Disappearance of pericardial effusion by suspected pericardial-pleural fistula in a Miniature Schnauzer dog

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(Received: February 2, 2018; Revised: May 14, 2018; Accepted: May 25, 2018)

Abstract: A 13-year-old spayed female Miniature Schnauzer was presented with complaints of intermittent syncope. Pericardial effusion was confirmed based on the physical examination, thoracic radiographs and echocardiography. Subsequently, prompt pericardiocentesis was performed. Clinical abnormalities were immediately improved after pericardiocentesis. However, the clinical signs associated with acute collapse recurred. After the second pericardiocentesis, thoracic radiographs revealed pleural effusion, and the clinical signs resolved rapidly. The dog underwent pleural aspiration. Analysis of pleural fluid revealed almost similar features as the previous pericardial fluid. It was possible that a pericardial-pleural fistula was created during the pericardiocentesis. The pericardial and pleural effusion disappeared after the procedures.

Keywords: canine, cardiac tamponade, pericardiocentesis, pleural effusion

Pericardial effusion (PE) is the accumulation of excessive fluid in the pericardial sac, which result in clinical signs of cardiac tamponade [2, 11]. Dogs with PE typically present with hypotension, muffled heart sounds, lethargy, exercise intolerance, or collapse [2, 8]. Regardless of the causes, the initial therapy in patients with PE and tamponade must be directed at reducing intrapericardial pressure as soon as possible to enhance cardiac filling.

Pericardiocentesis involves catheter insertion to remove pericardial fluid for diagnostic purposes and more often for therapeutic intervention against cardiac tamponade caused by PE [9]. Serious complications associated with pericardiocentesis are rare [5]. Potential complications include ventricular arrhythmias, atrial tearing, cardiac puncture, and laceration of the tumor or coronary artery resulting in intrapericardial hemorrhage [5, 9, 13].

The formation of pericardial-pleural fistula is an uncommon complication of pericardiocentesis in human medicine [14]. It might occur during either needle insertion or guide wire advancement [14]. However, to the best of the author’s knowledge, a pericardial-pleural fistula has not yet been reported in veterinary medicine. This case report describes the clinical features of a suspected pericardial-pleural fistula in a Miniature Schnauzer.

A 13-year-old spayed female Miniature Schnauzer weighing 6.80 kg was presented with acute collapse. The dog had a history of intermittent syncope. The owner reported that the first syncope was observed within 1 week. On presentation, generalized weakness, prolonged capillary refill time (> 2 sec), jugular venous distension, muffled heart sound and pulsus paradoxus were detected. Above all, PE was highly suspected. Complete blood count showed a decreased packed cell volume (22.1%; reference interval [RI], 37.3−61.7%).

Serum biochemistry profile revealed a decreased serum albumin concentration (1.9 g/dL; RI, 2.6−3.3 g/dL), an increased serum alkaline phosphatase activity (1423 IU/L; RI, 29−97 IU/L), and an increased C-reactive protein concentration (35 mg/L; RI, 0−20 mg/L). No evidence of coagulopathy was detected. Thoracic radiography revealed sharply delineated cardiac silhouette and widening of the caudal vena cava (Fig. 1A and B). With echocardiography, PE was identified as a circumferential anechoic space surrounding the heart (Fig. 1C).

The dog was stabilized via pericardiocentesis to remove fluid from the pericardial space and to examine the etiology. During the procedure, echocardiographic evidence of cardiac tamponade was presented by the collapse of the free wall of the right atrium (Fig. 1D). Pericardial fluid was removed and quantified, and analyzed. A total of 30 mL of pericardial fluid was removed. PE showed grossly port-wine color. Fluid analysis was conducted for the estimation of total protein concentration (2.9 g/dL), total red blood cell counts (155 cells/μL), and total nucleated cell counts (7,300 cell/μL). Hematocrit (11.1%) levels indicated hemorrhagic inflammatory exudate. Infectious agents or abnormal lymphoid cells were not iden-tified.

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tified and bacterial culture tested negative. After pericardiocentesis, the dog was immediately stabilized, and echocardiographic examination was performed to examine the etiology of PE. However, there was no evidence of neoplasia and left atrial rupture on echocardiography. Therefore, idiopathic PE or malignant mesothelioma was highly suspected. However, request for further tests for a definitive diagnosis were declined by the owner.

Three hours after pericardiocentesis, the dog showed acute collapse, tachycardia (> 180 beats/min), tachypnea (> 60 breaths/min), electrical alternans on patients monitoring device, and hypotension (< 90 mmHg). Echocardiograph revealed recurrence of PE, and pericardiocentesis was performed immediately. A total PE fluid volume of 25 mL was removed. A thoracic radiograph was obtained after the procedure, revealing a right pleural effusion that was not detected on the thoracic radiograph only 3 h before the pericardiocentesis (Fig. 2). Despite the pleural effusion, the dog’s clinical abnormalities improved immediately. The following day, the dog underwent thoracocentesis and 100 mL of pleural fluid was drained. Echocardiographic examination revealed no significant PE. Analysis of pleural fluid revealed hemorrhagic inflammatory exudate that same feature with PE as reported before (Table 1). We suspected the formation of a pericardial-pleural fistula during pericardiocentesis. No more PE was reported, and the dog is still alive without recurrence of PE or pleural effusion for 6 months.

