NOTE Internal Medicine

Focal Fatty Liver in a Heifer: Utility of Ultrasonography in Diagnosis

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ABSTRACT. A 30-month-old Holstein heifer presented with a history of decreased appetite and respiratory signs. Sonographic examination of the liver incidentally revealed an area of increased echogenicity between the portal vein and the gallbladder. The lesion was non-spherical and had no mass effect or displacement of the adjacent vessels. Its boundaries, to the liver, were geographic. The liver specimen was histologically compatible with a diagnosis of focal fatty liver change (FFLC). The sonographic features of focal fatty infiltration of the liver are characteristic. Recognition of the ultrasonographic data is important to differentiate FFLC from other lesions.

KEY WORDS: bovine, focal fatty liver change, ultrasonography.

In dairy cows, diagnosis of fatty infiltration of the liver has been based mostly on history, clinical signs and biochemical analyses. Recently, ultrasonography (US) has been used in the evaluation of fatty infiltration in dairy cows [1, 2], and the findings obtained agreed with the results of histologic examination of liver biopsy specimens. Focal fatty liver change (FFLC) is a pathological condition that has been recognized with increasing frequency, with the widespread use of US [8]. The cause of FFLC has not been clarified, although some authors have speculated that the regional disturbance of intrahepatic portal blood flow may be an important factor [3, 7]. Hepatic US in humans with FFLC demonstrates the presence of focal hyperechogenicity in a relatively non-echogenic organ [9, 12]. These focal areas, which may vary widely in size, shape and border characteristics, are generally observed in the periporal area, near the gallbladder [11]. A search of the veterinary literature disclosed little information in cattle [6]. In this note, we present a heifer demonstrating unusual infiltration of fat into the liver. The ultrasonographic, laboratory and clinicopathologic findings of the case are documented.

A 30-month-old Holstein heifer presented at the Veterinary Teaching Hospital of Rakuno Gakuen University for investigation of decreased appetite and respiratory signs. Cattle in the same herd had experienced some periparturient diseases such as mastitis, ketosis and fatty liver. On the first examination, the animal was thin, had only a moderate appetite, 38°C rectal temperature, 80 beats/min pulse rate and 16 breaths/min respiratory rate. Rumen movements were decreased. Auscultation of the thorax revealed hypervesicular sounds especially on the carinoventral lung lobes. Blood gas analysis was consistent with moderate hypoxaemia. Arterial blood gas results were PaO2 62.9 mmHg, PaCO2 49.8 mmHg and pH 7.47. ECG recordings did not reveal any abnormal findings. Higher activity of aspartate aminotransferase (319 IU/l) and high concentrations of total bilirubin (0.90 mg/dl) indicated a moderate degree of hepatic impairment. The thorax and the abdomen were scanned from dorsal to ventral, by means of an ultrasonic scanner equipped with a 3.5 MHz linear transducer (Model RT 2600, Yokogawa Medical Systems, Tokyo, Japan). Ultrasonic coupling gel was liberally applied to the wet skin to ensure good contact, and the transducer was held firmly against the body wall.

Compared to the hepatic parenchyma in a normal cow (Fig. 1A), the liver in the present case incidentally disclosed an area of increased echogenicity on the medial surface of the liver (Fig. 1B). The area was located between the portal vein and the gallbladder. Four distinctive features of the lesion were found on the sonograms. First, non-spherical; second, no bulge of the liver contour; third, geographic boundaries; and fourth, no mass effect or displacement of the adjacent vessels. On ultrasonographic examination, we diagnosed FFLC in the heifer associated with bilateral bronchopneumonia. Because of poor prognosis, the cow was donated to our clinic and then euthanatized. Postmortem examination revealed a slightly enlarged liver with an area of FFLC that appeared both on the capsule and on the cut surface (Fig. 2A). The lesion of the liver was located in the right lobe, extending between the portal vein and the gallbladder. Portal vessels were detected within the lesion with no evidence of venous outlet obstruction.

The triglyceride content was 123.4 mg/g of liver in the lesion compared with 40.7 mg/g in the surrounding parenchyma [10]. A liver specimen was fixed in buffered 10% formalin, embedded in paraffin and stained with hematoxylin and eosin. Histologic examination showed marked focal fatty infiltration of the hepatocytes with strong evidence of parenchymal necrosis. Hepatic cells within the focal lesion were isolated or organized in sheets. Compared to the adja-
cent normal liver parenchyma, cells within the lesion had large and clear intracytoplasmic vacuoles. These vacuoles displaced the nuclei to the periphery of the cells flattening them against the cytoplasmic membrane and giving these cells a signet-ring appearance. The steatotic area was considered to be consistent with the echogenic area on sonography. Normal hepatocytes were dispersed in the isoechogenic hepatic parenchyma (Fig. 2B). No embolism was evidenced by the observation of successive sections. Frozen sections of liver stained with Sudan III disclosed a multiplicity of red intracellular globules of fat that occupied almost all the cytoplasm (Fig. 2C). The histologic picture of the liver specimen was compatible with a diagnosis of FFLC.

In the normal cow, the hepatic ultrasonogram consists of numerous weak echoes distributed homogeneously over the entire area of the liver. The echo beam gradually attenuates as it passes through the normal liver tissue. The portal and hepatic veins can be seen within the normal echotexture, and the parenchymal edges are normally visible [5] (Fig. 1A). Pathologically, in the cow, fatty change in the liver is generally considered a diffuse process involving the entire organ [6]. Its diffuse nature and echogenicity are roughly proportional to the volume of fat vacuoles and the amount of triglyceride in livers proven histologically to contain fatty infiltration [6]. These findings are in keeping with the fact that the echogenicity of a structure depends on the number of solid-liquid interfaces contained. In cattle, diffuse fatty infiltration of the liver is easily recognized and well-established pathologically and ultrasonographically, but the presence, significance and differential diagnosis of FFLC, as far as we know, has had only a marginal description in a single report [6].

The present case was discovered incidentally during routine sonographic examination of the liver; and differential diagnosis included true hepatic tumor, such as hepatocellular and cholangiocellular adenomas and carcinomas. The ultrasonogram revealed portal blood flow within the lesion, attesting to the fact that it was not a true hepatic tumor but fatty infiltration of the liver [8]. Focal hepatic lesions, including slowly growing metastatic lesions, can have echogenicity values in a similar range [6]. Metastases in liver usually have spherical configuration [4]. Some metastatic lesions have the same echogenicity as liver but are misinterpreted as bulges in the contour of the liver [6]. The lesion reported here was nonspherical and had geographic

Fig. 1. A. Normal hepatic echotexture in a cow. The portal vein (PV) is characterized by a hyperechogenic wall and the hepatic vein (HV) by a hypoechoic one. B. The hepatic sonogram in the present case shows a hyperechoic area located anterior to the portal vein. L: liver parenchyma.
margins. Its location was between the portal vein and the gallbladder. Moreover, there was no mass effect and no displacement with the normal vascular branching pattern and bile ducts. The absence of a mass effect was extremely helpful in differentiating FFLC from other space-occupying lesions.

The causes of FFLC are still unclear. In human medicine, some authors [3, 7] have proposed that regional differences in blood flow might be an important factor in explaining the focal nature of the process, but it is also possible that these differences are a result, rather than a cause, of fat deposits. In the present case, the lesion did not appear to be ischemic, because the supplying vessels detected at necropsy were similar in size to those in normal parts, and there was no evidence of venous outlet obstruction. Moreover, no embolism was present as shown by examination of successive sections.

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