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Case Report
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Treatment of portal vein thrombosis using vascular access port implantation in a Dalmatian dog: A case report

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Abstract

Background: Portal vein thrombosis is a disease with potentially deleterious outcomes including portal vein hypertension and intestinal infarction. The factors contributing is various; however, dogs with with acute portal vein thrombosis or multiple thromboses are less likely to survive. Therefore, acute development of portal hypertension has a requires an immediate treatment.

Case Description: A 10-year-old Dalmatian was referred for syncope and azotemia, hyperammonemia. After each examinations including computed tomography scan, we diagnosed with acute portal vein thrombosis with unknown cause. A portal vein port was inserted to prevent and control the portal vein thrombus. The port was placed in abdomen subcutaneously after the position of the catheter were stabilized. Low-molecular-weight heparin was injected from the port to manage thrombosis after the operation. This case responded well to this treatment. Syncope and azotemia, hyperammonemia resolved and no relapse of thrombosis was found 6 months after the operation.

Conclusion: Implantable vascular access port is a drug delivery system with the advantage of dealing with treatment-resistant acute portal vein thrombosis.

Keywords: Portal vein, Access port implantation, Dog, Thrombosis.

Introduction

The factors contributing to the development of portal vein thrombosis in dog include hepatic, neoplastic, immune, infectious diseases, protein-losing nephropathy, hyperadrenocorticism, protein-losing enteropathy, and pancreatitis (Van Winkle and Bruce, 1993; Respess *et al*., 2012). Past paper reported that dogs with chronic portal vein thrombosis were more likely to survive, whereas those with acute portal vein thrombosis, multiple thromboses or systemic inflammatory response syndrome (SIRS) were less likely to survive (Respess *et al*., 2012). Therefore, acute development of portal hypertension has a requires immediate treatment. In veterinary medicine, fibrinolytic therapy is not recommended while combinations of antithrombotic to manage thrombosis have been reported (Morassi *et al*., 2016). The management of portal vein thrombosis is difficult when thrombosis aggravates acutely (Van Winkle and Bruce, 1993).

Here we report a case with exacerbating thrombosis despite antithrombotic treatment. To prevent further thrombogenesis, a venous access port was implanted into the portal vein. This case responded well to direct antithrombotic injection via implantable vascular access port.

Case Details

A 10-year-old castrated male 28 kg Dalmatian was referred for syncope and azotemia. On physical examination, the case showed consciousness disorder, sialorrhea, difficulty in standing, and poor physical status. A blood test was performed and revealed marked hyperammonemia (324 μg/ml) as well as a high D-dimer level of 10 μg/ml (reference range <2.0 μg/ml; Fujifilm Monolith Co., Ltd., Tokyo, Japan). No other abnormalities in the coagulation profile were recognized. On abdominal ultrasound, an extension of intrahepatic branches of the portal vein as well as a thrombus in the main portal vein lumen was recognized. For further investigation, computed tomography (CT) imaging was performed (day 1) which confirmed the existence of a thrombus within the main body of the portal vein

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(Fig. 1). No additional abnormal findings were found on CT images. A diagnosis of portal vein thrombosis was made, and antithrombotic therapy consisting of aspirin (Aspirin; Nichi-Iko Pharmaceutical Co., Ltd., Toyama, Japan, 0.5 mg/kg, PO, BID), clopidogrel (Plavix; Sanofi company, Paris, France, 2 mg/kg, PO, BID), and dalteparin sodium (Fragmin; Pfizer Inc., New York, NY, 100 IU/kg, SC, TID) was initiated. On day 3, an increasing trend in the thrombus size was recognized on ultrasound without improvement in the symptoms. To prevent further thrombogenesis, a venous access port was implanted into the portal vein. Anesthesia was solely induced by isoflurane inhalation (Isoflurane for Animal Use; Intervet, Osaka, Japan). After intubation, the dog was situated in a dorsal position. A midline incision on the abdomen with a scalpel from the caudal end of the xiphoid process to the umbilicus was made. After the mesenteric vein was catheterized with a 22 Gauge IV catheter, a 3 Fr sheath was then placed with facilitation by a guidewire to measure the portal vein pressure. The portal vein pressure reading was 20 mmHg. The contrast medium was injected from the sheath and the location of the portal vein thrombus was identified. Under fluoroscopic guidance, a 0.014″ × 175 cm guidewire has proceeded from the sheath placed at the mesenteric vein until the level of the thrombus. When the guidewire becomes in contact with the thrombus, the sheath was removed and replaced with a heparinized saline-infused 5 Fr Anthron P-U catheter (Roray Medical Co., Ltd, Tokyo, Japan). After fixing the Anthron P-U catheter to its insertion into the mesenteric vein with 3-0 silk suture at three places, the distal end of it was cut and connected to a kink-resistant tube and a 5 Fr P-U Celsite port (Roray Medical Co., Ltd, Tokyo, Japan). Following abdominal closure, a subcutaneous pocket was made and the P-U Celsite port was implanted with a 3-0 polydioxanone buried suture. All procedures were summarized in Figure 2. After surgery, the dog was transferred to a radiographic imaging room where postsurgical patency was verified by contrast imaging (Fig. 3).

