Hepatic Encephalopathy in a Fattening Heifer

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(Received 23 August 1990/ Accepted 22 November 1990)


KEY WORDS: cattle, cirrhosis, hepatic encephalopathy.

Hepatic encephalopathy, also called hepatocerebral disease, has been defined as encephalopathy resulting from hepatic failure in man [9]. Similar conditions have rarely been reported in cattle [2, 7, 11], but no case has been described in Japan. This paper describes the clinical signs, and laboratory and pathological findings in a heifer that suffered from encephalopathy with hepatic cirrhosis.

On June 9, 1987, a 12-month-old Holstein-Friesian heifer was presented for treatment with a history of anorexia, depression and diarrhea for the past 3 days. She had been introduced for fattening from Miyagi Prefecture 2 weeks ago. She was emaciated, weighing approximately 250 kg, and absence of the menace reaction and pupillary light reflex were suggestive of blindness. The heifer was reluctant to walk, stood vacantly, and had pica, eating feces or glass tubes. Clinical examination revealed an increase in heart rate (100/min) and respiratory rate (30/min), slight decrease in temperature (38.0°C), marked depression, and slight tenesmus with slimy diarrhea. The condition grew worse in spite of medical treatment including intravenous injection of fursultiamine (200 mg/day) and oxytetracycline (2 g/day) in 1000 ml of 5% glucose saline or Ringer's solution. The heifer was killed for necropsy on the 8th day of the illness.

For blood examination, erythrocytes (RBC) and leukocytes (WBC) were counted by using an automatic blood cell counter, and packed cell volume (PCV) was determined with a microhematocrit tube. Biochemical serum analysis included total protein (TP), albumin (Alb), zinc sulfate turbidity test (ZTT), blood urea nitrogen (BUN), glutamic oxalacetic transaminase (GOT), γ-glutamyl transferase (γ-GTP), total bilirubin (T. Bil), direct bilirubin (D. Bil) and bile acids. Table 1 shows the hemogram and biochemical values of the serum. Leukocytosis and neutrophilia suggested an infectious process, while the elevated serum GOT, γ-GTP, T. Bil and bile acids indicated an abnormal liver function. Moderate increase of BUN was also observed.

At necropsy, the liver was moderately atrophic and cirrhotic with scattered millet seed-sized reddish brown nodules on the cut surface. The gallbladder was extensively distended and filled with thick bile (Fig. 1a). In the kidneys, there were many rice grain-sized necrotic foci in the superficial zone of the thin cortex. Hydropericardium with reddish serous fluid, edematous thickening of the abdominal wall and small intestinal mucosa, and swelling of the mesenteric lymph nodes were also observed (Fig. 1b). No macroscopic lesions were noticed in the central nervous system. For histopathological examination, tissues from the whole body were fixed in 10% neutral formalin and sections were stained with hematoxylin and eosin (HE) and Azan.

Microscopically, there was collapse of the lobular structure in the liver with foci of the original lobular tissue surrounded by newly formed fibrous tissue (Fig. 2). This lesion was associated with regeneration of hepatocytes which were irregular in shape and size with large, pale nuclei (Fig. 3). There were lymphangiectasia and pseudobiliary proliferation in the periportal areas. In the gallbladder, mucosal hyperplasia and hypersecretion,
hemorrhage, edema, and infiltration of lymphocytes and plasma cells were noted in the lamina propria. Hyperemia and periarterial infiltration of plasma cells and lymphocytes were found in the spleen. The kidney showed moderate interstitial nephritis with fibrosis, lymphocytic infiltration, moderate fibrous thickening of the Bowman's capsules, and slight glomerular cellularity. These changes were accompanied by intratubular hyaline or cellular casts and hyaline droplet degeneration of the renal tubules (Fig. 4). Focal fibrotic lesions were observed in the right ventricular myocardium.

Vacuolation or spongy degeneration was noted in the corticomedullary zone and corpus striatum of the cerebrum (Fig. 5). In spongiform areas, there were karyorrhectic glial cells and ischemic pyramidal cells accompanying by perivascular and perineural edema. Some vascular walls exhibited hyaline degeneration with enlarged endothelial cells. In the cerebellum, vacuolization was present in the medulla but slighter in the Purkinje cell layer. Marked vacuolization without neuronal changes was observed in the pons and medulla oblongata (Fig. 6).

Toxicologically, lead content of the liver and kidney was less than 0.1 ppm, which was within the normal range. No significant bacteria were isolated.

From the results obtained, the present case was diagnosed as hepatic encephalopathy. Clinical, laboratory and pathological findings of this case were closely similar to those of the cases affected with hepatic encephalopathy, which were caused by ingestion of plants containing hepatotoxin or pyrrolizidine alkaloids [2]. In the epidemiological studies on spongy degeneration in the
central nervous system of 13 cattle by Hooper [7, 9] were caused by ingestion of plants containing hepatotoxins, 2 by congestive heart failure with liver necrosis, and the remaining 2 by an unknown syndrome of hepatic disease.

The hepatic, renal and gastrointestinal lesions of the present case resembled those of pyrrolizidine alkaloid poisoning [1, 2, 5]. The spongy or edematous degeneration of the central nervous system was considered to have been due to ammonia toxicity produced by hepatic failure [2, 6, 7, 11]. The generalized circulatory disturbance observed in the present case seemed to be related to hepatic cirrhosis, interstitial nephritis and focal cardiac fibrosis possibly produced by the toxic effects of plants, although the causative factors were not determined.

The reason for development of acute signs of hepatic failure and encephalopathy after the heifer had been introduced into the present farm was not known. An alteration in feed could be a factor, because she was fed on an abundant fattening ration (TDN, 6.0 kg; DCP, 0.85 kg). As a result of an increase in protein intake, blood ammonia which has been thought to be important in the pathogenesis of hepatic encephalopathy [6, 7, 9–11], might be elevated. In recent studies in man, however, blood amino acid imbalance, e.g. reduction of branched chain amino acids and an inverse increase of aromatic amino acids, caused by hepatic failure has been considered to be the main cause of hepatic encephalopathy [3, 4]. Accordingly, it would be indispensable to examine blood levels of ammonia and amino acids in additional cases.

ACKNOWLEDGMENTS. We express our thanks to Dr. T. Yoshikawa, School of Veterinary Medicine and Animal Sciences, Kitasato University, for his valuable suggestion.

REFERENCES