Scientific Report

A mediastinal mass causing Budd-Chiari-like syndrome in a cat

Suárez-Cabrera, F.¹; Encinoso, M.²; Artiles, A.³; Castellano, I.¹; Melián, C.^{2*} and Jaber, J. R.⁴

¹Ph.D. Student in Anatomy, Department of Morphology, Faculty of Veterinary Medicine, University of Las Palmas de Gran Canaria, Las Palmas de Gran Canaria, Spain; ²Veterinary Teaching Hospital, Faculty of Veterinary Medicine, University of Las Palmas de Gran Canaria, Las Palmas de Gran Canaria, Spain; ³Los Tarahales Veterinary Hospital, Las Palmas de Gran Canaria, Spain; ⁴Department of Morphology, Faculty of Veterinary Medicine, University of Las Palmas de Gran Canaria, Las Palmas de Gran Canaria, Spain;

*Correspondence: C. Melian, Veterinary Teaching Hospital, Faculty of Veterinary Medicine, University of Las Palmas de Gran Canaria, Las Palmas de Gran Canaria, Spain. E-mail: carlos.melian@ulpgc.es

10.22099/IJVR.2022.42203.6136

(Received 10 Nov 2021; revised version 24 Mar 2022; accepted 1 Nov 2022)

This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/)

Abstract

Background: Budd-Chiari syndrome (BCS) is considered a rare condition in cats that is characterized by the obstruction of the hepatic venous outflow tract from the level of the small hepatic veins to the level of the termination of the inferior vena cava into the right atrium in the absence of cardiac or pericardial disease, or sinusoidal obstruction syndrome. **Case description:** This report presents a 13-year-old cat with a two-week history of progressive lethargy, inappetence, weight loss, and abdominal distension. **Findings/treatment and outcome:** The radiological study was consistent with pleural effusion, as well as alveolar and interstitial pulmonary patterns. Ultrasonography confirmed hepatic venin congestion and ascites. Abdominocentesis revealed a modified transudate. A computed tomography (CT) angiography showed a mass at the level of the caudal mediastinum that compressed the caudal vena cava (CVC). Mediastinal lymphoma was considered the most likely differential diagnosis. These findings were interpreted as Budd-Chiari-like syndrome (BCLS) secondary to a mediastinal mass although, unfortunately, no further diagnostic or treatment procedures were accepted by the owners. BCLS is a rare condition in cats, where most of the reported cases occurred as a result of obstruction of the caudal vena cava. In this report, BCLS was caused by a mass located in the caudal mediastinum oppressing the caudal vena cava. **Conclusion:** This is the first report of BCLS in cats diagnosed by CT angiography, and it shows the value of this technique to define the origin and extent of the mass and to evaluate the presence or absence of the test.

Key words: Budd-Chiari syndrome, Cat, CT angiography, Mediastinal diseases

Introduction

Budd-Chiari syndrome (BCS) is characterized by the obstruction of the hepatic venous outflow tract from the level of the small hepatic veins to the level of the termination of the inferior vena cava into the right atrium in the absence of cardiac or pericardial disease, or sinusoidal obstruction syndrome (Valla, 2009). BCS has been well described in dogs, however, few studies have reported this rare condition in cats. Most studies have shown the obstruction of the caudal vena cava (Macintire *et al.*, 1995; Haskal *et al.*, 1999; Holt *et al.*, 1999; Schrope, 2010; Öztürk *et al.*, 2016) and hepatic vein occlusion or stenosis (Cave *et al.*, 2002; Schrope, 2010). To the authors' knowledge, this is the first report of Budd–Chiari-like syndrome in a cat causing compression of the caudal vena cava by CT angiography.

Case description

A 13-year-old 5 kg sexually intact male cat was referred from a shelter to Hospital Veterinario Los Tarahales (Las Palmas de Gran Canaria, Spain) with a two-week history of progressive lethargy, inappetence, weight loss, and abdominal distension. The cat was indoor/outdoor and had contact with other cats. There was no known history of trauma. The physical abnormalities examination included hypothermia (36.7°C), slightly pale mucous membranes, and distended abdominal and respiratory distress. Abdominocentesis showed a yellow, hazy fluid with a total protein content of 3.1 g/dl. This fluid had a specific gravity of 1005, consistent with a modified transudate. The Rivalta Test was done and revealed no evidence of feline infectious peritonitis (FIP). Feline leukemia virus

antigen (FeLV) and feline immunodeficiency virus antibody (FIV) serology testing were negative. Hematology confirmed mild to moderate anemia (hematocrit 22.5%), which was microcytic, normochromic, and nonregenerative. Biochemical findings included high-normal creatinine (1.6 mg/dl) and potassium (5.8 mmol/L), normal globulin (2.8 g/dl), albumin (3.8 g/dl), and sodium (165 mmol/L) concentrations. Liver enzymes and total thyroxine (T4) concentration (3.3 ug/dl) were within the reference ranges.