Administration of dalteparin sodium (100 IU/kg TID) via the implanted port was started from the first postoperative hour. From the day after surgery, plasma ammonia level was reduced while no increase in the size of the thrombus was recognized. The disappearance of the thrombus and stabilization of the plasma ammonia level was recognized the first week after surgery (33 µg/ml). Lavaging of the catheter had been carried out periodically and removal of the access port is under consideration on demand from the client. Upon 6 months follow-up, CT imaging reverified the disappearance of the thrombus in addition to the long-term stabilization of plasma ammonia level (35 µg/ml). Currently, the dog is additionally prescribed with clopidogrel (Plavix; Sanofi company, Paris, France, 2 mg/kg, PO, BID) while the frequency of low-molecular-weight-heparin administration via the access port route being tapered. The cause of thrombosis in this case is still unknown; however, we suspected it is a transient disorder such as intoxication. Histopathological examination revealed no abnormalities in the liver.

Discussion

Portal vein thrombosis is a vascular disease of the liver that occurs when a blood clot gets stuck in the hepatic portal vein, which leads to increased pressure in the portal vein system and reduced blood supply to the liver. Although portal vein thrombosis is a relatively common complication in patients with liver cirrhosis in human clinical settings, in veterinary clinical settings, portal vein thrombosis is caused by varius reason as hepatic, neoplastic, immune, infectious diseases, protein-losing nephropathy, hyperadrenocorticism, protein-losing enteropathy, and pancreatitis (Van Winkle and Bruce, 1993; Respess *et al*., 2012). As dogs with acute portal vein thrombosis were less likely to survive (Respess *et al*., 2012) and prompt treatment might greatly affect a patient's outcome, acute development of portal hypertension has a requires immediate treatment. In veterinary medicine, fibrinolytic therapy is not recommended while combinations of antithrombotic to manage thrombosis have been reported (Morassi *et al*., 2016). Management of portal vein thrombosis is difficult when thrombosis aggravates acutely (Van Winkle and Bruce, 1993). In this case, the general condition was not favorable at referral and demanded immediate treatment for the portal vein thrombosis. Despite

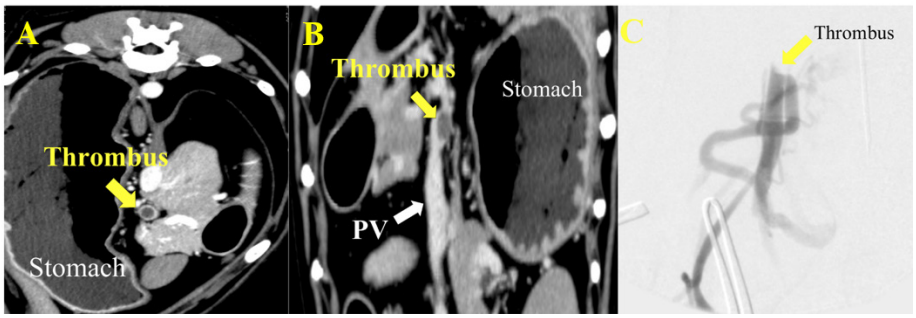

Fig. 1. CT image and contrast imaging. (A): Axial imaging. (B): Coronal imaging. (C): Contrast imaging. Yellow arrow indicates the thrombus.

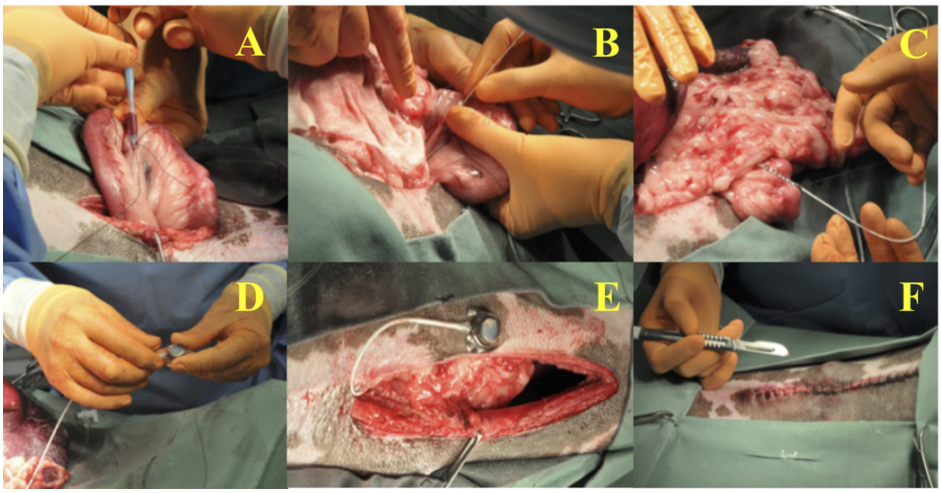

Fig. 2. Process of the venous access port implantation into the portal vein. (A): Inserting a guide wire. (B) and (C): Inserting the 5 Fr Anthron P-U catheter along the guidewire. (D): Connecting the Anthron P-U catheter to the P-U Celsite port. (E) and (F): Fixing the P-U Celsite port.

