Case Report

Management of refractory spontaneous pneumothorax complicated by secondary *Acinetobacter baumannii* pyothorax in a Siberian Husky using autologous blood patch pleurodesis

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Abstract

A 3-year-old intact male Siberian Husky was presented to Prasu Arthorn Veterinary Teaching Hospital with acute restrictive dyspnea and was diagnosed with spontaneous pneumothorax based on thoracic radiography and computed tomography, which revealed pulmonary bullae and pneumomediastinum. Initial thoracocentesis resulted in temporary improvement; however, recurrence necessitated placement of bilateral thoracostomy tubes. Because persistent air leakage continued despite thoracostomy tube drainage and conservative suction, and thoracotomy was not feasible, the pneumothorax was considered refractory to conservative management and autologous blood patch pleurodesis was performed as salvage therapy. The complication progressed to a pyothorax associated with *Acinetobacter baumannii* infection, confirmed by bacterial culture and antimicrobial susceptibility testing. Targeted therapy with marbofloxacin (5.5 mg/kg/day) resulted in clinical improvement. No recurrence of pneumothorax was observed during the four-month follow-up period. This case demonstrates that autologous blood patch pleurodesis may be an effective alternative for persistent pneumothorax, although infectious complications should be closely monitored.

Keywords: autologous blood pleurodesis, primary spontaneous pneumothorax, pulmonary bullae, pyothorax, thoracostomy tube

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Introduction

Pneumothorax is a potentially life-threatening condition characterized by the accumulation of free air within the pleural space, originating from either the respiratory tract or the external environment (Fossum, 2021). This accumulation disrupts negative intrathoracic pressure, leading to pulmonary collapse and potential cardiovascular compromise, especially in tension pneumothorax, where a one-way valve effect occurs (Sharma and Jindal, 2008). Pneumothorax is classified as acquired or spontaneous. Acquired results from trauma, surgery, or iatrogenic injury. Spontaneous occurs without trauma, divided into primary and secondary forms (Fossum, 2021). Primary spontaneous pneumothorax (PSP) is defined as the accumulation of air within the pleural cavity resulting from the spontaneous rupture of subpleural blebs or bullae in the absence of underlying macroscopic pulmonary disease or external trauma, a condition commonly documented in deep-chested breeds such as the Siberian Husky (Gilday *et al.*, 2021a). In contrast, secondary spontaneous pneumothorax (SSP) develops as a complication of identifiable pre-existing parenchymal lung pathology, such as neoplasia, pneumonia, pulmonary abscesses, or severe underlying infectious disease, which structurally weakens the lung tissue and leads to secondary air leakage (Puerto *et al.*, 2002; Sobel and Williams, 2009).

Dogs with pneumothorax show signs such as rapid breathing, increased respiratory effort, and abdominal movement. Lung sounds are muffled or absent, especially in dorsal lung areas where free air collects, and heart sounds may be reduced. Severity depends on air volume; mild cases may present with vague signs such as lethargy or cough, while severe cases, including tension pneumothorax, can cause respiratory distress, orthopnea, and cyanosis. Increased intrathoracic pressure can cause obstructive shock, marked by tachycardia, weak pulses, and delayed capillary refill. PSP often causes sudden distress in healthy, deep-chested breeds, whereas SSP may present with chronic pulmonary symptoms, such as weight loss or cough. Diagnosis of pneumothorax follows a tiered approach that prioritizes patient stability. In distressed patients, thoracocentesis is both diagnostic and lifesaving and should be performed before stressful imaging (Gilday *et al.*, 2021a). Thoracic focused assessment with sonography for trauma (TFAST), a point-of-care ultrasound technique, offers rapid, non-invasive screening by detecting the absence of a "glide sign," though its accuracy depends on the operator (Lisciandro *et al.*, 2008). Thoracic radiography is the primary diagnostic tool because of its high sensitivity, with signs such as lung retraction, dorsal heart elevation, and pulmonary atelectasis. In severe tension cases, diaphragmatic flattening or mediastinal shifts may happen (Thrall, 2018). Computed tomography (CT) is the gold standard for detecting small air volumes and lesions like bullae, aiding surgical planning, but is usually for stable patients due to sedation or anesthesia needs (Gilday *et al.*, 2021a).