Findings/treatment and outcome

Thoracic radiographs showed a standard cardiac silhouette. However, a fluid was gathered in the ventral part of the chest. The pleural effusion (240 ml) was drained that contained abundant degenerate neutrophils. In addition, there was an area of increased opacity in the caudal and dorsal aspects of the thorax (Fig. 1).



Fig. 1: Lateral thoracic radiograph performed at the referring veterinarian showed a standard cardiac silhouette and pleural effusion in the caudal ventral aspect. There is an oval structure in the caudal-middle aspect of the thorax (caudal mediastinum)



Fig. 2: Ultrasound image of the spleen and presence of free fluid in the abdomen

On ultrasonographic evaluation, a small amount of anechoic free abdominal fluid was evident in the cranial abdomen (Fig. 2). Moderate congestion of the hepatic veins was identified, but no significant findings were observed in the remainder of the abdominal ultrasound examination (Fig. 3). The echocardiography was performed and allowed us to exclude cardiopathies, although a marked pleural effusion sign was observed.



Fig. 3: Image of the liver on ultrasound evaluation showing slight dilation of hepatic vessels

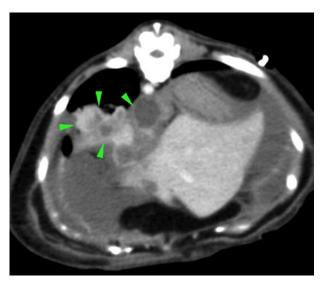


Fig. 4: Post-contrast transverse CT image in soft tissue algorithm. Amorphous, irregular, cavitated, and heterogeneous attenuated mass enhancement at the level of the caudal mediastinum (green arrows) and the thoracic caudal vena cava that is producing venous outflow obstruction

After clinical evaluation, the cat was taken under anesthesia to perform CT angiography using IV injection of iodinated contrast medium (600 mg/kg iobitridol, Xenetix, France). Following contrast medium administration, CT images were acquired with a multidetector scan (16 slices) in the arterial, portal, and venous vascular phases with the following parameters: 100 kV, 60 mA, pitch=0.94. The volume data were reconstructed in bone and soft tissue algorithms and isovolumetric transverse, sagittal, and dorsal planes at a slice thickness of 2 mm. The CT images revealed the enlargement of cranial mediastinal lymph nodes, a marked interstitial pattern of the medium and caudal right lung lobes, and an alveolar-interstitial mixed pattern observed at the left caudal lung lobe. Additionally, an irregularly-shaped cavitary mass was identified in the caudal mediastinum, disturbing the filling of the caudal vena cava at the diaphragmatic and hepatic regions. The contrast administration allowed the better enhancement of a mild heterogeneous soft-tissue attenuation coating the vessel, losing its wall continuity and caudal vena cava outflow (Figs. 4-6). The liver showed normal size, shape, and attenuation. Ultrasoundguided fine needle aspiration of the caudal mediastinal mass was proposed. Unfortunately, this diagnostic procedure and further treatment were not accepted by the owners.



Fig. 5: Post-contrast coronal CT image in soft tissue algorithm. Amorphous, irregular, and heterogeneous attenuated mass enhancement at the level of the caudal mediastinum

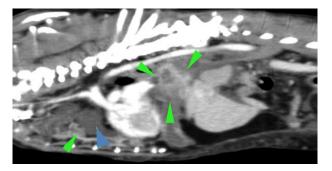


Fig. 6: Post-contrast sagittal CT image in soft tissue algorithm. This image depicts mass enhancement affecting the caudal mediastinum and thoracic caudal vena cava venous outflow (green arrows). In the cranial mediastinum is identified lymphadenopathy (green arrow) and pleural effusion (blue arrow)

