

Acta Scientiae Veterinariae. 38(4): 433-437, 2010.

CASE REPORT Pub. 936 ISSN 1679-9216 (Online)

Hemoperitoneum in a Dog with Hepatic Splenosis

Saulo Petinatti Pavarini¹, Eduardo Conceição Oliveira¹, Adriana da Silva Santos¹, Luciana Sonne¹, Djeison Lutier Raymundo¹, Gregory Duarte Juffo¹, Pedro Soares Bezerra Junior² & David Driemeier¹

ABSTRACT

Background: Ectopic splenic tissue results from the autotransplantation and seeding of splenic tissue, often secondary to splenic trauma or splenectomy. Splenic implantations occur mostly as nodules within the peritoneal cavity and constitute an incidental finding at necropsy, surgery, or imaging investigations. This report addresses a case of hemoperitoneum associated with hepatic splenosis in a dog that became ill several years after splenic trauma.

Case: A 9-year-old castrated female Dachshund was presented to the veterinary assistance due to an acute onset of abdominal pain and distension. After recovering from a severe trauma caused by a car accident about five years previously, the dog remained apparently normal. Upon admission the dog also showed anorexia, oliguria (dark yellow urine), increased pulse rate, tachypnea, and hypothermy (37.1°C). Palpation revealed abdominal distension attributed to large amounts of peritoneal fluid and a large multinodular mass detected in the left side of the abdomen. While white cell and platelet counts were within the normal range, hematocrit (26 to 24%) and erythrocyte counts (3.08 to 2.84 x 10⁶/mL), and hemoglobin content (8.1 to 7.8 g/ dL) decreased as indicated within 36 h after admission. Plasmatic levels of alanine transaminase (ALT-12.1U/L) and creatinine (1.04 mg/dL) were consistent with normal canine patterns. Abdominocentesis revealed a non-clotting and reddish effusion with density of 1.032 and numerous nucleated cells (neutrophils 61%, lymphocytes 24%, and macrophages 15%). Erythrophagocytosis, crystals of hematoidine, and activated macrophages were also observed. Necropsy revealed extensive hemoperitoneum (360 mL), lobular pattern accentuation and a soft dark red 15-cm multinodular mass adhered to the left hepatic lobe. The insertion of the nodular mass in the hepatic tissue consisted mainly of sanguineous fluid-filled cystic structures, some of which were disrupted. There were also multiple, firm, dark red nodules (0.3-2.8 cm) scattered through the liver parenchyma, and some smaller (0.3-0.5 cm) nodules attached to the omental and diaphragmatic surfaces. The abnormally shaped spleen was divided in two similar halves by a soft red nodule about 2 cm in diameter. Main microscopic hepatic changes were nodules formed by lymphocyte aggregates surrounded by fibrous connective tissue. Occasionally these nodules formed follicular structures surrounded arterioles and intermixed with delicate vascular formations, which were filled with erythrocytes and lymphocytes. Anti-CD3 immunostaining showed positive reactions in lymphocytes within the intrahepatic ectopic splenic tissue, especially in lymphocyte aggregates that surrounded arterioles.

Discussion: Some of the necropsy and microscopic findings resembled those previously reported in two dogs affected by hepatic splenosis. It has been suggested that intrahepatic splenosis may trigger death through hepatic insufficiency. However, in the present case, the dog had neither altered ALT activity nor suggestive changes in the reminiscent hepatic tissue that could indicate hepatic insufficiency. The association of a decreased hematocrit, depressed concentration of hemoglobin, and large volume of peritoneal effusion of probable hemorrhagic origin with the clinical signs seen here and the cyst rupture indicates that the severity of this case may probably be linked to hypovolemic shock.

Keywords: hepatic, splenosis, hemoperitoneum, dog.

