

UNUSUAL PRESENTATIONS OF AMOEBIC HEPATITIS.*

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I have chosen this disease which is very common in our part of the world for the simple reason that it might be an interesting subject for discussion to most of us, as it can present in most unexpected ways and can baffle many practitioners.

Case No. 1

Kedar 34 yrs. Male—was treated for acute pain in abdomen for 3–4 days as ? acute cholangitis and was then admitted in the surgical ward with features of collapse and diagnosed provisionally as a case of perforation and peritonitis. When I saw him the patient was in great pain. The whole of abdomen was rigid but more so in the right hypochondrium B. P. 90/60. Dullness over both the lung bases but more on the left side. At first acute pancreatitis with bilateral pulmonary embolism was suspected. But serum amylase and E. C. G. were not infavour. E. C. G. showed evidence of non specific myocarditis. With the possibility of an amoebic liver abscess which burst into the left pleural cavity in mind, dihydro-emetine was started. Gradually he showed signs of improvement, later typical "anchovy sauce" fluid was aspirated from the left pleural cavity and on completion of the emetine therapy, he made very good recovery.

Case No. 2.

Male 55 yrs old, with a history of pain in the right shoulder for the last 6 months. He came to me after being investigated at several hospitals. As the case seemed to be a difficult one, he was investigated again thoroughly as follows:—

- 1) X-Ray chest —normal
- 2) E. C. G.—inverted T waves over the right precordial leads.
- 3) E. S. R. 80 mm in the first hour.
- 4) Urine — Alb. +
- 5) Stool — NAD (Nil abnormal detected)
- 6) Barium meal — Normal
- 7) Oral Cholecystography — normal.

Till now the patient was afebrile — but he was markedly wasted and looked very ill, and I started thinking of malignancy, somewhere as the cause, in view of his age & high ESR. In the mean time he started getting high fever, continuous in nature. As the diagnosis was not clear several antibiotics were administered but without any benefit. The routine investi-

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gations were of no value. X-Ray picture of chest on the third day of fever was normal. But the second X-Ray chest on the 6th day of fever showed a big hump over the right dome of the diaphragm. Nature of the hump could not be assessed; the third X-Ray on the 9th day showed a second mass near the mediastinum and the 4th X-Ray on the 12th day showed a third mass peripherally. The Radiologist suggested an encysted fluid in the pleural cavity, and also such a rapidly developing lesion could be malignancy. Clinically also we thought it was probably malignant metastasis and therefore wanted to aspirate. On the first aspiration over the shadow we get some blood. And microscopic examination was reported as showing malignant cells. The diagnosis of malignancy was supported. But when we noticed some pus-like material at the bottom of the aspirated fluid we decided to put a needle in the liver. Now the real pathology was revealed as frank anchovy sauce fluid was aspirated. Aspiration was carried out thirteen times and ultimately after giving a full course of emetine I requested the surgeon to open the pleura and drain the fluid. The patient had a wonderful recovery and was discharged 50 days after the admission.

CASE NO. 3.

Male 60 yrs. He came to the hospital with haematemesis. A diagnosis of peptic ulcer with haematemesis was made. The way the patient was bleeding indicated the need for surgery and on the 4th day he was transferred to the surgical side. He was operated while his general condition was still low, and was found to be bleeding from one of the main branches of a gastric artery. A partial gastrectomy was performed. Till then he had received thirteen pints of blood. On the 6th day of operation he developed deep jaundice and I was asked to see the patient. I could not detect any significant abnormality and told the surgeon that it was probably an iatrogenic obstructive jaundice. Repeated urine exam. showed absence of urobilinogen and presence of bile, and serum bilirubin was reported 38 mg, suggesting possibility of a haemolytic jaundice. So far the patient was afebrile but did not look very cheerful and continued to look cachectic. ESR was persistently high. In the mean time he developed fever which was intermittent and also complained of pain over the right side of the chest. On examination there was dullness over the right infrascapular region. By then the jaundice had cleared. There was tenderness over the tenth intercostal space and also a pleural rub. X-Ray showed fluid in the right pleural cavity. In retrospect a diagnosis of an amoebic liver abscess which had burst into the pleura was made. So long as the abscess was in the liver tissue in a tense situation he was developing jaundice and when it burst out the pressure was released and the jaundice had improved. So we started dihydroemetine and the patient started feeling better and the temperature gradually subsided. At that stage the pleural fluid was aspirated and to our great surprise it was straw coloured. In three consecutive aspirations it remained straw coloured. In order to confirm the diagnosis of amoebic liver abscess we punctured the liver but got only blood. We were still hoping to get amoebic pus inside the liver so we punctured the liver again. Though only blood was coming through aspiration we went on pushing the needle further with a vacuum in the syringe and at a certain depth inside the liver we obtained pus. We felt very happy and the pus was sent to the laboratory for microscopic examination and culture. Microscopic examination did not reveal much

except a lot of pus cells and culture showed exuberant growth of *E. coli*. *E. coli* might have produced an ascending cholangitis and ultimately reached necrosed liver cells. Clinically it was a case of amoebic liver abscess and growth of *E. coli* was not the primary cause of the liver abscess.