Management begins with conservative stabilization via intermittent or continuous suction using thoracocentesis or thoracostomy tubes (Kramek and Caywood, 1987). Surgery, including bullectomy or pulmonary lobectomy, remains the definitive treatment for persistent air leaks but is often precluded by diffuse pulmonary pathology, patient instability, or financial constraints (Puerto *et al.*, 2002; Oppenheimer *et al.*, 2014). In such cases, particularly for patients with metastatic neoplasia where quality of life is key, surgical options may be inappropriate (Shinsako *et al.*, 2022). Alternatively, pleurodesis offers a minimally invasive way to seal leaks by inducing pleural adhesions. While chemical agents can cause inflammation, autologous blood patch pleurodesis (ABPP) is safer and cost-effective. Injecting the patient's blood into the pleural space establishes a biological seal with fewer complications than synthetic agents, thereby effectively managing refractory pneumothorax when surgical intervention is not feasible.

This case report describes the clinical presentation, diagnostic challenges, and therapeutic interventions for a dog with spontaneous pneumothorax complicated by pulmonary abnormalities and pyothorax. Specifically, it highlights the decision-making process for managing complex thoracic conditions and explores the use of ABPP as a minimally invasive, cost-effective approach to persistent air leaks. By detailing the patient's course and long-term outcome, this case aims to provide clinical insights into managing refractory pneumothorax when conventional surgical options are limited.

Case Description

A 3-year-old male Siberian Husky was presented to Prasu Arthorn Veterinary Teaching Hospital with increased respiratory effort. The owner reported signs began one day before and worsened overnight, with no prior medication or treatments. No history of open-mouth breathing, coughing, sneezing, or abnormal respiratory sounds was noted. There was no trauma or foreign body ingestion. The dog was mainly indoors with supervised outdoor time, current on vaccinations, and received routine ectoparasite and heartworm prevention. His diet was cooked chicken liver and rice, with no recent changes. No prior medical conditions or chronic illnesses were documented. Diagnostic procedures were performed to determine the cause of respiratory distress and guide treatment. On the first visit, a physical exam showed the patient was bright, alert, and responsive (BAR). Clinical parameters included pink mucous membranes, capillary refill time (CRT) of less than 2 seconds, and a rectal temperature of 102.6°F. No ocular or nasal discharge was observed. Thoracic auscultation revealed tachypnea with a restrictive pattern—short, shallow breaths with increased abdominal effort. Lung sounds were mildly increased, with dull areas in specific lobes, and heart sounds were muffled. Femoral pulses were strong, and hydration, lymph nodes, and abdomen were normal. CBC and biochemistry were within normal limits (Table 1). An IDEXX SNAP 4DX test was negative for vector-borne diseases. At this stage, no C-reactive

protein (CRP) measurement was available; therefore, the initial assessment of systemic inflammation was based primarily on the physical examination and routine hematological findings, including the absence of fever and leukocytosis. These findings did not indicate marked systemic inflammation at presentation and supported the patient's clinical stability before sedation for thoracocentesis. Thoracic radiography on the first day confirmed a significant pneumothorax (Figs. 1a, 1b). Key radiographic findings included lung lobe retraction from the thoracic wall and dorsal elevation of the cardiac silhouette above the sternum, both indicating incomplete pulmonary expansion. The following day, a modified Knott's test was performed to rule out *Dirofilaria immitis* microfilariae as a potential underlying cause of secondary spontaneous pneumothorax; however, the test results were negative. Thoracic CT performed three days later further characterized the pulmonary pathology (Figs. 2a–2d). Multiple pulmonary bullae were identified in the left caudal, right middle, and accessory lung lobes. Imaging also revealed pulmonary fibrosis and pneumomediastinum. Focal changes suggestive of localized inflammation or infiltrative disease were noted in the right cranial and right middle lung lobes. Incidental findings included splenomegaly, considered nonspecific, and right-sided abdominal cryptorchidism. Overall, the advanced imaging findings were consistent with spontaneous pneumothorax associated with pulmonary bullae.

