Purulent Pericarditis in a Dog Administered Immune-Suppressing Drugs

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ABSTRACT. A 5-year-old castrated mongrel dog was brought to our hospital with anorexia and vomiting. Laboratory testing revealed immune-mediated hemolytic anemia (IMHA), and so treatment was initiated with multiple immune-suppressing drugs, achieving partial remission from IMHA. However, cardiac tamponade due to purulent pericarditis was identified as a secondary disease. Culture of pericardial fluid yielded numerous Candida albicans and multidrug-resistant Acinetobacter sp. Pericardiocentesis was performed, and the condition of the dog improved. However, the dog died the next day.

NOTE

Pericardial effusion is found in 7% of canine cardiac diseases incidentally during echocardiography or as a result of clinical presentation associated with cardiac tamponade [13]. When pericardial effusion increases, intrapericardial pressure rises above the diastolic intracardiac pressure and results in cardiac tamponade. In cardiac tamponade, cardiac output is reduced, and this results in hypovolemia in peripheral tissue and ultimately cardiogenic shock [6, 13, 14]. Pericardial effusion is mostly commonly the result of a tumor or unknown cause (about 90% of cases), and other causes, such as infection or cardiac rupture, are rare [6, 12]. Purulent pericarditis has been described in association with coccidioidomycosis, aspergilllosis, actinomycosis, nocardiosis and tuberculosis [2, 4, 6, 13, 14]. The treatment for purulent pericarditis is removal of pericardial fluid by pericardiocentesis and administration of a suitable antibiotic. However, if the dog is stable enough for surgery, pericardectomy is recommended [6, 13, 14]. The present report describes a case of cardiac tamponade due to purulent pericarditis associated with Candida albicans and Acinetobacter sp. in a dog administered immunosuppressive drugs for treatment of immune-mediated hemolytic anemia (IMHA).

A 5-year-old neutered male mongrel dog was brought to our hospital with vomiting, depression and anorexia. On clinical examination, the dog was found to be obese with slight palor of the visible mucous membranes. No cardiac murmur was evident. Blood testing revealed anemia (packed cell volume 18%; reference range 33 to 55%) and increased levels of alkaline phosphatase (1628 U/l; reference range 47 to 254 U/l) and total bilirubin (0.8 mg/dl; reference range 0.1 to 0.5 mg/dl). No autoagglutination of blood was observed. Anosocytosis and polychromasia with a moderate number of spherocytes were observed. A large number of reticulocytes was also evident. A direct Coombs’ test yielded positive results. Abdominal radiography showed severe splenomegaly, but ultrasonography of the spleen showed normal results.

The clinical diagnosis for this dog was IMHA, so administration of prednisolone (2 mg/kg, once daily) was initiated. However, the packed cell volume decreased the next day, and high-dose steroid pulse therapy (methylprednisolone sodium succinate, intravenous, once daily at 20 mg/kg on the first 3 days, then 10 mg/kg, 5 mg/kg and 2.5 mg/kg for the next 3 days, and 2 mg/kg of prednisolon once daily thereafter), 2 mg/kg azathioprine once daily and 2.5 mg/kg orbifloxacin once daily were administered. Intravenous human immunoglobulin (2.5 g) and whole blood transfusion (200 ml) were administered on day 3. The packed cell volume rose to 20% by day 7, and danazol (5 mg/kg, twice daily) was initiated as maintenance therapy. The intravenous catheter was removed on day 7. However, because the packed cell volume decreased on day 16, cyclosporine twice daily at a dose of 5 mg/kg was substituted for azathioprine. Remission was obtained on day 19. The white blood cell count subsequently increased to 65,600/µl on day 19, and so 30 mg/kg cefalexin twice daily was substituted for orbifloxacin (Fig. 1). The white blood cell count subsequently decreased to 31,500/µl, but nasal discharge was observed, and the white blood cell gradually elevated again. Five mg/kg enrofloxacin once daily was substituted for cefalexin on day 30. The white blood cell count rose to 92,400/µl on day 35, and a blood culture was cultured. However, the dog was brought to our hospital due to weakness and anorexia on day 39.

