Case Report

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Nonsurgical pneumoperitoneum in a dog secondary to blunt force trauma to the chest

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Abstract

Objective – To describe the medical management of pneumoperitoneum without surgical intervention in a dog that sustained blunt force trauma to the thorax. To review the mechanisms of how a thoracic injury (ie, extra-abdominal source) can lead to pneumoperitoneum.
Case Summary – A 4-month-old Shih Tzu puppy was attacked by a larger dog and sustained various injuries including a pneumothorax, pneumomediastinum, and a pneumoperitoneum. The dog presented minimally responsive and in respiratory distress secondary to pulmonary contusions and noncardiogenic pulmonary edema. No penetrating wounds to the abdomen or thorax were identified. As no immediate surgical lesion was identified the dog was treated conservatively without the need for surgical intervention. The dog was successfully managed and discharged after a few days of supportive care with oxygen therapy. Before discharge, repeat radiographs revealed complete resolution of the pneumothorax, pneumomediastinum, and pneumoperitoneum.
New or unique information provided – Cases of nonsurgical pneumoperitoneum have rarely been reported in the veterinary literature. A thoracic source of pneumoperitoneum should be considered when the suspicion of a ruptured viscus is low based on diagnostic procedures (eg, ultrasound, computed tomography, and diagnostic peritoneal lavage), in addition to physical examination (eg, lack of fever and absence of abdominal pain) and laboratory findings (eg, absence of inflammatory leukogram).

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Introduction

Pneumoperitoneum, or free peritoneal air, is often associated with a gastrointestinal tract rupture in veterinary medicine.[1,2] However, many different etiologies for pneumoperitoneum have been reported and are often placed in the category of spontaneous or traumatic in origin. For the majority of veterinary cases, immediate surgical intervention is recommended.[1,2] To the authors' knowledge, nonsurgical pneumoperitoneum secondary to blunt force trauma to the chest has not been reported in veterinary medicine. An awareness of nonsurgical pneumoperitoneum that derives from a pneumomediastinum or a pneumothorax is important as it may reduce the need for an unnecessary emergency laparotomy, particularly in patients with life-threatening comorbidities. The awareness of a thoracic cause of pneumoperitoneum may be especially important in veterinary medicine since the financial implication of surgical intervention alone has the potential to lead to the decision of euthanasia by the client. In people, nonsurgical pneumoperitoneum is considered for any patient without historical or diagnostic evidence of a perforated abdominal viscus.[3] This case report describes successful conservative management of a young dog that sustained blunt trauma to the chest with radiographic evidence of a pneumoperitoneum. The mechanisms by which a nonsurgical pneumoperitoneum can occur from a thoracic source of free air secondary to trauma are discussed, as well as a review of the pertinent human literature.

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Case Summary

A 4-month-old, 3.3 kg intact male Shih Tzu presented to a private referral hospital for difficulty breathing after the owners witnessed the dog being attacked by a larger dog 30 minutes before arrival. The dog was pinned down on his back while attempting to rise, as the attacking dog seemed to be biting over the patient's body. Soon after

this incident, the patient became limp and developed respiratory distress.

