Clinical and other Notes.

AN INTERESTING LIVER CASE.

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The case is one of acute yellow atrophy of the liver, occurring in a non-commissioned officer, aged 30, of ten years' service, whose medical history sheet showed no entry of interest other than amoebic dysentery, of twenty-six days' duration at Jubbulpore in 1929.

He was admitted to hospital with a history of headache and insomnia for three days, and pyrexia for the previous twenty-four hours. After admission his fever and symptoms quickly subsided. Blood slides were negative for malaria. Two days later he was seen to be slightly jaundiced and the following day he vomited profusely. The same night he became violently delirious, requiring restraint and morphia.

At examination next morning the symptoms, except for the normal temperature and slight jaundice, suggested the onset of cerebrospinal meningitis; the white cell count revealed a leucocytosis of 18,000, with polymorphonuclear leucocytes eighty-five per cent. The optic discs were seen to be intensely congested. The urine at this stage was suppressed. Lumbar puncture revealed a clear fluid not under pressure. He was removed to the Government Fever Hospital, where he died the same evening, a provisional diagnosis of septicaemia being made.

Examination of the cerebrospinal fluid showed a clear, colourless fluid and no pellicle on standing. Protein estimated by the proteinometer was 40 or 50 milligrammes per 100 cubic millimetres; globulin, nil. Cell count, 4 cells per cubic millimetre. Centrifugalized deposit showed scanty mononuclear cells and no organisms on staining. Culture proved negative.

As there seemed some doubt as to the actual cause of death a post-mortem examination was carried out the following day.

The body was well nourished but on the lean side. Skin jaundiced, but no petechial haemorrhages were detected. The brain was removed, but except for some general adhesions of the dura to the skull appeared quite normal. The heart showed evidence of fatty infiltration, but no dilatation or hypertrophy. The myocardium appeared pale. The mitral valves showed a few old patches of granulation. The lungs showed adhesions to parietal pleura, but were otherwise normal.

The abdomen: no ascites or adhesions in the peritoneum; stomach and intestines appeared normal. Spleen: no enlargement or apparent softening. Kidneys: showed some congestion, otherwise normal. Liver: much diminished in size, especially the left lobe, with a wrinkled capsule and a flabby consistence, flattening out upon the table. Weight, thirty
ounces. No subserous hæmorrhages were detected. The cut surface showed a somewhat mottled appearance, mainly red in colour. A culture taken from the spleen was sterile.

An examination of a series of sections of the different pieces of liver has shown the following:

1. The liver capsule is markedly thickened and there is cirrhosis of a diffuse type associated with some thickening of the walls of the vessels.

2. There is a generalized necrosis of the liver cells with no evidence of regeneration in any area examined, and few cells left at the periphery of the lobules. Kupfer’s cells are obvious.

3. There is some inflammation of the larger bile ducts with desquamation of the lining epithelium.

4. The sections stained by the Levaditi method are negative for spirochaete.

5. There are no hæmorrhages, either subcapsular or in the substance of liver tissue.

The well-established cirrhosis of the liver makes the case interesting from an etiological point of view.

Weil’s disease was not suggested either by the clinical picture or by the appearance of the liver.

If there was a preceding acute infection there was no evidence of its nature. Possible causes of the underlying cirrhosis can only be conjectured.

There was no history of syphilis, and the microscopic picture does not suggest the adult type of liver cirrhosis.

The possibility of treatment by arséné-benzine derivations from an unauthorized source was considered. This might have accounted for the cirrhosis by a replacement fibrosis. There is nothing to suggest such a theory and chemical examination of the liver by the Marsh-benzine method proved negative for arsenic.

There was no entry of previous hepatitis in the medical history sheet, and it does not seem to have complicated the amoebic dysentery referred to.

Inquiry elicited the fact that the patient had been a heavy beer drinker, but was not partial to spirits. The post-mortem examination showed no macroscopic evidence of the pathological picture of alcoholic excess.

The cause of the rapid necrosis of the liver cells was not elicited, but all authorities admit that there are still a certain number of such cases in which the cause must remain uncertain; for example, Steihn and Hockett1 record a similar case. It was considered that the rarity of the disease justified its being recorded.

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A CASE OF MYOTONIA ATROPHICA.

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Pte. F., aged 22, service ten months and serving in a Highland Regiment, reported sick at the Regimental Medical Inspection Room, Kowloon, China, on April 4, 1930.

He stated that through no fault of his own he was often "checked" for being slow at his drill. He said that when doing arms drill, for example, his movements were slow, stiff, and awkward at first, but that this stiffness gradually wore off as the drill proceeded and that he was then able to carry on in a normal manner. He had noticed this peculiar stiffness for the past three years.

The man was asked to "shake hands" and the effect was very dramatic, for when he attempted to release his grip the slow relaxation of the flexors of the hand, so characteristic of this disease, was at once apparent and the hand temporarily assumed a claw-like appearance, till relaxation became complete. The time taken for complete relaxation of the grip was about ten seconds. The procedure of shaking hands was then continued, and it was observed that on repetition the relaxation of the grip improved, until finally it took place at a normal rate. He walked with a stiff gait which gradually wore off on continuing the movements. The flexors of the hand appeared to be atrophied, but not to any marked degree. No appreciable atrophy was noticed of the sternomastoids, face, anterior thigh muscles or the flexors of the ankles.

The knee-jerks were normal; abdominal and pupillary reflexes were slow; there was no nystagmus.

There were no signs of disease in any of the viscera.

There was no history of any nervous disease in the family.

The man was sent to hospital on May 19 with a provisional diagnosis of myotonia atrophica.

In hospital it was noticed that he experienced difficulty in relaxing certain muscles, particularly the flexors of the hands and the sternomastoids; the trunk muscles were not affected.

He complained of slight stiffness in the legs, wearing off after continued use, which made him feel unsteady on his legs. There was no pain in any of his muscles and no muscular atrophy could be discerned except in the small muscles of the hand. Electrical reactions were normal. Reflexes and cutaneous sensibility were all normal. The eyes were normal.