Initial management targeted immediate stabilization and pneumothorax resolution. On presentation day, emergency thoracocentesis was performed after diagnosis. Sedated with butorphanol (0.3 mg/kg IV) (Zoetis) and propofol (0.8 mg/kg IV, titrated) (B. Braun), 952 mL of air was aspirated from the right hemithorax at the 8th–9th intercostal space. Post-procedure radiographs showed reduced pleural air. Reassessed on days two and three, radiographs revealed recurrent pneumothorax, worsening since initial drainage. On day four, a thoracic CT scan was done (GE Healthcare), followed by bilateral thoracostomy tubes at the 8th–9th intercostal space for continuous air evacuation. The patient received premedication with diazepam (0.2 mg/kg IV) (GPO, Bangkok) and morphine sulfate (0.4 mg/kg IM) (M&H) induced with propofol (4 mg/kg IV, titrated) (B. Braun), and perioperative amoxicillin–clavulanic acid (20 mg/kg IV) (Zoetis). Despite continuous thoracic drainage and suction, the pneumothorax persisted. By day ten, follow-up thoracic radiographs showed persistent pneumothorax despite thoracostomy tube drainage and suction, with mild subcutaneous emphysema also noted. Given the continued air leakage and failure of conservative management over several days, the pneumothorax was considered refractory to conventional therapy. Thoracotomy was considered as the definitive treatment; however, CT had demonstrated multiple pulmonary bullae involving several lung lobes, including the left caudal, right middle, and accessory lung lobes, which would have required extensive surgical exploration and potentially multiple pulmonary resections. In addition, the owner had concerns regarding surgical intervention. Therefore, thoracotomy was not pursued, and autologous blood patch pleurodesis (ABPP) was selected as a minimally invasive salvage therapy. The patient weighed 24 kg. The target volume of autologous blood was calculated using an approximate dose of 10 mL/kg, resulting in a total target volume of 240 mL. For the ABPP procedure, the patient was sedated with butorphanol (0.3 mg/kg IV) (Zoetis) and propofol (0.8 mg/kg IV, titrated) (B. Braun). Under strict aseptic technique, a total of 240 mL of autologous whole blood was collected and immediately instilled into the pleural space via the bilateral thoracostomy tubes. The total volume was divided between the two hemithoraces, with 140 mL administered into the left pleural cavity and 100 mL into the right pleural cavity (Fig. 3). Acetated Ringer's solution was given at 125 mL/h during the procedure. Postoperatively, amoxicillin–clavulanic acid (25 mg/kg PO) was prescribed twice daily for 7 days. From the tenth to fifteenth day, the patient was stable with thoracostomy tubes. Air drainage decreased to 0.48–2.4 mL/kg/day on the left and 0.28–1.6 mL/kg/day on the right, with radiographs showing gradual lung re-expansion (Figs. 1c, 1d). On the twentieth day, the patient developed acute depression and hyperthermia (107°F). No specific clinical evidence of thermal injury was observed. Thoracostomy tube discharge was serosanguineous to purulent. Blood tests showed leukopenia, neutropenia, and elevated C-reactive protein (CRP). Cytology and bacterial culture confirmed pyothorax from *Acinetobacter baumannii*. Treatment with marbofloxacin (5.5 mg/kg/day) was started. The patient became fever-free within 24 hours, and clinical improvement was noted as the effusion decreased. Bloodwork normalized the next day. Thoracostomy tubes were removed on day 24 after radiographic resolution. Over four months, the patient remained free of dyspnea with no evidence of recurrence.

