INFECTIVE HEPATITIS WITH HEPATIC CIRRHOSIS

BY

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The patient was a thirty-four-year-old British soldier who had been a prisoner of war in German hands from 1940 until he was liberated in 1945. He was on the "thousand-mile forced march" from Poland to Germany in the face of the advancing Russian Army. While in captivity he had little food to eat and his meals were very small. On return to the U.K. he found that he could never eat more than very small meals without feeling sick and vomiting. This state of affairs continued, but in spite of that he must have eaten adequate food as he appeared to be of average nutrition.

He was admitted to B.C.O.F./29 General Hospital on 16th February, 1951. A fortnight previously, he reported sick when he began to complain of anorexia and nausea. About nine days later, he noticed that his eyes and skin were yellow in colour and that his urine was dark. On admission to hospital he was deeply jaundiced, the liver was palpable two finger-breadths below the right costal margin and it was tender. The spleen was just palpable, the stools were pale and the urine contained bile pigments, bile salts and an increased amount of urobilinogen, but nothing abnormal was noted on microscopic examination. He was febrile (T. 100.8° F.), but he became afebrile the next day. Blood films, thick and thin, showed no evidence of malarial parasites, etc.

He was treated with a high protein, high carbohydrate, high vitamin and fat restricted diet.

21st February, 1951: Serum bilirubin, 12.8 mg. per cent. Thymol turbidity, 8.0 units.

7th March, 1951: W.B.C., 8,000 per cu. mm. (Polys, 62 per cent. Lymphs, 32 per cent. Monos, 2 per cent. Eosins, 4 per cent.)

8th March, 1951: Serum bilirubin, 9.75 mg. per cent. Thymol turbidity, 10 units. Takata Ara—positive.

On 9th March, 1951, he appeared to be making slow but definite progress. His appetite was better and his stools were darker. However, on 10th March, 1951, he complained of flatulent abdominal discomfort, his appetite was poor, the liver was palpable three finger-breadths below the right costal margin and
Infective Hepatitis with Hepatic Cirrhosis

he began to run an irregular pyrexia. He vomited on 12th March and again on 13th March, when examination revealed marked abdominal distension with bulging in the flanks, shifting dullness and a fluid thrill. There were no petechiae. Paracentesis abdominis was carried out and 82 ounces of bile-stained fluid were removed. The laboratory report on the fluid was as follows: R.B.C. + + +. Nucleated cells. No bacteria seen. Small lymphos, 95 per cent. Endothelial cells, 4 per cent. Neutrophils, 1 per cent. No A. & A.F.B. seen. Total protein, 2.7 g. per cent. Culture: Sterile after 48 hours.

He was given procaine penicillin, 300,000 units intramuscularly daily, but this had to be stopped when he developed a generalized urticarial reaction which was treated with pyribenzamine, an anti-histamine preparation.

13th March, 1951: Blood urea, 62 mg. per cent. Serum bilirubin, 14.2 g. per cent. Plasma proteins, 7.134 g. per cent. Urine cultures (6), sterile. Stool cultures (6), no pathogens isolated. Repeated microscopic examination of stools revealed no evidence of intestinal parasites.

X-ray of abdomen: Calcified glands on both sides of abdomen. No other abnormality seen.

15th March, 1951: He was still febrile. The urine was heavily bile-stained and contained a trace of albumin, with plentiful casts, which were chiefly granular, though a few were epithelial and hyaline. A single leucine crystal was seen. Very occasional pus cells and R.B.Cs.

Serological tests for leptospirosis were negative.

On 17th March, 1951, it was evident that paracentesis abdominis was necessary and 76 ounces of bile-stained fluid were removed. Repeated blood counts showed no evidence of mononucleosis.

19th March, 1951: Plasma proteins, 6.02 g. per cent. Serum bilirubin, 12.8 mg. per cent.

He was still febrile (101.4° F.) and there were numerous fine crepitations at the base of the right lung. The total and differential white blood cell count was within normal limits. He was ordered chloromycetin 4 g. stat. and ½ g. six-hourly with evident improvement.

He became afebrile on 21st March, 1951, when examination of his heart revealed occasional extra systoles. He was now eating well and his stools were obviously bile stained. Chloromycetin was stopped on 23rd March, 1951, and jaundice appeared to be lessening appreciably.

He became febrile again on 26th March, 1951, and on 27th March, 1951, he felt very ill. There was now pitting oedema of both feet, ankles and legs, and pitting on pressure over the sacrum. The abdomen was markedly distended. Paracentesis abdominis was carried out for the third time and five pints of bile-stained fluid were removed. The fluid had the same characteristics as on the first occasion. B.P. 130/88. The pulse rate swung between 70 and 90. Blood culture was sterile. Chloromycetin was exhibited again.