At triage, the dog had a depressed mentation, moderate to marked respiratory effort, and tachypnea at 68/min. The dog was brought to the critical care unit and provided flow-by oxygen via a face mask. With the mask in place, the initial oxygen saturation via pulse oximetry was 95%. He was nonambulatory and too weak to stand. He had pale mucous membranes with a heart rate of 124/min and a systolic blood pressure measurement of 78 mm Hg via Doppler. Lung sounds were harsh bilaterally and no heart murmur was auscultated. The abdomen was distended with mild tenderness on palpation. No areas of bruising, bleeding or outward signs of trauma were identified on the thorax or abdomen, even after the haircoat was clipped to ensure the absence of puncture wounds. Intravenous access was obtained and he was administered 4 mL/kg hypertonic saline[a] and subsequently 4 mL/kg synthetic colloid[b] IV over 30 minutes. After resuscitative IV fluids, the systolic blood pressure improved to 130 mm Hg. Furosemide[c] was administered at 1 mg/kg IV once due to the patient's respiratory distress and concern of noncardiogenic pulmonary edema (NCPE). An initial minimum database was obtained and revealed a PCV of 34%, reference interval, 28–46% and total plasma protein of 50 g/L, reference interval 45–73 g/L [5.0 g/dL; reference interval, 4.5–7.3 g/dL]. Abnormalities on a venous blood panel[d] included a respiratory acidosis (pH 7.172; reference interval, 7.344–7.441; pCO_2 58.9 mm Hg; reference interval, 32.6–48.3 mm Hg), hyponatremia (141 mmol/L, reference interval 142–149.3 mmol/L [141 Eq/L, reference range, 142-149.3 mEq/L]), hypochloridemia (108.5 mmol/L, reference interval 112.7–118.3 mmol/L [108.5 mEq/L, reference interval 112.7-118.3 mEq/L]), hyperglycemia (11.2 mmol/L, reference interval 4.1–6.4 mmol/L [202 mg/dL, reference interval, 75–116 mg/dL]), an increased BUN (42 mg/dL; reference interval, 8–30 mg/dL), and hyperlactatemia (10.9 mmol/L, reference interval, 0.7–2.8 mmol/L). A complete blood count (CBC) and serum biochemistry profile were submitted for analysis. An arterial blood gas was attempted, but could not be obtained due to the patient's respiratory distress and small size. The respiratory distress also precluded the ability to perform a complete thoracic focused assessment with sonography for trauma (TFAST).[4] However, a brief ultrasound (US) of the thorax did not reveal any pleural fluid. The abdomen was evaluated via the focused assessment with sonography for trauma (AFAST)[5,6] method in both right and left lateral recumbency. Four views were evaluated according to previous FAST recommendations: diaphragmatic-hepatic, splenorenal, cysto-colic, and the hepato-renal.[5,6] No free peritoneal fluid was noted and the urinary bladder seemed intact. Right and left lateral, as well as ventrodorsal radiographs revealed a small pneumothorax with a multifocal, mixed unstructured interstitial coalescing to alveolar pattern most severe in the caudodorsal lung, consistent with NCPE. An alveolar pattern was also noted, consistent with bilateral pulmonary contusions, in the right middle and left caudal lung lobes. A small volume of free peritoneal air outlined the abdominal aspect of the diaphragm, consistent with a mild pneumoperitoneum. No subcutaneous emphysema was noted. A constant rate infusion (CRI) of furosemide was initiated at 0.1 mg/kg/h and was administered over the next 12 hours. The dog was maintained in an oxygen cage with a FiO_2 of 40%. Hydromorphone[e] 0.05 mg/kg IV was administered every 6 hours. One and a half hours after presentation and resuscitative IV fluids, a recheck venous blood sample revealed continued, yet improved, respiratory acidosis (pH 7.313; reference interval, 7.344–7.441; pCO_2 53.3 mm Hg, reference interval, 32.6–48.3 mm Hg), hyperglycemia (6.9 mmol/L, reference interval 4.2–6.4 mmol/L [124 mg/dL, reference interval, 75–116 mg/dL]), and an increased BUN (11.1 mmol/L, reference interval 2.9–10.7 mmol/L [31 mg/dL, reference range, 8–30 mg/dL]). The lactate (<2.8 mmol/L) concentration was within reference interval at this time. The CBC obtained at admission was within the reference interval. Serum biochemistry abnormalities included an increased aspartate aminotransferase (AST) (1,059 U/L; reference range, 13–50 U/L) and an increased alanine aminotransferase (ALT) (1,224 U/L; reference range, 21–97 U/L). Thoracic and abdominal radiographs were repeated approximately 3 hours after admission, which showed minimal improvement in the alveolar pattern and a scant persistent pneumoperitoneum. Serial monitoring of the thorax and abdomen overnight every 4 hours via US continued to demonstrate the absence of free pleural or peritoneal fluid. Although the patient's presenting respiratory effort raised concerns about the possible development of respiratory fatigue, the breathing pattern normalized within 12 hours of presentation. Pulse oximetry readings ranged from 90% to 94% on room air over the next 24 hours. The dog continued to receive oxygen due to persistent hypoxemia.

Approximately 14 hours after presentation, a more comprehensive diagnostic abdominal US was performed and no free fluid, free air, or parenchymal lesions were detected. Concurrently, a single thoracic radiograph was obtained and revealed improvement, but not resolution of the NCPE and pulmonary contusions. On this radiograph, only a mild pneumomediastinum was present. The pneumoperitoneum was not identified on subsequent radiographs since the day of presentation. About 12 hours after admission, the patient regained his

appetite. Over the next 2 days the dog continued to improve clinically. Oxygen therapy was discontinued on the third day of hospitalization. Thoracic radiographs obtained 4 days after presentation revealed complete radiographic resolution of the NCPE, pulmonary contusions, and the pneumomediastinum. The dog was discharged from the hospital with no medications or further treatment recommendations.

