1) It is accepted that chloroquin cures 50% of rheumatoid arthritis and duration of treatment is at times even 6 months. Amoebic hepatitis too is cured by the same drug after such many days of treatment.

(2) Clinical Evidence

(3) Autoimmunity is accepted

(4) It is accepted that tissue invasion of amoeba
A Humoral antibody Response mainly ^{PROVOKE} IgG and for
R.A. Autoantibody Specificity is immunoglobulin G

(5) Presence of immune complexes at sites of Articular and extra-Articular Lesions,

(6) It is accepted that R A is both an extravascular immune complex disease and a disorder of cell mediated immunity

(7) Amoebic hepatitis gives a persistent antigenic stimulation,

(8) R A develops usually after 30 years, that is time Lapse necessary for Amoebic hepatitis to develop.

HICCUP

Even though various causes are adduced for the hiccup the most common cause (specially in tropics) is amoebic hepatitis. That is the enlarged liver of amoebic hepatitis press on the diaphragm thus irritating the nerves of respiratory muscles etc. And thus producing the hiccup. The hiccup usually disappears within 5 days of treatment for amoebic hepatitis.

Primary (Idiopathic) Epilepsy

Very often intractable tonic clonic of the idiopathic epilepsy responds to amoebic hepaticides (i.e. A few percentage of all epilepsies)

Exact etiology is not understood but most probably certain protein products by the action of bacteria on protein in gut may pass liver, due to transient portal hypertension caused by amoebic hepatitis, and enter into brain and cause the fits.

Atopic dermatitis (Main Eczema)

Both in children and adults amoeba appears to be the main culprit of atopic dermatitis in tropics it tops the list of all eczemas.

Ingested Amoebic antigens produce excess IgE antibodies and thus causing dermatitis. Another way by which dermatitis (Hyper sensitivity) is produced by amoeba is by the unmetabolised vasopressin. Since amoebic infected liver cannot metabolise vasopressin completely.

Dermatitis is aggravated by bacterial skin infection contact dermatitis, change in humidity, wool garment, primary irritants etc.

Treatment refer page |

Some times chloroquin itself causes pruritus.

Pregnancy Induced Hypertension

Multiple pregnancy, Hydatidiform mole hydramnios etc. accounts for few percentage of P I H.

Whereas the main cause of pregnancy induced hypertension is due to the infection (intestine) or tissue invasion by amoeba this hypothesis is support by the following

1. Clinical trials
2. Pregnancy is an immunodeficiency state where the percentage of amoebic infection is more than in normal people.
3. Immuno fluorescent techniques have established diposition of immune complexes & IgM in renal glomeruli and spiral arterioles of the placenta.
4. It is mostly found in women of the poor and underpriveleged communitis That is chances for amoebic infection are more.
5. Frequency of PIH in the first pregnancy than in the subsequent pregnancies can be explained by supposing that certain amount of immunity might have been conferred by the amoebic infection in the first pregnancy.

TREATMENT

In the first trimester flagyl is contra indicated even though it is not teratogenic in the 2nd or 3rd trimester the earlier the treatment commenced the better. Usually by the first week of treatment symptoms disappear. There is the possibility of reinfection after one or two months which may warrant treatment again

If detected just before delivery all symptoms may or may not disappear (very often it is possible to knock out at least one or two of the three symptoms)

Dosage of flagyl refer page. 3

Secondary Dysfunctional Uterine Bleeding

Medically it is established that liver disease is one of the cause of dysfunctional uterine bleeding.

It remains to be accepted that amoebic hepatitis is also a liver disease and in tropics almost 70% of the DUB is due to amoebic hepatitis (Malaria, Schistosomiasis etc. also may cause the bleeding if untreated)

ETIOLOGY

Diseased liver (A - Hepatitis) does not metabolise the oestrogen readily. When the oestrogen level rises FSH goes down by feed back so oestrogen level falls then shedding of endometrium takes place.

Treatment refer page |

First metronidazole should be given for 5-10 days after bleeding has stopped we can give chloroquin.

Reinfection is common, hence recurrence is common.

Infertility or Subfertility

From the above chapter it is clear that A-hepatitis can cause DUB and brings down the level of FSH. When FSH goes down ovulation cannot take place and hence infertility or subfertility. And it is found that correction of A-hepatitis restored ovulation. Other liver diseases can also cause similar condition.

The above hypothesis is supported by the fact that anovulation is treated by clomiphene citrate which is an antiestrogen.

Corollary Evidence

We commonly encounter cases where a single patient has all the diseases such as hypertension, diabetes mellitus, Bronchial asthma etc.

Where the underlying factor is amoebic hepatitis if amoebic hepatitis was knocked out early all the diseases could have been prevented.

In one family very often all members have A-hepatitis.

Schizophrenia

In clinical practice it is observed that some cases of schizophrenia have root cause as amoebic hepatitis. Still others were found to have malaria as the cause. (both afflict liver)

Anyway further trials necessary to say anything conclusively.