CASE No. 4.

Male 55 yrs. He came to us with deep jaundice and swelling of the abdomen. On examination the swelling was the liver which had extended more downwards and was quite tender. As his condition was low I decided that the liver abscess should be drained by a surgical procedure. Five pints of anchovy sauce pus was aspirated and a drainage tube left behind. We also started emetine. He made a wonderful recovery.

CASE No. 5.

Male 70 yrs. He came to the medical O. P. D. with a swelling in his abdomen. He had no other complaints. On examination there was a round swelling of the size of a tennis ball occupying the epigastric region. It was not very tender and was moving with respiration freely. Diagnosis was undetermined, but the fever made us suspicious of amoebic liver abscess in the left lobe of liver, and in the second place a malignant tumour was also thought of. So I advised a laparotomy-amoebic liver abscess was found over the left lobe.

CASE No. 6.

Male 60 yrs. He had fever and pain abdomen for which he consulted a doctor who treated him as a case of amoebic hepatitis. He was jaundiced and had a tender liver. Because of shortage of bed he was discharged although he was not cured. Seven days after being discharged the patient was brought back to the hospital and was admitted with the complaints of severe pain abdomen and intermittent fever reaching upto 105 F.

He looked very toxic and was also slightly jaundiced. There was a swelling occupying the right and left hypochondria but more definitely over the epigastric region and was tender only on that spot. With the provisional diagnosis of amoebic liver abscess we put him on emetine and gradually he felt better. His temperature subsided. Epigastric swelling persisted and he still had some tenderness there. I referred the patient to the surgeon to be explored but the patient was unwilling to undergo any operative procedure. He went home only to return after a fortnight and was then operated. On laparotomy the epigastric swelling was of the left lobe of the liver and on aspiration it was found to be an amoebic abscess.

DISCUSSION

Six cases have been presented and I think we met problems in almost all of them. I will now discuss the problems encountered in these cases of amoebic abscess.

Of the six, three cases presented with jaundice. Jaundice is a very uncommon finding in amoebic liver abscess. The question arises about the possible mechanisms of this jaundice.

Is it mechanical pressure by the abscess mass on the Caudate lobe thereby pressing on the bile duct? It could be, but jaundice is not present in the majority of cases of amoebic liver abscess. So mere pressure could not be the cause of jaundice. Could it be toxic? Manson Bahr mentions in his book that the absorbed necrotic material could act as toxins and give a picture of hepatitis but in our small series we got an obstructive type of jaundice. Finally, is it possible to have sclerosing cholangitis? Nowhere in the literature I know has sclerosing cholangitis been recorded as causing jaundice in amoebic liver abscess. Probably both pressure and toxins play a role. Whether some other factor is also responsible remains to be studied.

The second interesting finding was to get straw coloured fluid from the pleura instead of anchovy sauce pus. I think this was sympathetic effusion above the amoebic pus. But the amount we aspirated in one of the jaundiced patients was quite large about six pints in three aspirations. So in cases suspected of an amoebic liver abscess bursting into pleura, if such fluid is aspirated it should not disappoint us as it is not necessarily against an amoebic liver abscess. Frank blood in pleura in amoebic liver abscess which has burst there has been mentioned by Manson Bahr. He says sometimes it may be confused with malignancy. It is not an uncommon finding.

The third point of interest was about aspiration. Normally if we puncture through the maximum tender spot we get amoebic pus. But if the abscess is deep enough and has burst out through other places then the area of maximum tenderness may not be of great importance. When the liver is punctured through such a spot we may get frank blood to start with and if the aspirating needle is pushed further creating vacuum in the syringe we may get pus at a certain depth. This is specially true in difficult cases.

Case no. 1 who was admitted with features of collapse in the surgical ward with a provisional diagnosis of perforated viscus was even more of a problem as it would be difficult for anybody to diagnose bursting of a liver abscess into the pleura as the cause of profound shock.

The cases which have been discussed were diagnostic problems because either the patients were brought as an emergency needing concentrated efforts in resuscitation or the patients did not have any significant hepatic enlargement either upwards or downwards and the possibility of liver abscess was not at first thought of. Scanning the liver after administering isotopes as Rose Bengal dye would help in earlier diagnosis of such difficult cases.

Although a very high ESR in such cases maybe of some value, inversion of T waves in a few of the chest leads or presence of albumin and pus in urine should not mislead us in such cases to a diagnosis of myocarditis or chronic pyelonephritis. Toxins liberated by necrosed liver cells could produce these changes. Probably the best way to avoid pitfalls in diagnosis in such cases is to keep the possibility of amoebic liver abscess constantly in mind while practising in Nepal, particularly in pyrexias of unknown origin if long continued or pyrexia with unexplained shock or shoulder pain of long duration.

Ref : Manson's Tropical Diseases by Sir Phillip Manson Bahr. 16th Edition 1966.