This report describes a case of a presumptive pericardial-pleural fistula during pericardiocentesis in a Miniature Schnauzer. The dog showed rapid resolution of PE and development of immediate pleural effusion after pericardiocentesis. During pericardiocentesis, a pericardial-pleural fistula was created during the needle insertion or after the needle removal from pericardial sac. However, the formation of pericardial-pleural fistula is an uncommon event. In this case, it was created due to high intrapericardial pressure caused by the combination of rapid PE accumulation and a relatively low pericardial compliance.

Volumes as low as 50–150 mL (for a 20 kg dog) of PE that accumulate rapidly cause large increases in intrapericardial pressure [5], which induces acute cardiac tamponade [4]. In the present case, 25 mL of PE was removed during the second pericardiocentesis, indicating rapid accumulation of relatively small volumes of PE within 3 h. Furthermore, it suggested relatively high intrapericardial pressure. In this scenario, a fistula was created by the insertion of the needle for pericardiocentesis, PE fluid, therefore, might be drained from the pericardial sac into the pleural cavity due to the high intrapercardial pressure.

### Table 1. Comparison of the results of analysis between pericardial fluid and pleural fluid occurred after the second pericardiocentesis

| Analytes                  | Pericardial fluid | Pleural fluid | Blood |
|---------------------------|-------------------|---------------|-------|
| Total protein (g/dL)      | 2.9               | 3.9           | 6.0   |
| Hematocrit (%)            | 11.1              | 8.0           | 22.1  |
| Total RBC (cells/µL)      | $1.62 \times 10^6$ | $1.19 \times 10^6$ | $3.09 \times 10^6$ |
| Total nucleated cell count (cells/µL) | 7,300            | 3,850         | 6,970 |
The intrapericardial pressure depends on the rate of fluid accumulation as well as the volume of effusion [9]. Reduction of pericardial compliance also increases the intrapericardial pressure, following pericardial neoplasia or chronic pericardial inflammation. Echocardiography is the most sensitive and noninvasive diagnostic test for the detection of PE and intrapericardial neoplasia [1]. However, mesothelioma, a malignant neoplasm that affects the epithelial cells lining the serosal surfaces of body cavities including the pericardial space, represents a significant diagnostic challenge because differentiation from idiopathic PE is often very difficult [10]. Indeed, echocardiography does not identify 100% of the intrapericardial tumors [7, 9]. Furthermore, mesothelioma can be difficult to diagnose because of the similarity between neoplastic and reactive mesothelial cells during the cytologic evaluation of PE fluid obtained by pericardiocentesis [9]. Surgical or thoracoscopic biopsy for histopathologic examination is necessary for a definitive diagnosis [12], however, the owner denied the procedures. Chronic pericardial inflammation of the pericardium associated with idiopathic PE [12] also reduced pericardial compliance in the present case. Idiopathic PE is a diagnosis of exclusion in cases without intrapericardial masses following a comprehensive echocardiographic evaluation, and the results of pericardial fluid analysis failed to disclose the etiology [3]. Pericardial histopathological examination of dogs with idiopathic PE demonstrated a spectrum of changes that include extensive fibrosis, mixed inflammatory cell infiltrate, and perivascular lymphoplasmacytic aggregates [3]. Although these findings are often indistinguishable from those detected in dogs with neoplastic PE, pericardial histopathologic examination facilitates the diagnosis of idiopathic PE. Furthermore, all the diagnostic tests (radiography, echocardiography, cytology, and bacterial culture of fluid obtained by pericardiocentesis) described in the present report failed to demonstrate a definitive cause of pericardial fluid accumulation [12]. However, in the absence of any other possible etiology of PE, we suggest that a reduction of pericardial compliance with either mesothelioma or idiopathic PE was involved in the formation of a pericardial-pleural fistula in the present case without pericardial histopathologic examination.

In the present case, pleural effusion was identified immediately after the second pericardiocentesis. It was not apparent during thoracic radiography only 3 h before the second pericardiocentesis. In addition, similar features of pleural effusion were identified with PE, suggesting evidence of pericardial-pleural fistula. Although rarely reported in human medicine, a pericardial-pleural fistula should be considered in the following circumstances: advent of difficult pericardiocentesis, evidence of significantly increased intrapericardial pressure during echocardiography, poor drainage of pericardial fluid, and rapid resolution of PE without aspiration of expected fluid [5]. In addition, echocardiographic detection of a newly formed pleural effusion during the procedure facilitates immediate diagnosis.

A pericardial-pleural fistula might be one of the complications caused by pericardiocentesis, but it can induce drainage of pericardial fluid into the pleural cavity and this fluid can be absorbed [14]. Some dogs with idiopathic PE can resolve spontaneously after one or more pericardiocentesis [6] but the exact mechanism of this recovery has not been clearly known. In our case, the dog’s clinical abnormalities related to PE were completely recovered and fortunately, it did not recur for 6 months. Its possible reason might be explained with that a pericardial-pleural fistula allowed drainage of the fluid caused by idiopathic PE into the pleural cavity from which they can be more readily absorbed, similar to the clinical feature after thoracoscopic pericardial window [6]. However, clinician should be aware that drainage of the PE caused by infection or neoplasia such as mesothelioma or other neoplastic reason can spread all the infectious organisms or neoplastic cells to the pleural cavity.

The case reported here illustrates rapid resolution of a PE after pericardiocentesis in a Miniature Schnauzer. It is possible that a pericardial-pleural fistula was created during pericardiocentesis. To the author’s knowledge, this phenomenon has not been previously documented in dogs.

Acknowledgments

This study was supported by the Basic Science Research Program, through the National Research Foundation of Korea, which was funded by the Ministry of Science, ICT, and Future Planning (NRF-2016R1A1A05005395).

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