Table 1 Hematological and serum biochemical profiles of the patient at different time points (first visit, Day 20, and Day 21).

Parameters	Reference (Absolute)	Day 0 (presentation)	Day 20	Day 21
WBC (cells/μL)	6,000–17,000	12,980	4,800	13,960
Monocyte	150–1530	600	290	837
Neutrophil	3,000–11,500	7,970	2,780	12,000
Lymphocyte	1,000–4,800	3,190	1,150	837
Eosinophil	100–1,250	1,140	580	0
Basophil	<100	0	0	0
RBC (10^6/μL)	5–9	6.76	7.04	5.84
Hb (g/dL)	10–18	16.8	15.3	12.8
HCT %	35–55	48.2	43.9	36.4
Band	0	0	0	2
Platelet (10^3/μL)	200–500	223	225	203
CRP (mg/dL)	0–19	N/A	72.8	N/A
Albumin (g/dL)	2.7–3.8	3.4	N/A	N/A
ALP (U/L)	23–212	198	N/A	N/A
ALT (U/L)	10–100	24	N/A	N/A
BUN (mg/dL)	7–27	11	N/A	N/A
Creatinine (mg/dL)	0.5–1.8	1.37	N/A	N/A
Glucose (mg/dL)	77–125	100	N/A	N/A
Total protein (g/dL)	5.2–8.2	7.0	N/A	N/A
Globulin (g/dL)	2.5–4.5	3.6	N/A	N/A

WBC: white blood cell, RBC: red blood cell, Hb: hemoglobin, HCT: hematocrit, CRP: C-reactive protein, ALP: alkaline phosphatase, ALT: alanine aminotransferase, BUN: blood urea nitrogen, N/A: not available.