On 29th March, 1951, the liver edge was palpable two to three fingerbreadths below the right costal margin and the oedema of the legs appeared to
have gone, but there was still some pitting over the sacrum. He was seen by Lieut-Colonel J. C. Watts, M.C., Surgical Specialist, who considered that there was no indication for surgery and concurred with the diagnosis of infective hepatitis with hepatic cirrhosis.

On 31st March, 1951, he felt much better although he still had a low grade pyrexia. Serum bilirubin was now 8 mg. per cent. The urine was less obviously bile-stained. He was now eating extremely well; in actual fact he had two meals at each meal time, i.e., he was eating larger meals than he had ever eaten in his life before.

On 1st April, 1951, his abdomen was markedly distended and once more there was gross pitting edema of the lower limbs and over the sacrum. Paracentesis abdominis was carried out and 76 ounces of bile-stained fluid removed. The plasma proteins were now 4.46 g. per cent. and serum bilirubin 5.8 mg. per cent. He now had obvious hypoproteinaemia and two pints of plasma were given slowly intravenously. Chloromycetin was continued. He was given two pints of plasma on 3rd April, one pint on 4th April and two pints on 5th April. Paracentesis abdominis was repeated on 4th April, 1951, and 65 ounces of fluid obtained. B.P. 130/90. On 5th April he developed lymphangitis of the right arm where he was having plasma. Chloromycetin was stopped and aureomycin 1 g. stat. and 250 mg. six-hourly exhibited. B.P. 125/85. Plasma proteins were now 4.9 g. per cent. X-ray of chest revealed obliteration of the left costophrenic angle, due to fluid.

6th April, 1951: Appetite excellent. Blood urea, 28 mg. per cent. R.B.C., 3.5. million per cu. mm. Hb., 10 g. per cent. He was given a further two pints of plasma intravenously.

7th April, 1951: Paracentesis abdominis repeated and 65 ounces of bile-stained fluid obtained, of protein content 2.5 g. per cent. He was now given Campolon 10 ccs. intramuscularly for five days and two tablets of ferrous sulphate thrice daily.

8th April, 1951: No oedema apparent anywhere. Eating extremely well. Feels very much better but is still febrile.

9th April, 1951: Transfusion of one pint of fresh blood. Plasma proteins, 5.5 g. per cent. Temperature had now become normal.

10th April, 1951: Aureomycin continued. Paracentesis abdominis carried out for the seventh time and 72 ounces of bile-stained fluid removed. He was now very much better and fit to be evacuated to the U.K. by air. His name was removed from the D.LL and placed on the S.LL. He left on his long journey to the U.K. on 11th April, 1951. He was afebrile and, though he still had demonstrable fluid in the abdomen and his liver was easily palpable, his appetite was excellent, the serum bilirubin was falling, the urine contained appreciably less bile, the stools appeared to be of normal colour and the plasma proteins were rising. He was given a supply of aureomycin to last the journey to the U.K.

He arrived at the Military Hospital, Tidworth, on 24th April, 1951, where Major J. P. Baird, R.A.M.C., reported as follows: "Feels well. Appetite good and abdomen feels comfortable. No evidence of anorexia. Liver enlarged three

**Blood:** Total proteins, 6.0 g. per cent.; albumin, 4.2 g. per cent.; globulin, 1.8 g. per cent.; R.B.C., 4 million per cu. mm.; Hb., 14.8 g. per cent.; serum bilirubin, 1.1 mg. per cent.; thymol turbidity, 1 unit; blood cholesterol, 112 mg. per cent.; W.R. and Kahn, negative; Rhesus group, negative.

**Urine:** No bile salts or bile pigments present. Urobilinogen, negative.

**Progress:** Aureomycin was stopped on day of admission. No evidence of pyrexia. His clinical condition has steadily improved. The ascites slowly disappeared and further paracentesis was not needed. His weight has steadily increased and there is now (7th June, 1951) no evidence of jaundice. Now active all day and feels very fit.

**Present Condition (7th June, 1951):** Weight, 11 st. 4 lb. No jaundice. No evidence of anorexia. No free fluid in abdomen. Liver edge palpable one finger—fine and nodular. No œdema.

**Blood Chemistry:** Total proteins, 6.5 g. per cent.; albumin, 4.8 g. per cent.; globulin, 1.7 g. per cent.; serum bilirubin, 0.3 mg. per cent.; thymol turbidity, 1 unit; thymol flocculation, negative.

**Urine:** No albumin. No abnormal biliary constituents.

**Disposal:** Sent on sick leave for one month. To return for further observation.

The following additional information was received from Major J. P. Baird, R.A.M.C., in a letter dated 16th July, 1951:

“He remains well, has plenty of energy and an excellent appetite. He has been fully active while on leave. Examination shows no jaundice and he is not clinically anaemic. There is no glandular enlargement and the liver and spleen are not palpably enlarged. His weight is 12 st. 5 lb. and the following investigations were performed:

**Blood:** Hb., 15.5 g. per 100 ml. (104 per cent.); R.B.C., 5.3 millions; E.S.R., 5 mm. in first hour (Wintrobe); serum bilirubin, 0.4 mg. per 100 ml.; serum protein, 7.5 g. per cent.; albumin, 5.0 g. per cent.; globulin, 1.9 g. per cent.; thymol turbidity, 1 unit.

