A CASE OF CHRONIC ATROPHY OF THE LIVER.

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O.C. a female aged 45 years, was admitted to hospital complaining of recurrent attacks of jaundice during the last three years. Careful questioning elicited the following history.

Patient had been quite well until about four and a half years ago when she suffered from an indefinite dyspepsia of which, however, she took little notice. About the same period she noticed intensive irritation of the skin of the whole of the body and limbs, for which no cause was discovered and which in spite of treatment persisted for some months.

Four years ago, i.e., six months after the onset of the dyspepsia, the patient suffered a moderately large intestinal hæmorrhage, for which she was treated by rest in bed and a peptic-ulcer diet. A barium meal examination at this time is reported to have shown a duodenal ulcer. After a few months in bed the patient resumed her household duties and observed an ulcer-diet régime, which has been continued until the present.

Three years ago the patient first noticed the attacks of which she is now complaining. These attacks are of the following nature. There is first a sensation of chilliness accompanied by headache and nausea but no vomiting. At the same time there is also a sense of discomfort in the epigastrium and right hypochondrium which may extend to the back. Jaundice then becomes noticeable, increases for a few days and thereafter diminishes in intensity. The urine is dark in colour and the stools pale during the attack. During the first forty-eight hours of the attack there is some pyrexia and the patient is confined to bed.

At first these attacks were infrequent and the patient felt quite well in the intervals. Recently, however, the attacks had become more frequent and during the intervals slight jaundice had persisted, and the patient had felt poorly though still able to carry on. The weight is stated to have remained stationary.

Past History. Thirty-five years ago tuberculous adenitis (groin).

Family History. Four children alive and well, no history of jaundice or any blood disease in the family.

Condition on Examination. She is a well-nourished woman, slightly pale, and definitely jaundiced. The veinules on the cheeks are slightly more noticeable than usual. Tongue is clean; teeth well cared for. Heart and lungs and central nervous system reveal nothing abnormal. B.P. 140/90. Abdomen well covered and lax. There are no abnormal contours and no dilated veins. Some tenderness to palpation present in the right hypochondrium. Liver and spleen are *definitely not* palpable. No ascities present. Rectal examination is negative.

The following special investigations were carried out.

Blood Count. R.B.C. 3,560,000 per c.mm.; Hb. 60%; C.I. 0.86.

W.B.C. 5,000 per c.mm.

Polymorphs 60%.

Lymphocytes 36%.

Large mononuclears 3%.

Eosinophils 1%.

Halometer 7.6μ ; red cells show some anisocytosis.

Fragility of Red Cells. Hæmolysis commences with .35% saline and is complete with .25%. The cells are thus less fragile than normal.

Van den Bergh Reaction. Immediate direct reaction, positive. Indirect reaction, 6 units.

Wassermann Reaction negative.

After the patient had taken Ferri ammon. cit. 3grs. t.d.s. for seven days a further blood count gave the following result:—

R.B.C. 5,000,000 per c.mm.

Hb. 78%.

C.I. 0.78.

W.B.C. 8,000 per c.mm.

Reticulocytes 2%.

Radiological examination of the gall bladder after oral dye:—no evidence of concentration of the dye; no evidence of opaque gall-stones found.

Commentary.

From the above history and physical examination the following conditions had to be considered in the differential diagnosis.

1. Stone in the common duct.
2. Chronic hæmolytic jaundice.
3. Syphilis of the liver.
4. Portal cirrhosis.
5. Hanot's or biliary cirrhosis.
6. Banti's disease.
7. Hodgkin's disease.
8. Carcinoma of the liver.
9. Chronic subacute atrophy of the liver.

The negative Wassermann reaction and the absence of other *syphilitic* manifestations ruled out the possibility of syphilis of the liver.

The long history negated *carcinoma* of the liver.

Portal cirrhosis was considered but a portal cirrhosis of four years' standing which had progressed to the stage of producing marked jaundice would almost certainly have produced marked enlargement of the liver and ascites. The absence of these findings was considered therefore sufficient to make this diagnosis unlikely.

Banti's disease had to be carefully considered, particularly so because it might be that the initial hæmatemesis was not due to a duodenal ulcer but was, as is not uncommonly the case, the first sign of the Banti syndrome. Again, the blood count, showing a secondary anæmia with a low white cell count, is often found in Banti's disease, but in Banti's disease the spleen is always enlarged, and by the time jaundice has developed the liver also has enlarged and ascites has appeared. Therefore in the absence of these signs Banti's disease was excluded.

Hodgkin's disease can produce a chronic jaundice of many years' standing, but the absence of an enlarged spleen or palpable glands was considered to render this diagnosis most improbable.

The history of recurrent attacks of jaundice over a long period was extremely suggestive of a *chronic hæmolytic jaundice*, but in this condition the jaundice would have been acholuric which was not so in this instance. Again, in this condition the spleen is usually definitely enlarged. Hence this diagnosis was not expected to be correct and this opinion was confirmed by the slightly diminished "fragility of the red cells," a result to be expected in a case of chronic jaundice, while in a case of chronic hæmolytic jaundice the fragility would have been increased.

Hanot's cirrhosis was suggested by the chronic jaundice, the attacks of fever and pain in the hepatic region, and also by the absence of ascites, but Hanot's cirrhosis is almost always associated with a marked enlargement of the spleen, and in the absence of this feature the diagnosis of Hanot's cirrhosis was considered unlikely.

Thus the differential diagnosis was left between a *stone in the common duct* and a *chronic subacute atrophy*.

The history of attacks of pain in the hepatic region, with some febrile reaction followed by jaundice and pale stools and dark urine is common to both conditions. The fact that the pain had never been very severe was in favour of a chronic atrophy, as was the fact that an unexplained pruritis had preceded the onset of the illness. On the other hand, the negative result of the cholecystogram did not exclude the possibility of stone in the common duct.

When two diagnoses seem equally likely, it is a good rule to remember that the commoner condition is the more likely, but in this case, neither of the diseases under consideration could be said to be more common than the other.

The unexplained pruritis one year before the onset of jaundice made the diagnosis of a chronic atrophy a "slight favourite," but it was argued that this symptom in itself was not sufficient to contraindicate an exploratory operation, which might reveal a common duct stone, and at the same time permit of its removal which would "cure" the patient. It was therefore decided to explore the abdomen. The operation was performed by Mr. Meyrick Thomas who found the common duct and gall bladder to be free of stones and that a probe passed into the duct was offered no resistance. The spleen was found to be normal in size, the liver was not enlarged, was smooth, and looked healthy, but a piece of the liver was removed for section, and the abdomen was closed. Apart from some delayed bleeding from the wound and an attack of *Bacillus coli* pyelitis, the patient made a good recovery from the operation.

The section of the liver was reported as showing **chronic degenerative hepatitis**.

Such cases of chronic subacute hepatitis are rare but they are seen from time to time. The diagnosis is always difficult and can only be made by a process of exclusion, and then only tentatively unless the liver is explored. In the two other examples which have come under my observation this procedure had to be resorted to before a diagnosis could be definitely established.

The prognosis in this disease is always serious, though life may be prolonged for four to ten years from the time the symptoms first appear.

Treatment offers very little hope. Obviously any septic focus, if found, should be dealt with. The diet should be poor in protein and fat but high in carbohydrate. The giving of glucose and insulin once or twice a day helps to increase the glycogen store in the liver cells and is considered a procedure of some value.