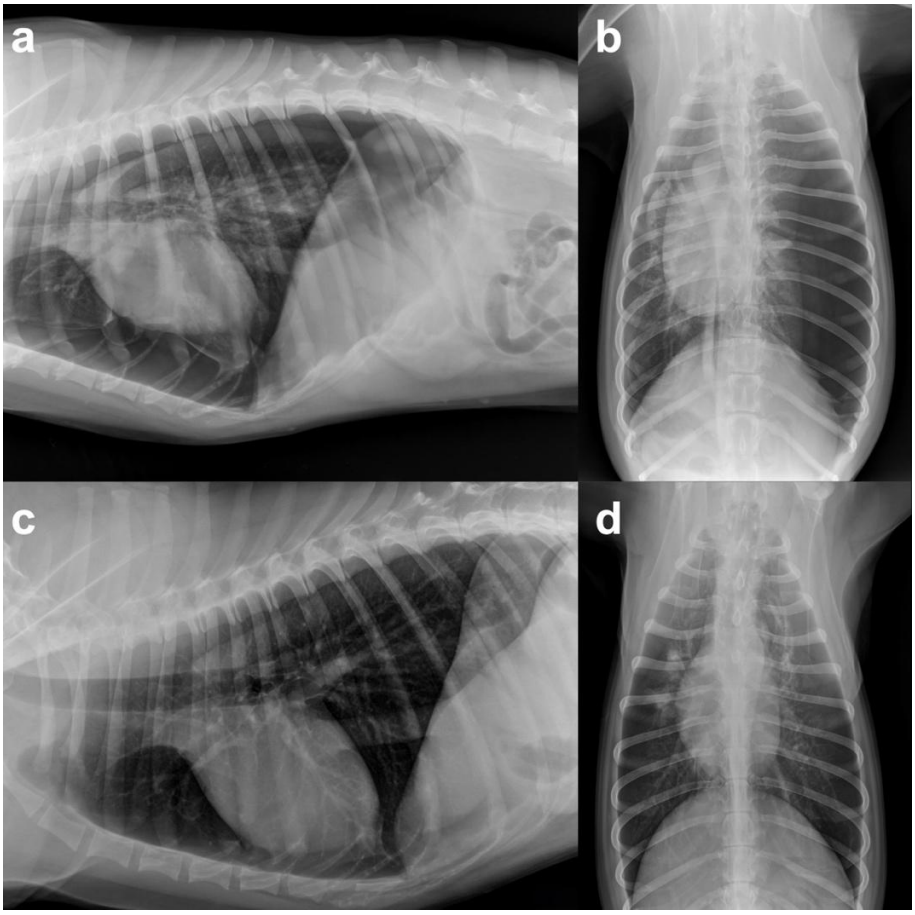


Figure 1 Lateral (a) and ventral-dorsal (b) views of plain thoracic radiographs revealed lung lobe retraction from the thoracic wall and elevation of the cardiac silhouette above the sternum, consistent with pneumothorax, pulmonary bullae, and atelectasis. Lateral (c) and ventrodorsal (d) views of a plain thoracic radiograph revealed no evidence of recurrent pneumothorax, fluid accumulation, or other significant intrathoracic abnormalities over the past 2 months.

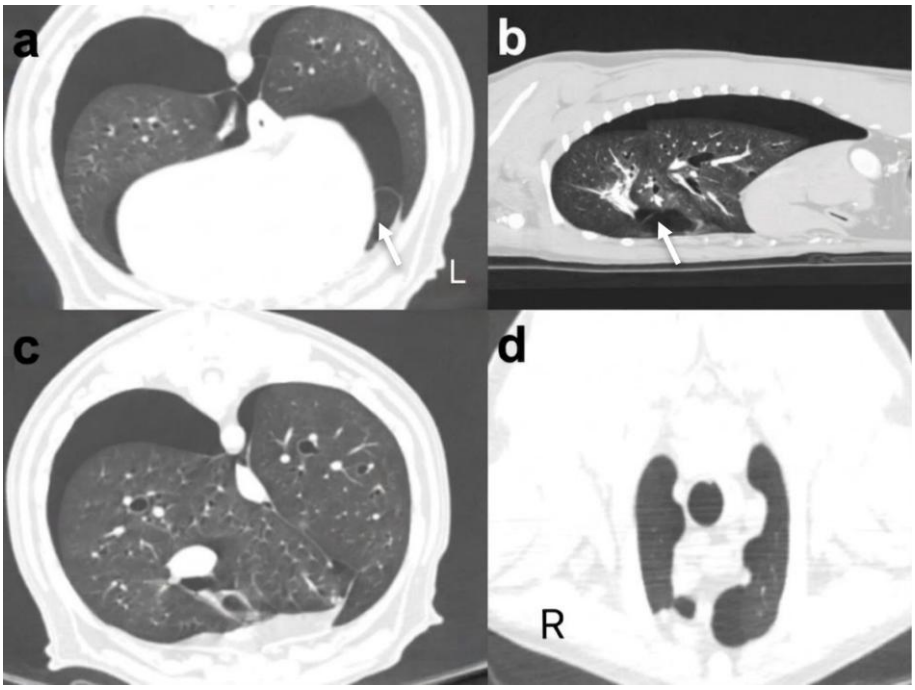


Figure 2 Thoracic computed tomography (CT) images of a canine patient. (a) Transverse axial image showing a large, thin-walled, air-filled bulla in the right caudal lung lobe; significant pneumothorax (arrow) is evident, with free air in the pleural space causing partial collapse (atelectasis) of the surrounding lung lobes and displacement of the cardiac silhouette. (b) Sagittal CT reconstruction of the thorax demonstrating a large, thin-walled bulla in the ventral aspect of the right middle lung lobe (arrow), with extensive dorsal pneumothorax causing ventral displacement and compression of the lung parenchyma. (c) Transverse axial image showing multiple thin-walled, air-filled bullae in accessory lung lobes; bilateral pneumothorax is present, with free air in the pleural space resulting in ventral displacement and partial collapse of the affected lung lobes. (d) CT image showing a bulla in the right cranial lung.