Received: March 2010

www.ufrgs.br/actavet

Accepted: June 2010

¹Setor de Patologia Veterinária (SPV), Departamento de Patologia Clínica Veterinária, Faculdade de Veterinária (FaVet), Universidade Federal do Rio Grande do Sul (UFRGS), Av. Bento Gonçalves n. 9090, CEP 91540-000 Porto Alegre, Bairro Agronomia, RS, Brazil. ²Departamento de Medicina Veterinária, Universidade Federal de Lavras, MG, Brazil. CORRESPONDENCE: D. Driemeier [davetpat@ufrgs.br - FAX: +55 (51) 3308-7305].

INTRODUCTION

Hepatic splenosis has been sporadically reported in humans [3,4,7] and animals [8,10,12]. splenic tissue results Ectopic from the autotransplantation and seeding of splenic tissue, often secondary to splenic trauma or splenectomy [2,5,4,14]. Splenic implantations occur mostly as nodules within the peritoneal cavity and constitute an incidental finding at necropsy, surgery, or imaging investigations [3,14]. Most human cases of intrahepatic splenosis had no clinical significance [5,7]. Although severe gross changes have been described in two dogs suffering from splenosis, limited information on clinical features associated with these cases is available [8,10]. This report addresses a case of hemoperitoneum associated with hepatic splenosis in a dog that became ill several years after splenic trauma.

CASE REPORT

A 9-year-old castrated female Dachshund was presented to the veterinary assistance due to an acute onset of abdominal pain and distension. After recovering from a severe trauma caused by a car accident about five years previously, the dog remained apparently normal. According to the owner, two weeks prior to admission the dog presented abdominal distension. The owner also informed proper care, including vaccination and anthelmintic administration. Upon admission the dog also showed anorexia, oliguria (dark yellow urine), increased pulse rate, tachypnea, and hypothermy (37.1°C). Palpation revealed abdominal distension attributed to large amounts of peritoneal fluid and a large multinodular mass detected in the left side of the abdomen. The dog showed spontaneous pain reaction in response to pressure exerted during abdominal palpation. Ocular, oral, and anal mucosae were pale. Radiographic examination showed that the mass was connected with the liver, and a presumptive diagnosis of hepatic neoplasia was established. Samples of blood, intraperitoneal fluid, and feces were collected. Initial diuretic therapy showed low but transitory improvement of general condition.

While white cell and platelet counts were within the normal range, hematocrit (26 to 24%) and

erythrocyte counts (3.08 to 2.84 x 10⁶/mL), and hemoglobin content (8.1 to 7.8 g/dL) decreased as indicated within 36 h after admission. Plasmatic levels of alanine transaminase (ALT-12.1U/L) and creatinine (1.04 mg/dL) were consistent with normal canine patterns. Abdominocentesis revealed a non-clotting and reddish effusion with density of 1.032 and numerous nucleated cells (neutrophils 61%, lymphocytes 24%, and macrophages 15%). Erythrophagocytosis, crystals of hematoidine, and activated macrophages were also observed. Parasitological exams were negative. Due to the severity of the case and continuous deteriorating conditions, the owner decided for the euthanasia of the dog.

Necropsy revealed extensive hemoperitoneum (360 mL) (Figure 1), lobular pattern accentuation and a soft dark red 15-cm multinodular mass adhered to the left hepatic lobe (Figure 2). The insertion of the nodular mass in the hepatic tissue consisted mainly of sanguineous fluid-filled cystic structures, some of which were disrupted (Figure 3). There were also multiple, firm, dark red nodules (0.3-2.8 cm) scattered through the liver parenchyma, and some nodules (0.3-2 cm) attached to the omental and diaphragmatic surfaces. The abnormally shaped spleen was divided in two similar halves by a soft red nodule about 2 cm in diameter (Figure 4). Fragments from numerous organs were collected, fixed in 10% buffered formalin, processed by routine histologic methods, and stained with hematoxylin and eosin. Samples of altered liver were stained by the Trichrome Masson-Goldner method and by anti-CD3 immunostaining (IS). IS was performed through streptavidin-biotin-alkaline phosphatase method with a monoclonal antibody¹, which was diluted at 1/500 in Phosphate Buffered Saline (PBS). Permanent red¹ was used as chromogen.