**Urine:** No bile pigments or salts present.

The patient has been downgraded to P7 (H.O.) and returned to his depot.”

**Commentary**

It is accepted that malnutrition can result in cirrhosis of the liver. It is possible that during several years of captivity this soldier’s liver suffered damage due to sub-nutrition and that when he developed infective hepatitis it suffered further and severe damage resulting in marked cirrhosis with ascites, hypo-proteinæmia and secondary pitting œdema of the extremities and over the sacrum.

This was combated by plasma intravenously supplemented by a transfusion of one pint of fresh blood to help counteract the anaæmia and as a tonic in this case of prolonged active infection. He had large doses of liver extract intramuscularly as well as ferrous sulphate and vitamin supplements by mouth.
There is no reason to doubt that this was a case of infective (viral) hepatitis, and not just a manifestation of leptospirosis, enteric-group fever, infective mononucleosis, staphylococcal septicæmia, amœbiasis or other conditions. The prolonged and irregular pyrexia was most unusual in our experience of infective hepatitis, but it may just have been "hepatic fever" and, therefore, just a measure of active and extensive liver damage. Of course, secondary infection had also to be considered. The voracious appetite developed by the patient while he was still extremely ill was taken as a most welcome prognostic sign. It is felt that one of the best liver function tests is a good appetite.

The rapid response to treatment with chloromycetin when first exhibited was of interest, as was the response to treatment with aureomycin following the development of lymphangitis during the intravenous administration of plasma whilst the patient was having a second course of chloromycetin.

What the future of this case is likely to be is difficult to say. The liver has great powers of recovery, but if it has suffered severe and extensive damage and there is gross post-necrotic scarring, then he may not live more than a year or so. There is no doubt that the patient did extremely well and it is felt that in some measure this was due to the manner in which the case was managed and nursed. However, it is not the worst case of infective hepatitis that we have seen recover.

Our knowledge of the mode of spread of this disease has increased during the past decade. However, in spite of the fact that some individuals continue to speak of post-arsphenamine jaundice, homologous serum jaundice, etc., it would appear to be fairly well established that these terms are synonyms for one disease in which the virus may enter the human body by droplet infection, by the ingestion of contaminated food and water, or be introduced through the skin, the result of venepuncture, blood transfusions, infusions, injections of any type, vaccinations, tattooing, and insect bites, especially by blood-sucking insects which are really viable needles and syringes.

The principles of treatment of infective hepatitis are a high protein, high vitamin, high carbohydrate and fat-restricted diet. Where the appetite is poor, extra protein can be given as Casinal (Bengers) or Casilan (Glaxo). Such cases normally have barley sugar by the bedside and have extra glucose in fruit drinks as well as vitamin supplements. Severe and urgent cases, especially those of acute hepatic necrosis, are best treated on the lines indicated in the Brit. med. J., 30th September, 1950, and the J. Roy. Army Med. Corps, December, 1947, No. 6, Vol LXXXIX, pp. 290—300.

All cases of infective hepatitis are normally kept in bed until the urine is normal, and after one week up and about they are sent on a fortnight's convalescence at the Rehabilitation Centre. None of these cases are subsequently accepted as blood donors.

It is normal practice in the Army to advise all cases to avoid alcohol and most anaesthetics except in an emergency for six months at least. In our extensive experience of this disease the only relapses we have seen have been in those who did not follow our advice regarding alcohol.

It is known that the virus of infective hepatitis is passed in the urine and stools.
Primary Carcinoma of the Stomach

It may be of interest to record that one medical officer, who zealously tests for bile pigments every day in these cases by the “froth” test, developed infective hepatitis. It was suggested that his fingers had become contaminated with the infected urine. Whether or not such was the mode of transmission of the virus of infective hepatitis in the case of this M.O., it is at least feasible and possible and is well worth bearing in mind.

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Primary Carcinoma of the Stomach in a Woman Aged Twenty-Five

by

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The following case is of interest owing to the rarity of the condition at this age.

Mrs. S., wife of a soldier, was admitted to the Louise Margaret Hospital, Aldershot, on 6th March, 1951, with seven months’ history of loss of weight accompanied by vomiting. Vomiting was almost daily in occurrence, large amounts of food, sometimes mixed with blood, being brought up. During this period she developed a sense of fullness in the epigastrium after meals, but it is interesting to note that at this stage she did not experience any true anorexia. There were no other relevant symptoms apart from amenorrhea for the previous eight months.
Hepatic Cirrhosis

Infective Hepatitis with Hepatic Cirrhosis

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