Physical examination of the dog showed tachypnea and muffled heart sounds, and pyrexia was noted (39.5°C). Blood testing showed a decreased white blood cell count (37,500/µl) with a shift to the left. The blood concentration of cyclosporine was 336 ng/ml. Radiography of the thorax revealed gross enlargement of the cardiac silhouette with a globular appearance (Fig. 2). Echocardiography indicated...
severe pericardial effusion with a thick pericardium and collapse of the right atrium, suggesting cardiac tamponade (Fig. 3).

Pericardiocentesis was performed and 650 ml of bloody fluid containing a large number of white blood cells and budding yeasts was removed (Fig. 4). Microbiological culture of pericardial effusion yielded growth of *C. albicans* and *Acinetobacter sp.* Based on various findings, the clinical diagnosis was purulent pericarditis associated with *C. albicans* and *Acinetobacter sp.* The clinical findings improved after pericardiocentesis, and the cardiac silhouette was diminished on thoracic radiography. Intravenous administration of 20 mg/kg cefpiramide sodium twice daily and peroral administration of 10 mg/kg ketoconazole twice daily were initiated. Pericardiotomy and placement of a thoracostomy tube were scheduled, but the dog died from multi-organ failure on day 40. A postmortem examination could not be performed.
About 2.5–15 ml of pericardial fluid is present in a normal 20-kg dog, and pooling of 50–150 ml of pericardial fluid causes no serious clinical symptoms. If pooling of pericardial fluid occurs slowly, even several hundred milliliters of fluid can accumulate [6].

The present case was thought to have been comparatively chronic, as large quantities of pericardial fluid were removed by pericardiocentesis. If this was the case, the possibility of purulent pericarditis at the time of IMHA diagnosis could not be excluded. However, the likelihood of purulent pericarditis preceding IMHA was considered to be low due to the clinical presentation. In addition, the C. albicans and Acinetobacter sp. identified in the pericardial fluid in this case are both causative organisms for opportunistic infection. Long-term use of wide-spectrum antibiotics and strong immunosuppressive multi-drug therapy was considered the most probable cause of infection. The route by which the infection extended to the pericardium was unidentified, but wound-related extension appeared unlikely, and systemic infection spread via the circulation appeared very likely. No other organs appeared to be infected in the present case. However, other infected organs might have been found if we had been able to perform a postmortem examination.

In humans, pericarditis associated with Candida species is seldom reported [3, 8–10]. The risk factors of pericarditis associated with Candida species in humans are thoracic surgery, malignancy, steroids, diabetes, abdominal surgery and transplantation. Survival rates differ according to the route of infection.

As IMHA is a fatal disease if remission is not obtained, strong immunosuppressive therapy is required for intractable IMHA. Strong immunosuppressive therapy is considered to increase the risk of infection. Little investigation of this issue has been performed in the field of veterinary medicine, but bacterial abscesses in the brain of a dog administered cyclosporine and toxoplasmosis in a dog that received immunosuppressive multi-drug therapy have been reported [1, 11]. The possibility of secondary illness resulting from the use of immunosuppressive agents must always be considered.

There is room for argument in regard to preventive use of antibiotics for animals in an immunosuppressed state. In the present case, the sustained rise in the white blood cell count and the upper respiratory tract infection meant that use of an antibiotic could not be avoided. However, had blood culture been performed early and an appropriate antibiotic selected, the infection might have been controllable.

In humans, systemic anti-fungal therapy using amphotericin B is the major management strategy used for purulent pericarditis caused by candida species [10]. We treated this case with peroral ketoconazole, which was the only anti-fungal drug we could use immediately. However, intravenous amphotericin B might be a more suitable therapy.

Reports of a dog developing purulent pericarditis as a result of immunosuppressive therapy appear to be rare. Cases with similar characteristics in dogs under strong immunosuppressive treatment must be accumulated before the details of purulent pericarditis can be considered in the future. The present report appears to represent the first description of purulent pericarditis associated with Candida species in dogs.

REFERENCES