Main microscopic hepatic changes were nodules formed by lymphocyte aggregates surrounded by fibrous connective tissue. Occasionally these nodules formed follicular structures surrounded arterioles and intermixed with delicate vascular formations, which were filled with erythrocytes and lymphocytes (Figure 5). There were also numerous cystic structures overlaid by a flat epithelium and filled with a few erythrocytes, extramedullary hematopoiesis, and moderate hemosiderosis. Mild Pavarini S.P., Oliveira E.C., Santos A.S., Sonne L., Raymundo D.L., Juffo G.D., Bezerra Junior P.S. & Driemeier D. 2010.Hemoperitoneum in a Dog with Hepatic Splenosis.Acta Scientiae Veterinariae. 38(4): 433-437.



Figure 1. Abdominal cavity with large amount of reddish fluid (hemoperitoneum).



Figure 2. Abdominal cavity of a dog with a dark red multinodular mass (arrows) attached to the left hepatic lobe. Ectopic splenic foci (arrowheads) attached to the liver and omentum.

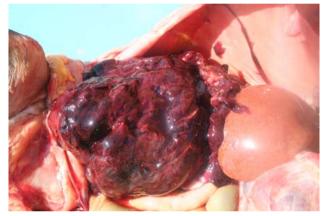


Figure 3. Nodular red mass in the hepatic tissue constituted mainly of sanguineous fluid-filled cystic structures, some of which were disrupted.



Figura 4. Spleen divided in two similar halves by a red nodule and multiple red nodules attached to the omental.

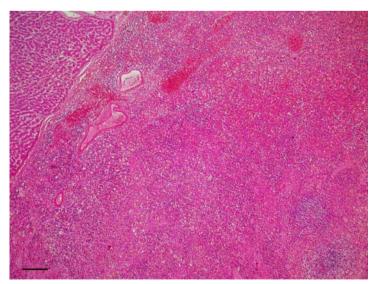


Figure 5. Hepatic splenosis: hepatic tissue bearing ectopic splenic tissue surrounded by fibrous connective tissue. Hematoxylin and eosin (Bar = $150 \,\mu$ m).

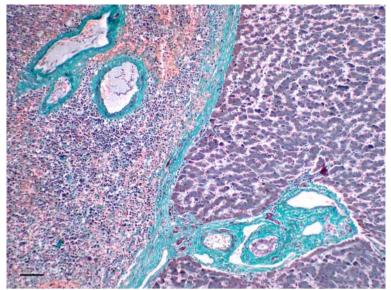


Figure 6. Hepatic splenosis: connective tissue bordering the intrahepatic ectopic splenic foci, arteriole, and portal triad evidenced by the Trichrome Masson-Goldner staining (Bar = $70 \,\mu$ m).

centrilobular canalicular cholestasis and fibroblastic proliferation were also observed in the borders of the lesions. Anti-CD3 IS showed positive reactions in lymphocytes within the intrahepatic ectopic splenic tissue, especially in lymphocyte aggregates that surrounded arterioles. Connective tissues which surrounded lesions were evidenced by Trichrome Masson-Goldner staining (Figure 6). Spleen showed mild lymphoid hyperplasia, retraction of the red pulp, fibrosis, hemosiderosis, and extramedullary hematopoiesis.

DISCUSSION

It has been suggested that, after traumatic rupture of the spleen, splenic cells may reach the hepatic parenchyma by embolic route as follow, from the splenic vein to the gastrosplenic vein and then, to the liver by the portal vein [5,8,11]. The highest concentration of splenosis in the left hepatic lobe, close to the entry of blood from the spleen supports the hypothesis of an embolic route [5,8]. Alternative hypothesis indicates that erythrocytic progenitor cells may reach the liver by portal vein and develop in response to the tissue hypoxia [9].