Figure 3 An autologous blood patch pleurodesis (ABPP) is performed on an anesthetized male Siberian husky. Collection of whole blood from the jugular vein and infusion of whole blood into the thoracic cavity.

Results and Discussion

In this case, PSP was likely based on the dog's signalment, acute respiratory distress, and lack of trauma or pre-existing pulmonary disease. Clinical signs of PSP vary from lethargy and anorexia to cyanosis and exercise intolerance, depending on air accumulation speed (Gilday *et al.*, 2021a). Siberian Huskies are predisposed to spontaneous pulmonary bulla rupture, making up a large part of 36%–68% of canine spontaneous pneumothorax cases (Puerto *et al.*, 2002; Gilday *et al.*, 2021a). Thoracic radiography confirmed pleural air and a restrictive breathing pattern, but CT was needed to identify multifocal bullae and pneumomediastinum (Gilday *et al.*, 2021a). The CT showed multifocal involvement in the left caudal, right middle, and accessory lobes, which explains why the air leak persisted despite conservative treatment. Since multifocal disease often requires more extensive intervention, this information is vital for surgical planning and prognosis. Initial stabilization with thoracocentesis led to immediate improvement, but recurrence within 48 hours showed the pulmonary defect was still active. While thoracocentesis is simple and effective for acute issues, failure to resolve the leak in two to five days with suction indicates medical management failure (Gilday *et al.*, 2021b). In this case, despite thoracocentesis and intermittent evacuation of pleural air, persistent air leakage and recurrent pneumothorax were observed for ten days. Because the pneumothorax failed to resolve despite conventional management for ten days, it was considered refractory (persistent), consistent with previous veterinary reports defining persistent pneumothorax as failure to respond to conservative management for ≥2–3 days (Oppenheimer *et al.*, 2014; Théron *et al.*, 2021). Since surgery was not performed, bilateral thoracostomy tubes were used to drain air and improve comfort, though long-term use increases risks (Lynch and Campos, 2018). Prolonged thoracostomy tube placement may have contributed to local inflammation, bacterial contamination, and secondary pleural infection. Although thoracostomy tubes are a vital bridge to surgery or pleurodesis, delaying surgery often leads to longer hospital stays, higher costs, and more infections (Boullhesen Williams *et al.*, 2022). Autologous blood patch pleurodesis (ABPP) was chosen as salvage therapy for persistent air leakage after conservative management failed. It works by promoting clot formation over the leaking pleura, then inducing local inflammation and permanent adhesion (Oppenheimer *et al.*, 2014). Previous reports have described ABPP as a treatment option for persistent pneumothorax in dogs, particularly when conventional management does not achieve resolution (Oppenheimer *et al.*, 2014). In the present case, ABPP was associated with progressive resolution of the pneumothorax and sustained lung re-expansion, with no recurrence during the four-month follow-up period. These findings are favorable when considered in the context of previous reports; however, surgical treatment remains associated with lower recurrence rates than nonsurgical management. In a retrospective study of 64 dogs with spontaneous pneumothorax, recurrence occurred in 1 of 30 dogs (3.3%) treated surgically compared with 6 of 12 dogs (50%) managed without surgery (Puerto *et al.*, 2002). Therefore, although the outcome in the present case suggests that ABPP may provide effective long-term control in selected cases, its success should not be interpreted as equivalent to that of definitive surgical treatment. Rather, ABPP may serve as a salvage option when surgery is not feasible, particularly in cases with multifocal pulmonary lesions or other limitations to surgical intervention. However, in this case, ABPP was followed by the subsequent development of culture-confirmed bacterial pyothorax during prolonged thoracic management. Although follow-up CT was not performed after ABPP, post-treatment CT could show sustained lung expansion and no recurrent pneumothorax, supporting successful pleurodesis, as seen in human studies (Quinones-Gonzalez *et al.*, 2025). While ABPP is often more affordable than surgery, clinicians must consider the potential for longer hospitalization and cumulative costs.