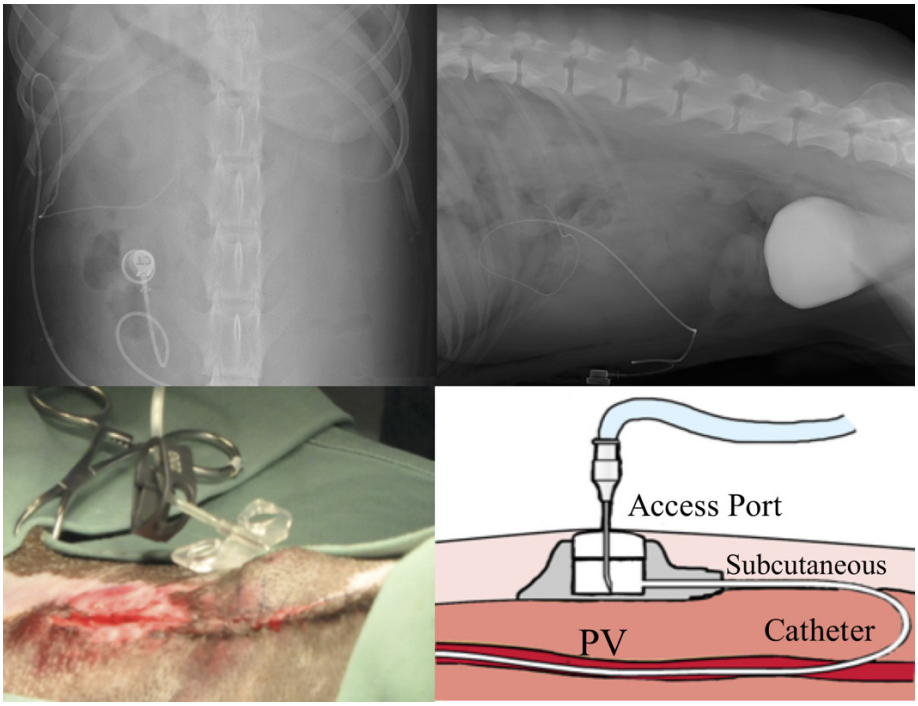

Fig. 3. Radiographic contrast imaging via the venous access port after surgery.

reports on the effectiveness of antithrombotic therapy on preventing novel thrombogenesis, antithrombotic administration via oral and subcutaneous routes in the case did not resolve the thrombosis. Instead, the thrombosis was worsened (Winter *et al*., 2012). To cope with the situation, we established an access port to the thrombotic lesion, through which direct administration of low-molecular-weight-heparin allowed a favorable response. The implantable vascular access port is originally a device for drug delivery in human medicine indicated in cases such as administrating high doses of stimulant medicine or relieving peripheral venous pain (Yamasaki *et al*., 2021). In addition, in human medicine, some paper reports about intraportal administration of antithrombotic (Tokunaga *et al*., 2021). In this case, by direct dalteparin sodium injection into the thrombotic lesion after access port placement, the portal vein thrombosis, previously rafractory to combination treatment with dalteparin sodium, aspirin, and clopidogrel, was successfully managed with improvement in the condition. Besides, the implant well sustained a 6-month infection-free

period without failure. However, caution should be taken when considering the implantable vascular access port. Previous reports have documented a complication rate of 6.6% in cases where the venous access port was placed with the port infection (2.2%), thrombosis (1.3%), extravasation (1.3%), and catheter fracture (1.8%) (Yildizeli *et al*., 2004) Therefore, the risks associated with the port implantation into the portal vein should be considered. On the other hand, as a report stating that the port-induced portal vein thrombosis is preventable by dalteparin sodium also exists, the risk of thrombosis secondary to the implant placement into the portal vein is considered low in this case (Monreal *et al*., 1996). In conclusion, implantable vascular access port provides a practicable solution to refractory cases of portal vein thrombosis. Nevertheless, consideration should be given to several risks when the implantation is indicated.

Conflict of interest

The authors declare no conflict of interest.

Author contributions

TY and KS designed the original draft. AM and AT acquired and analyzed the data. LH, HE, and YO interpreted the results and critically reviewed the results. AU and RT edited the manuscript and approved the final version of the manuscript.

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