Some of the necropsy and microscopic findings resembled those previously reported in two dogs affected by hepatic splenosis [8,10]. The anti-CD3 immunostaining pattern observed in

lymphocytes, especially those surrounding arterioles is a characteristic of T lymphocytes from splenic tissue [13]. It has been suggested that intrahepatic splenosis may trigger death through hepatic insufficiency [8]. However, in the present case, the dog had neither altered ALT activity nor suggestive changes in the reminiscent hepatic tissue that could indicate hepatic insufficiency.

The cystic dilatation of lymphatic vessels and billiary ducts that may result from the compression exerted by the autotransplanted splenic tissue on the adjacent hepatic tissue has been linked to cyst rupture and abdominal pain [8], both of which were reported here. The association of a decreased hematocrit, depressed concentration of hemoglobin, and large volume of peritoneal effusion of probable hemorrhagic origin with the clinical signs seen here and the cyst rupture indicates that the severity of this case may probably be linked to hypovolemic shock [1]. Hemoperitoneum in the dog has strongly been associated with splenic hemagiossarcoma, a condition linked to decreased total solids concentrations and platelet counts [6], but also a probable differential diagnosis of hepatic splenosis.

SOURCES AND MANUFACTURERS ¹DAKO, Carpentaria, California, USA

REFERENCES

- 1 Alleman A.R. 2003. Abdominal, thoracic, and pericardial effusions. *The Veterinary clinics of North America Small animal practice*. 33(1): 89-118.
- 2 Carr N.J. & Turk E.P. 1992. The histological features of splenosis. Histopathology. 21(6): 549-553.
- 3 Davidson L.A. & Reid I.N. 1997. Intrahepatic splenic tissue. Journal Clinical Pathology. 50(6): 532-533.
- 4 De Vuysere S., Van Steenbergen W., Aerts R., Van Hauwaret H., Van Beckevoort D. & Van Hoe L. 2000. Intrahepatic splenosis: imaging features. *Abdominal Imaging*. 25(2): 187-189.
- 5 Fremont R.D. & Rice T.W. 2007. Splenosis: a review. Southern Medical Journal. 10(6): 589-593.
- 6 Hammond T.N. & Pesillo-Crosby S.A. 2008. Prevalence of hemangiosarcoma in anemic dogs with a splenic mass and hemoperitoneum requiring a transfusion: 71 cases (2003-2005). *Journal of the American Veterinary Medical Association*. 232(4): 553-558.
- 7 Imbriaco M., Camera L., Manciuria A. & Salvatore M. 2008. A case of multiple intra-abdominal splenosis with computed tomography and magnetic resonance imaging correlative findings. *World Journal Gastroenterology*. 14(9):1453-1455.
- 8 Knostman K.A.B., Weisbrode S.E., Marrie P.A. & Worman J.L. 2003. Intrahepatic splenosis in a dog. Veterinary Pathology. 40(6): 708-710.
- 9 Kwok C.M., Chen Y.T, Lin H.T., Su C.H. & Chiu Y.C. 2006. Portal vein entrance of splenic erythrocytic progenitor cells and local hypoxia of liver, two events cause intrahepatic splenosis. *Medical Hypotheses*. 67(6): 1330-1332.
- 10 Patnaik A.K., Lieberman P.H. & Macewen G. 1985. Splenosis in a dog. Journal of Small Animal practice. 26(1): 23-31.
- 11 Rickert C.H., Maasjothusmann U., Probst-Cousin S., August C. & Gullotta F. 1998. A unique case of cerebral spleen. American Journal of Surgical Pathology. 27(7): 894-896.
- **12 Tanimoto T. & Ohtsuki Y. 1993**. Heterotopic splenic tissue in the liver of a swine. *Journal of Veterinary Medical Science*. 55(3): 485-486.
- 13 Valli V.E. 2007. Vetrinary Comparative Hematopathology. Iowa: Blackweel publishing, 558p.
- 14 Vento J.A., Peng F., Spencer R.P. & Ramsey W.H. 1999. Massive and widely distributed splenosis. Clinical Nuclear Medicine. 24(11): 845-846.

