Canine Splenic Hemangiosarcoma with Abdominal Dissemination

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ABSTRACT. Disseminated hemangiosarcoma was encountered in a 12-year-old female Maltese dog. Tumor tissues were dispersed on the serosal surface of the liver, kidney, digestive tract, omentum and diaphragm. Metastatic lesions were not observed in the parenchyma of the lung and heart. The spleen was enlarged with rupture at the anterior region of the hilus. The disseminated protruding tumor masses could be easily peeled off from the organ surfaces. The tumor cells were round or spindle in shape, with hyperchromatic nuclei containing prominent nucleoli. Various-sized vascular spaces containing erythrocytes and serum could be identified in this tumor. This case was diagnosed as hemangiosarcoma originating from the spleen with abdominal dissemination.—KEY WORDS: canine, hemangiosarcoma, spleen.

Hemangiosarcoma (HSA) occurs frequently in dogs originating from the spleen and right atrium [2–5]. The lung [2, 6], liver, omentum and mesentery [9, 10] are the common organs with the metastasis. There are a few reports of disseminated pattern of splenic HSA in detail after tumor rupture in dogs [7, 10], although it is one of the most common metastatic pattern in human beings [1, 8]. In most dog cases there is metastasis from the original site through blood vessels or lymphatics [6]. This report describes a case of splenic HSA showing implantation on the serosa of the gut, stomach, liver, kidney, diaphragm and omentum.

The case in question was a 12-year-old, female, Maltese dog. The spleen and omentum were removed by surgery at the veterinary hospital attached to Miyazaki University and she died within 38 days after the surgery. Her body weight was 4.7 kg before and 3.8 kg after surgery. Ranges of the major clinical parameters were as follows: temperature 38.2–39.4°C; WBC 8.8 × 10⁹ – 73.5 × 10⁹/μl (neutrophils 54–80%, lymphocytes 5–15%, eosinophils 1–4% and monocytes 17–25%); RBC 1.75 × 10¹² – 3.69 × 10¹²/μl; Hgb 4.5–9.3 g/dl; PCV 13.2–26.0%; MCH 22.5–31.7 pg; MCHC 30.0–38.1 g/dl; PLT 2.8 × 10⁹ – 16.3 × 10⁹/μl; glucose 34–96 mg/dl; T-Chol 248–262 mg/dl; BUN 9–24 mg/dl; T-Bil 0.3–0.4 mg/dl; GOT 47–82 IU/l; GPT 18–138 IU/l; creatinine 0.7–1.1 mg/dl. The dog was necropsied immediately after death and tissues were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 4 μm, and stained with hematoxylin and eosin (HE). Selected sections of the tumor tissue were stained with Masson’s trichrome method.

The spleen was markedly enlarged, measuring about 10 times larger than normal (Fig. 1). The tumor in the anterior region of the splenic hilus was soft, reddish-gray in color and ruptured. On cut surface, the growths consisted of various-sized cysts and areas of necrosis and hemorrhage with much reddish-blue exudate. Tumor masses, ranging from a pin head to about 5 cm in size, were disseminated over the surface of the omentum (Fig. 1). The tumor tissue was soft in consistency and reddish-blue in color.

At necropsy about 500 ml of blood-stained, putrid ascitic fluid containing much fibrous material was found in the abdominal cavity. In the liver, tumor masses were limited to the serosal surface. These tumor masses, similar in size to those in the omentum, were seen protruding from the serosal surface of the liver with apparent demarcation from the normal liver tissue and could be easily peeled off from the serosa (Fig. 2). They were solid in consistency and reddish-yellow in color, with oozing of small amounts of blood fluid on cut surface. Many similar tumor masses were also seen on the serosa of the abdominal face of the diaphragm, digestive tract (Fig. 2) and kidney without deep tissues invasion.

Histopathologically, the spleen was replaced by tumor tissues. These tissues were composed of various-sized vascular channels or spaces (Figs. 3a and 3b) and some of which contained erythrocytes and serum were sometimes enlarged to form cysts. The channels were lined by round or spindle-shaped neoplastic endothelial cells with hyperchromatic nuclei and abundant cytoplasm in some places. In other sites the tumor tissue was solid, without any vascular channels, and the cells were larger, round or

Fig. 1. Spleen and omentum (after 10% formalin fixation). The spleen was cut (small arrowhead), and there is large spleen head (arrow) and ruptured regions (large arrowheads). Numerous disseminated masses of various sizes on the omentum. Bar = 4 cm.

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The same histopathologic features as those in the spleen, although the growths were limited to the serosal surfaces of the respective organ being demarcated by fibrous tissue. There were no tumor lesions in any of the other organs that were examined including the lung and heart.

Based on the gross and microscopic findings, we conclude that this is a case of HSA originating from the spleen with subsequent rupture and implantation on the peritoneum. HSA can metastasize to other tissues from the original site through blood vessels, especially to the lung [2] or liver [9]. We consider that tumor cells can also be easily implanted to adjacent tissues. Tumor cells were directly implanted onto the serosal surface of the organs in the abdominal cavity from the ruptured spleen in this case. Abnormal blood vessels are formed in HSA tissue and these newly formed blood vessels might easily rupture, so that implantative metastasis can occur more frequently than other malignant tumors. It has been reported that rupture of the spleen occurs in about 34% of cases of splenic HSA in humans [1]. Because of the different pattern of metastasis in this case, there were no tumor lesions in the heart and lung. However, tumor cells were found in splenic veins and lymphatics. The early splenectomy might have minimized the possibility of tumor cell metastasis through the blood vessels or lymphatics. Clinical diagnosis of HSA may be difficult, because it is easily confused with other tumors. In our laboratory the incidence of HSA is much less than others.

REFERENCES