Discussion

The term "Budd-Chiari Syndrome" was initially used in human medicine to describe obliterating endophlebitis of the terminal hepatic veins, resulting in veno-occlusive disease. Causes are typically associated with hypercoagulability, inherited deficiencies of protein C, protein S, and antithrombin III, as well as acquired myeloproliferative disorders, paroxysmal nocturnal hemoglobinuria, antiphospholipid syndrome, pregnancy, and oral contraceptive use (Smalberg et al., 2006; Horton et al., 2008). BCS can be divided into primary and secondary types. It is considered primary when the cause of the obstruction is a venous disease such as thrombosis or phlebitis, while it is considered secondary when it is due to compression or invasion of an injury that originates outside of the veins such as tumors, abscesses, intrahepatic cysts, and hematomas (Aydinli and Bayraktar, 2007; Plessier and Valla, 2008; Gaviria et al., 2016).

Documented causes include myeloproliferative disorders, polycythemia vera, paroxysmal nocturnal hemoglobinuria, Behcet's disease, thrombophilia, pregnancy, oral contraceptives, malignancy, infections, and venous stenosis (Valla *et al.*, 1985; Hoekstra *et al.*, 2009).

Similar classification has been reported in dogs with BCS, where Budd-Chiari-like syndrome has been associated with congenital cardiac defects, blunt trauma to the caudal vena cava, and perivenular fibrosis of the hepatic veins (Miller *et al.*, 1989; Cohn *et al.*, 1991; Grooters *et al.*, 1995; Mitten *et al.*, 2001; Langs, 2009; Schlicksup *et al.*, 2009; Gulcubuk *et al.*, 2012). Secondary kinking and extraluminal obstruction of the caudal vena cava flow as a result of a thoracic wall or adrenal gland neoplasia, trauma, and cardiac disease can also lead to the development of BCS (Cornelius *et al.*, 1985; Schoeman *et al.*, 2001; Whelan *et al.*, 2007; Rosa *et al.*, 2012).

Previously reported feline cases of BCLS (Macintire *et al.*, 1995; Cave *et al.*, 2002; Schrope 2010; Hoehne *et al.*, 2014; Öztürk *et al.*, 2016) used abdominal ultrasonography to establish a diagnosis and localize the vascular lesion. Nonetheless, in the cat of the present report, ultrasonography did not identify the lesion but did reveal CVC and hepatic vein distension as indicators of a more cranial CVC stenosis. Chest CT and radiography are both routinely used for detecting thoracic masses in cats. However, CT is more sensitive than radiographs for determining the extent and location of the disease. In our case, thoracic angio-CT allowed the characterization and extent of the thoracic mass.

Diagnosis of this syndrome is based on clinical findings, medical history, liver function tests, and imaging studies (Darwish *et al.*, 2009). Due to the advances in diagnostic imaging techniques such as ultrasonography or computed tomography angiography, new ways of visualizing this process have been reported (Nuñez *et al.*, 2003; Gaviria *et al.*, 2016). Treatment for BCS can be divided into medical therapy

(anticoagulation, thrombolysis), radiological (balloon angioplasty and stenting, transjugular intrahepatic portosystemic shunt *TIPS*), and surgical procedures (portosystemic shunts, liver transplantation). Nonetheless, the therapy of choice depends on the individual clinical and anatomical characteristics (Gaviria *et al.*, 2016).

Any process that disrupts capillary or interstitial hydrostatic or oncotic pressures, lymphatic drainage, or vessel integrity can result in pleural fluid accumulation. In cats, the most common causes of pleural effusion are right and/or left congestive heart failure, effusive feline infectious peritonitis, infectious pleuritis (pyothorax), and neoplasia. Other causes of pleural effusion include trauma and idiopathic chylothorax. Mediastinal, bronchopulmonary, or primary pleural neoplasia may cause pleural effusion with the characteristics of a modified transudate, exudate, or chylous effusion (Beatty et al., 2010). Mediastinal lymphoma accounted for most neoplasia-associated pleural effusions reported in cats (Davies et al., 1996), the most common cranial mediastinal mass, followed by thymomas, idiopathic mediastinal cysts, ectopic thyroid, chemodectoma, and other less common neoplasms. Feline mediastinal lymphoma is typically found in the chest cavity, and most affected cats are 10 to 12 years of age (Fabrizio et al., 2014). Unvaccinated outdoor cats are at greater risk than indoor cats due to their greater exposure to FeLV infection.

