Three Cases of Feline Sclerosing Lymphocytic Cholangitis

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Feline cholangitis has been reported in a few cases [7, 10] and is considered to be one of the causes of feline icterus [6, 7, 10]. This condition is histopathologically characterized by lymphoid infiltration, fibrosis and biliary hyperplasia. The disease has been noticed because of the similarity of clinical and pathological features to human primary biliary cirrhosis. This report describes three cases of feline sclerosing lymphocytic cholangitis.

Clinical and gross pathological findings of these cases are summarized in Table 1. Case A was a 5-year-old female Persian cat that had recurrent icterus for more than a half year. Weight loss and anemia were also noted. Abdominal palpation was suggestive of hepatomegaly and there were increased levels of serum bilirubin, alkaline phosphatase (ALP), glutamate-oxalate transaminase (GOT) and glutamate-pyruvate transaminase (GPT). Serologically, the cat was verified to be free from feline leukemia virus (FeLV) and feline infectious peritonitis virus (FIPV). At autopsy, the subcutis was yellowish in color. The liver was firm and swollen (Fig. 1), and there was no obstruction of the extrahepatic bile duct. Kidneys were firm, swollen and yellowish brown in color.

Case B was a 7-year-old castrated male Himalayan cat. He had a sudden onset of depression and exhibited pain in the hind limbs which were associated with edema and phlegmonous change. At autopsy, the generalized subcutis was yellowish and there was a diffuse severe supplicative inflammation in the subcutis of the lumber region as well as the hind limbs. The liver was firm and fibrotic with distinct lobular appearance. Some hyperplastic nodules, 0.5 to 3 cm in diameter, were seen on the cut surfaces. There was fibrous thickening of the wall of the gallbladder. The extrahepatic bile duct was not occluded.

Case C was a female Japanese domestic cat of unknown age. The cat had a supplicative lesion in the face and a sign of anemia due to erythroblastic hypoplasia. A liver biopsy was obtained and thereafter she died. Autopsy was not performed.

Histopathological features in the livers were characterized by lymphocyte aggregation, fibrosis and bile duct proliferation. In case A, increased fibrous tissue encircled the lobules, forming a network-like pattern (Fig. 2). Fine collagenous fibers extended into the parenchyma along sinusoids. Mild to moderate lymphocyte aggregation was observed in some portal areas, especially around bile ducts (Fig. 3). Bile pigment deposition was noted in both hepatocytes and Kupffer cells. Bile plaques were seen in some bile canaliculi. The nature of the liver histopathologicaly in case B was almost the same as that in case A. However, lymphocyte infiltration was more diffuse and severer, and proliferation of bile ducts was milder (Fig. 4). The former change, associated with lymphoid follicles (Fig. 5), was located in the thickened wall of the gallbladder and around the larger intrahepatic bile ducts. Bile pigments were present in hepatocytes and Kupffer cells. Case C showed the severest lymphocyte infiltration among the three cases, which resulted in destruction of bile ducts in the portal area (Fig. 6). Fibrosis and proliferation of bile ducts were milder than the other two cases.

In the pancreas, lymphocyte aggregation with lymphoid follicles was observed around some large-sized intrapancreatic ductules in case B (Fig. 7), and very slight periductulitis was seen in case A. There was a small aggregation of lymphocytes in the interstitium of thyroids in cases A and B. No available information was obtained from the pancreas and thyroid gland of case C.

Two thoughts have been reported on the feline cholangitis. One is characterized by neutrophil infiltration in and around bile ducts with consequent fibrosis and bile duct hyperplasia [2-4, 8]. It was thought to be a secondary disease initiated by inflammation or bile duct occlusion and was often referred to as “ascending cholangitis”. Prasse et al. [10] and Lucke and Davies [7] reported another type in several cases, in which the lesions are characterized by lymphocyte infiltration, fibrosis and bile duct hyperplasia. They called the lesions ‘chronic or progressive lymphocytic cholangitis’. The present cases may be included in the latter disease entity. However, we use the name, ‘sclerosing lymphocytic cholangitis’, in this report, because not only lymphocyte infiltration but also sclerotic change were the major characteristic features in the present three cases. The differences in histopathology among the present cases may be due to the different stages of the disease. Although the etiology of this disease is still unknown, the lesion may be initiated by lymphocyte infiltration around intrahepatic bile ducts and ductular destruction. Consequently proliferation of fibrous tissues with new formation of bile ducts may occur. The fibrous tissue tends to encircle each lobule and partly extends into hepatic parenchyma, resulting in hepatic cirrhosis.

It has reported that pancreatic involvement coexisted in some cases [3, 10]. Lucke and Davies [7] stated that this could be due to concomitant ascending infection and had been often described in cases of feline ascending cholangitis. In our cases, the inflammation was limited to the periductular areas without involvement of the epithelial layer.
Fig. 1. Cut surface of the liver from case A. The liver is hard and hepatic lobules are distinct due to proliferating connective tissue.

Fig. 2. Liver of case A. Proliferating connective tissue encircle the lobule, forming a network-like pattern. Lymphocyte aggregation is also seen (arrowheads). HE. × 140.

Fig. 3. Liver of case A. Fibrosis and bile duct hyperplasia are distinct. Lymphocyte aggregation is also seen. HE. × 120.

Fig. 4. Liver of case B. Lymphocyte infiltration is more marked and diffuse than in case A. Fibrosis and bile duct proliferation are also observed. HE. × 120.

Table 1. Clinical and gross pathological findings in three cases of feline cholangitis

<table>
<thead>
<tr>
<th>Case</th>
<th>Breed</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Clinical findings</th>
<th>Gross findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>Persian</td>
<td>F</td>
<td>5</td>
<td>Weight loss, Anemia, Icterus, Increased ALP, GOT and GPT</td>
<td>Hepatic fibrosis, Nephritis</td>
</tr>
<tr>
<td>B</td>
<td>Himalayan</td>
<td>CM</td>
<td>7</td>
<td>Icterus, Edema and suppurative lesion in the hind limb</td>
<td>Suppurative inflammation in the hind limb, Hepatic fibrosis, Nephritis, Cardiac degeneration</td>
</tr>
<tr>
<td>C</td>
<td>JDC&lt;sup&gt;a&lt;/sup&gt;</td>
<td>F</td>
<td>?&lt;sup&gt;c&lt;/sup&gt;</td>
<td>Suppurative lesion in the face, Anemia</td>
<td>Hepatic change (Biopsy case)</td>
</tr>
</tbody>
</table>

<sup>a</sup> F: Female, CM: Castrated male. <sup>b</sup> Japanese domestic cat. <sup>c</sup> Unknown.
The clinical and pathological features of the feline cholangitis including the present cases are very similar to those of human primary biliary cirrhosis [1, 5]. In human, the middle-aged (40–50 years old) population is mainly affected, and females are much more susceptible than males (male: female = 1:10). In the feline cases, however, it appears that young- to middle-aged (3–9 years old) animals are susceptible to this condition without any sex predilection. Human primary biliary cirrhosis may occur through an immunological mechanism, as suggested by the presence of autoantibodies like anti-mitochondrial antibody in patient sera and by other immunological abnormalities concurrent with the disease [5, 9]. In a feline case [7], weak positive antinuclear factor was found but the anti-mitochondrial antibody was not detected. The formation of lymphoid follicles in the liver and the lymphocytic nature of thyroiditis and pancreatic periductulitis in the present cases suggest an immunological mechanism for this disease.

REFERENCES