Acute Necrodegenerative Hepatitis

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SUMMARY

Seven cases manifesting a strikingly uniform clinical picture of hepatitis are presented. In 4 of the patients a histological study was possible, revealing a lesion of varying severity but similar morphology. The aetiological factors remain obscure, but it is suggested that traditional herbal medications were responsible.

S. Afr. Med. J., **48**, 833 (1974).

CASE REPORTS

Case 1

A 14-year-old Mosotho male was admitted with a history of abdominal swelling for 2 weeks. The distension had come on fairly rapidly, with some pain. Apart from weakness and general tiredness, there were no other significant features. The patient was well nourished, afebrile, anicteric, and mentally clear. There was a tense ascites, which on tapping produced a clear, straw-coloured fluid. There was marked liver enlargement, smooth, and non-tender. The spleen was impalpable. Bilateral pleural effusions were confirmed on X-ray film.

Laboratory investigation showed that the ascitic fluid had a specific gravity of 1,020; protein was found to be 3 g/100 ml; microscopy and bacteriology were normal. Serum bilirubin was 2,5 mg/100 ml, 80% conjugated. Total serum proteins were 5,2 g/100 ml, with an albumin of 2,4 g/100 ml; SGOT 62 units; alkaline phosphatase 21,9 units. Prothrombin index was 77%, which rose to a mere 80% after intramuscular vitamin K; full blood count and erythrocyte sedimentation rate were normal; Mantoux test was negative; and sputum was negative for TB.

During the stay in hospital, the ascites had to be removed several times to relieve discomfort. Each time there was rapid reaccumulation within 2 days in spite of diuretics. Progressively, however, the fluid seemed to rebuild less readily. The patient survived an episode of acute liver failure after the fourth aspiration, after which he fortunately remained static. He was discharged on trial antituberculous treatment, which has made no difference to date, 6 months later. Moderate ascites persists. There is noticeable weight loss, but no jaundice, no anaemia and no gastro-intestinal bleeding.

A liver biopsy done on admission was reported as showing marked necrosis, most pronounced in the central lobule with collapse of reticular tissue, fatty degeneration, areas of regeneration and fibrosis. There was no inflammatory cellular reaction.

The patient admitted to having had a herbal enema for general health a week before his illness.

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Case 2

A 45-year-old Mosotho male presented with a tense ascites and bilateral pleural effusions. He had been ill for 2 weeks with abdominal distension of rapid onset. He was a non-drinker. The abnormal physical findings were confined to ascites and a 3-cm, smooth, non-tender hepatomegaly. His nutritional state was excellent.

Laboratory investigations showed no abnormalities; he had a normal serum albumin and a normal bilirubin. No useful information was obtained from examination of the ascitic fluid. It was surprising that the pleural exudates, like those in case 1, resolved in the face of an ascites that was resistant to diuretics, and never reformed in spite of ascitic reaccumulation. The latter reformed within 48 hours after two aspirations. The patient luckily survived a near-fatal episode of acute hepatic encephalopathy following the second aspiration (a partial aspiration to relieve discomfort). Treatment consisted of the conventional regimen of oral neomycin, protein restriction, vitamin K, high carbohydrate diet, and regulated bowel motions. A liver biopsy showed features of central venous congestion. The patient recovered completely clinically in two months. He is being followed-up. He disclosed that a witchdoctor had given him an enema for backache a few days before he fell ill.

Other Cases

The other 5 cases are summarised in Table I. There was only one female. She is still alive but very ill in hospital, emaciated, with a tense recalcitrant ascites, easily provoked acute hepatic encephalopathy, but preservation of other liver functions.

Case 4 had a haemorrhagic ascites. He died with the pleural effusions having resolved but with persistent ascites. The liver biopsy showed extensive hepatocellular necrosis in centrizonal areas, hyaline in type, not associated with inflammatory cell infiltrate. There was some pigment in phagocytic cells, probably haematoidin. There was marked dilatation of sinusoids in centrizonal areas, suggesting liver damage due to congestion and pressure atrophy of the epithelioid cells rather than actual active cellular necrosis.

The patients who did not survive, or in whom the general condition precluded a proper evaluation and liver biopsy, presented clinical pictures identical with the others. The duration of symptoms was one week in all these patients.

TABLE I. SUMMARY OF CLINICOPATHOLOGICAL FEATURES

Patient	Age	Sex	Physical signs	Laboratory findings	Liver biopsy	Progress	Special features
3	30	F	Ascites, pleural effusions, hepatomegaly	Serum albumin 2 g. Normal liver functions and other tests	Central venous congestion and fatty changes	Emaciation, recurrent liver failure	No jaundice. Herbal enema a week before illness. Fluid reaccumulation.
4	40	M	Ascites, pleural effusions, non-tender hepatomegaly	Normal	Extensive hepatocellular necrosis in centrizonal areas, etc.	Died in hepatic coma	Herbal enema a few days before
5	16	M	Massive ascites, pleural effusions	Not done	Not done	Died shortly after admission in liver failure	No evidence of chronic illness. Herbal enema a week before illness
6	30	M	Massive ascites, pleural effusions	Not done	Not done	Died in hepatic coma	No history of herbal toxin available; no evidence of chronic illness
7	40	M	Massive ascites, pleural effusions	Not done	Not done	Died in hepatic coma	No history of herbal toxin available; no evidence of chronic illness

DISCUSSION

The uniform clinical pattern in these patients is noteworthy. All presented with an acute illness, rapid development of ascites, and bilateral pleural effusions. There was little other evidence of hepatocellular disorder. One case had slight hypo-albuminaemia, and another some biochemical jaundice. The 2 survivors with persistent ascites (cases 1 and 3), retained this feature as the sole sign of liver dysfunction. It would seem, therefore, that the fluid accumulation is dependent on the pathological changes demonstrable in relation to the hepatic central veins. The disappearance of the pleural exudates (bilateral in all, the right larger than the left, if unequal in volume), is unexplained. A concomitant pleural involvement appears unlikely. Other features of the ascites were its resistance to diuretics and rapid reformation.

Severe acute hepatic encephalopathy was a consistent finding, with a fatal outcome in 4 patients. Seen on the whole, especially clinically, there seems to have been classical neurological ammonia intoxication. There was, however, no evidence of portal hypertension.

The antecedent history of herbal use deserves special mention, though the validity thereof may be minimised by the universality of this practice among our patients. The patients came from different areas and the herbs used could unfortunately not be obtained for analysis. Further investigations are warranted. Pyrrolidine alkaloids are incriminated, on circumstantial grounds, in the pathogenesis of veno-occlusive disease.

This condition occurs commonly in malnourished children, as reported from the West Indies and India, but is apparently rare in adults.[1] Some cases have been reported from South Africa. There were 2 children in our series. The striking feature in all the patients was their excellent nutritional state. The special vulnerability of the central zone of the liver in both veno-occlusive disease and the disorder described here is puzzling. It will be interesting to follow the long-term course of the 2 surviving patients particularly as regards the development of cirrhosis.

Viral hepatitis is common in Lesotho. The clinical picture is fairly typical and easily recognised. Other adult hepatopathies readily identified include tuberculosis, primary hepatoma, haemosiderosis, and parasitic and alcohol damage.

REFERENCE

1. Woodruff, A. W. and Bell, S. (1968): *A Synopsis of Infectious and Tropical Diseases*. Bristol: John Wright & Sons.