Budd-Chiari-like syndrome is a rare phenomenon in veterinary medicine, whose diagnosis can be arduous. Both animals and human patients often present with a vague history and progressive abdominal distension (Schrope, 2010). The presence of hepatomegaly, ascites (modified transudate with elevated protein concentration), and abdominal pain have been described as the triad of clinical signs occurring with Budd–Chiari-like syndrome in dogs and cats (Darwish Murad *et al.*, 2009; Schrope, 2010; Hoehne *et al.*, 2014). In this report, BCS was caused by a mass located in the caudal mediastinum oppressing the caudal vena cava, and mediastinal lymphoma was considered the most likely differential diagnosis.

In conclusion, we describe a case of a presumptive mediastinal lymphoma arising from the caudal mediastinum, compressing the CVC, and associated with Budd-Chiari syndrome in a cat. This case also shows the value of a CT angiography to define the origin and extent of the tumor and evaluate the presence or absence of metastatic lesions. However, a biopsy is needed for a definitive diagnosis. Knowledge of the different morphological patterns expressed by lymphoma or other neoplasia in the chest of cats allows veterinary clinicians to arrange appropriate diagnostic procedures, including immunophenotype and clonality studies, along with therapeutic protocols and prognostic evaluations.

Acknowledgements

In loving memory of Alvaro Domingo Rodriguez

Garcia. We also thank M. Mohamad and J. Jaber for their support and constructive comments.

Conflict of interest

The authors declare that there is no conflict of interest that could inappropriately influence the content of the paper.

References

- Aydinli, M and Bayraktar, Y (2007). Budd-Chiari syndrome: etiology, pathogenesis and diagnosis. World J. Gastroenterol., 13: 2693-2696.
- Beatty, J and Barrs, V (2010). Pleural effusion in the cat. A practical approach to determining aethiology. J. Fel. Med. Surg., 12: 693-707.
- Cave, TA; Martineau, H; Dickie, A; Thompson, H and Argyle, DJ (2002). Idiopathic hepatic veno-occlusive disease causing Budd-Chiari-like syndrome in a cat. J. Small Anim. Pract., 43: 411-415.
- Cohn, LA; Spaulding, KA; Cullen, JM; Bunch, SE; Metcalf, MR; Hardie, EM; MacLachlan, NJ and Breitschwerdt, EB (1991). Intrahepatic postsinusoidal venous obstruction in a dog. J. Vet. Intern. Med., 5: 317-321.
- **Cornelius, L and Mahaffey, M** (1985). Kinking of the intrathoracic caudal vena cava in five dogs. J. Small Anim. Pract., 26: 67-80.
- Darwish Murad, S; Plessier, A; Hernandez-Guerra, M; Fabris, F; Eapen, CE; Bahr, MJ; Trebicka, J; Morard, I; Lasser, L; Heller, J; Hadengue, A; Langlet, P; Miranda, H; Primignani, M; Elias, E; Leebeek, FW; Rosendaal, FR; Garcia-Pagan, JC; Valla, DC and Janssen, HL (2009). Etiology, management, and outcome of the Budd-Chiari syndrome. European network for vascular disorders of the liver. Ann. Intern. Med., 151: 167-175.
- **Davies, C and Forrester, SD** (1996). Pleural effusion in cats: 82 cases (1987 to 1995). J. Small Anim. Pract., 37: 217-224.
- Fabrizio, F; Calam, AE; Dobson, JM; Middleton, SA; Murphy, S; Taylor, SS; Schwartz, A and Stell, AJ (2014). Feline mediastinal lymphoma: a retrospective study of signalment, retroviral status, response to chemotherapy and prognostic indicators. J. Feline Med. Surg., 16: 637-644.
- Gaviria, SC; Ramírez, AC; Espinoza, YP and Restrepo, JC (2016). Review of Budd-Chiari syndrome. Rev. Colomb. Gastroenterol., 31: 241-250.
- Grooters, AM and Smeak, DD (1995). Budd-Chiari-like syndromes in dogs. In: Bonagura, JD (Ed.), *Current veterinary therapy XII*. Philadelphia, PA, USA, W. B. Saunders Co., PP: 876-879.
- Gulcubuk, A; Bozkurt, ER and Erturk, M (2012). Case report: Budd-Chiari-Like syndrome in a dog. Rev. Med. Vet., 163: 7-10.
- Haskal, ZJ; Dumbleton, SA and Holt, D (1999). Percutaneous treatment of caval obstruction and Budd-Chiari syndrome in a cat. J. Vasc. Interv. Radiol., 10: 487-489.
- Hoehne, SN; Milovancev, M; Hyde, AJ; deMorais, HA; Scollan, KF and Nemanic, S (2014). Placement of a caudal vena cava stent for treatment of Budd-Chiari-like

Syndrome in a 4-month-old Ragdoll cat. J. Am. Vet. Med. Assoc., 245: 414-418.