The development of pyothorax later in illness is a significant complication. At initial presentation, there was no marked leukocytosis or fever, whereas inflammatory changes became evident later in the clinical course, including leukopenia, neutropenia, and increased CRP at the time of pyothorax. Cytologic findings of neutrophilic inflammation and intracellular bacteria align with descriptions of canine pyothorax (Johnson *et al.*, 2023). The focal pulmonary infiltrative changes identified on the initial CT were considered nonspecific, as infection could not be confirmed at that stage. Differential considerations included infectious inflammation, sterile inflammatory changes, and pulmonary changes associated with the underlying pneumothorax or thoracic instrumentation. Given the timing and prolonged thoracic instrumentation, the pyothorax was considered more likely to be associated with the thoracostomy tubes than with the pulmonary infiltrative changes identified on the initial CT. Pyothorax developed 10 days post-procedure following bilateral chest tube insertion and ABPP. Prior studies have linked thoracostomy tube use to infectious risks (Boullhesen Williams *et al.*, 2022). Notably, experimental research in healthy dogs demonstrated that long-term indwelling tubes induced septic suppurative pleuritis in 75% of cases (6/8 dogs), with pyothorax typically emerging between days 4 and 6 (Hung *et al.*, 2016). These data imply that extended thoracic drainage likely increased infection susceptibility in this dog. Nevertheless, because ABPP was administered alongside tube placement, isolating the precise clinical impact of each intervention remains challenging. However, because thoracostomy tube placement and ABPP were performed on the same day, it was not possible to determine whether either procedure contributed independently to the subsequent infection. Infectious complications have also been reported following ABPP in dogs, including hospital-acquired infection with *Acinetobacter spp.* (Oppenheimer *et al.*, 2014), although a direct causal relationship with the procedure has not been established. Therefore, the infection in this dog may

have been associated with prolonged thoracic instrumentation, hospitalization, ABPP, or a combination of these factors.

The presence of *Acinetobacter baumannii* heightened suspicion of a nosocomial origin, given its role as a typical hospital-associated pathogen. Based on bacterial culture and antimicrobial susceptibility testing, marbofloxacin (5.5 mg/kg/day) was initiated, with a recommended treatment duration of 5–10 days. The dog showed clear clinical improvement. These findings emphasize the importance of vigilant post-procedure monitoring, particularly evaluating changes in pleural fluid or systemic inflammatory markers for canine patients managed with prolonged chest tubes or blood patch pleurodesis.

This case report details the management of spontaneous pneumothorax in a young Siberian Husky with pulmonary bullae confirmed by CT. While lung lobectomy is standard for persistent air leaks, this case shows that conservative management combined with autologous blood patch pleurodesis can resolve the condition if surgery is not an option. A key finding was the development of *Acinetobacter baumannii* pyothorax, highlighting the potential infectious risks associated with prolonged thoracic instrumentation and intrapleural intervention. Despite this complication, the patient had a favorable long-term outcome with culture-directed antimicrobial therapy, with no recurrence during a four-month follow-up. Ultimately, this case demonstrates that ABPP may be a useful salvage option for refractory spontaneous pneumothorax when definitive surgical intervention is not feasible. The subsequent development of *A. baumannii*-associated pyothorax highlights the importance of strict aseptic technique and vigilant monitoring for infectious complications during prolonged thoracic instrumentation.

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Declaration of artificial intelligence use: The authors used Gemini (Gemini 1.5 Pro, Google) exclusively for language editing, professional vocabulary enhancement, and grammatical refinement of this case report. The documentation of the patient's clinical history, diagnostic findings, and discussion was executed entirely by the authors. The authors have meticulously verified all content and assume ultimate responsibility for the academic integrity of this work.

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