- Hoekstra, J; Leebeek, FWG; Plessier, A; Raffa, S; Darwish Murad, S; Heller, J; Hadengue, A; Chagneau, C; Elias, E; Primignani, M; García-Pagan, JC; Valla, DC and Janssen, HLA (2009). Paroxysmal nocturnal hemoglobinuria in Budd-Chiari Syndrome: Findings from a cohort study. J. Hepatol., 51: 696-706.
- Horton, JD; San Miguel, FL and Ortiz, JA (2008). Budd-Chiari syndrome: illustrated review of current management. Liver Int., 28: 455-466.
- Langs, LL (2009). Budd-Chiari-like syndrome in a dog due to liver lobe entrapment within the falciform ligament. J. Am. Anim. Hosp. Assoc., 45: 253-256.
- Macintire, DK; Henderson, RH; Banfield, C and Kwapien, RP (1995). Budd-Chiari syndrome in a kitten caused by membranous obstruction of the caudal vena cava. J. Am. Anim. Hosp. Assoc., 31: 484-491.
- Miller, MW; Bonagura, JD; DiBartola, SP and Fossum, TW (1989). Budd-Chiari–like syndrome in two dogs. J. Am. Anim. Hosp. Assoc., 25: 277-283.
- Mitten, R; Edwards, GA and Rishniw, M (2001). Diagnosis and management of cor triatriatum dexter in a Pyrenean Mountain Dog and an Akita Inu. Aust. Vet. J., 79: 177-180.
- Nuñez, O; de la Cruz, G; Molina, J; Borrego, GM; Marín, I; Ponferrada, A; Catalina, V; Echenagusia, A and Bañares, R (2003). Interventional radiology, angioplasty and tips in Budd-Chiari Syndrome. J. Gastroenterol. Hepatol., 26: 461-464.
- Öztürk, AS; Altuğ, N; Köse, SI and Öztürk, OH (2016). The effects of L-carnitine in Budd-Chiari Syndrome in a domestic cat. Mac. Vet. Rev., 39: 123-127.
- Plessier, A and Valla, D (2008). Budd-Chiari syndrome.

Semin. Liver Dis., 28: 259-269.

- **Rosa, C; Schoeman, JP and Dvir, E** (2012). Budd Chiari-like syndrome associated with a pheochromocytoma invading the right atrium in a dog. Isr. J. Vet. Med., 67: 180-185. Use of endovascular stents in three dogs with Budd-Chiari syndrome.
- Schlicksup, MD; Weisse, CW; Berent, AC and Solomon, JA (2009). Use of endovascular stents in three dogs with Budd-Chiari syndrome. J. Am. Vet. Med. Assoc., 235: 544-550.
- Schoeman, JP and Stidworthy, MF (2001). Budd Chiari-like syndrome associated with an adrenal phaeochromocytoma in a dog. J. Small Anim. Pract., 42: 191-194.
- Schrope, DP (2010). Hepatic vein stenosis (Budd-Chiari syndrome) as a cause of ascites in a cat. J. Vet. Cardiol., 12: 197-202.
- Smalberg, JH; Murad, SD; Braakman, E; Valk, PJ; Janssen, HL and Leebeek, FW (2006). Myeloproliferative disease in the pathogenesis and survival Budd-Chiari syndrome. Haematologica. 91: 1712-1713.
- Valla, DC (2009). Primary Budd-Chiari syndrome. J. Hepatol., 50: 195-203.
- Valla, D; Casadevall, N; Lacombe, C; Varet, B; Goldwasser, E; Franco, D; Maillard, J; Pariente, EA; Leporrier, M; Rueff, B; Muller, O and Benhamou, J (1985). Primary myeloproliferative disorder and hepatic vein thrombosis. A prospective study of erythroid colony formation *in vitro* in 20 patients with Budd-Chiari syndrome. Ann. Intern. Med., 103: 329-334.
- Whelan, MF; O'Toole, TE; Carlson, KR; Sutherland-Smith, J and Berg, J (2007). Budd Chiari-like syndrome in a dog with chondrosarcoma of the thoracic wall. J. Vet. Emerg. Crit. Care. 17: 175-178.