Discussion

Pneumoperitoneum has been associated with a number of conditions in the veterinary literature. Iatrogenic causes of pneumoperitoneum are most often related to recent invasive procedures, such as a celiotomy, laparoscopy, or abdominocentesis, after which free peritoneal air is an expected finding. Few veterinary reports have evaluated the variables that influence the duration of pneumoperitoneum; however, one report in dogs cited 9–25 days for radiographic resolution of pneumoperitoneum, depending on the amount (mL/kg) of air experimentally introduced into the abdomen.[7] Another report illustrated the progressive resolution of pneumoperitoneum postoperatively using US monitoring, with most patients having complete resolution by day 22.[8]

The source of a pathologic pneumoperitoneum can often be grouped into 2 categories: spontaneous or traumatic. Retrospective studies have shown that the most common etiology for a spontaneous pneumoperitoneum is gastrointestinal tract perforation; the 2 most frequent causes being secondary to gastric dilatation and volvulus (GDV) and gastrointestinal neoplasia.[1,2] Traumatic causes of pneumoperitoneum are most often associated with body wall penetration, such as dog bite wounds, gun shot wounds or motor vehicle accidents.[1] In veterinary medicine, the treatment recommendation for pneumoperitoneum is immediate surgical intervention, regardless of the underlying etiology.[1,2] Early surgical intervention is intended to correct the source of air leakage and reduce the magnitude of enteric contamination of the peritoneal space.[9] In contrast, 15% of pneumoperitoneum cases in people have a benign etiology and do not require emergency surgery.[3] Conservative, nonsurgical treatment is indicated in human medicine when serial evaluations of abdominal pain and distension are minimal and no signs of intra-abdominal injury are found via diagnostics. These diagnostics may include a diagnostic peritoneal lavage (DPL), abdominal US, or computed tomography (CT).[3,9]

Two potential mechanisms for the passage of air from the thoracic to the abdominal cavity have been proposed in human reports: (1) the direct passage of air through pleural and diaphragmatic defects, natural microscopic fenestrations, or the epiploic foramen, and (2) the indirect passage of air, via the mediastinum, along perivascular sheaths or major diaphragmatic portals, such as the aortic and esophageal hiatus, to the retroperitoneum and finally into the peritoneum.[3,9–11] When the bronchioles, alveolar ducts, and alveoli are overdistended by an increase in intrathoracic pressure, air collections leak into the perivascular tissues, which then migrate along a path of least resistance.[12] Pneumomediastinum occurs secondary to lung trauma when excessive intraalveolar pressures lead to rupture of perivascular alveoli. Subsequently, air escapes into the perivascular connective tissue, with subsequent dissection into the mediastinum.[3,9,13,14]

The human literature has placed the etiology of nonsurgical pneumoperitoneum into 5 categories: thoracic, intraabdominal, gynecological, iatrogenic, and miscellaneous.[3,9,10,13,15] Thoracic causes of nonsurgical pneumoperitoneum in people have been associated with cardiopulmonary resuscitation (CPR), intermittent positive-pressure ventilation, barotrauma, pneumothorax, increased intrathoracic pressure, asthma, blunt trauma, spontaneous rupture of pulmonary blebs, and severe pulmonary disease.[3,9–11,13,14,16–18] Abdominal pressure usually exceeds intrathoracic pressure by 20–30 cm H_2O during both inspiration and expiration in people. However, intrathoracic pressures over 50 cm H_2O can result in a pneumoperitoneum.[15] An increase in intrathoracic and intraalveolar pressures can occur in the previously mentioned conditions, while noncompliant lungs and preexisting pulmonary disease may further increase the risk of a nonsurgical pneumoperitoneum.[9] It is suggested that free air in the abdomen post-CPR is due to a combination of increased airway pressures and the application of blunt force trauma to the chest.[13] In people, ventilated, pediatric population, nonsurgical pneumoperitoneum can occur in up to 3% of the population with a concurrent pneumomediastinum or pneumothorax.[9] Two human pediatric reports propose that an analysis of the partial pressure of oxygen (PaO_2) in the intraperitoneal free gas obtained via abdominal paracentesis be compared to the PaO_2 delivered by the ventilator. If the value of the intraperitoneal PaO_2, obtained via a pH blood gas analyzer, is in agreement with the PaO_2 provided by the ventilator, or if the intraperitoneal PaO_2 exceeds the PaO_2 of room air (ie, swallowed air), the authors suggest this may provide evidence of a thoracic source of pneumoperitoneum, in contrast to a ruptured viscous.[19,20] If a human neonate develops a pneumomediastinum while receiving mechanical ventilation, the risk of nonsurgical pneumoperitoneum increases 10-fold.[3]

For people with an exclusive thoracic source of pneumoperitoneum, nonsurgical treatment is recommended.

In the human literature, an algorithm for the evaluation and treatment of pneumoperitoneum has been published and the decision for surgical intervention is not based solely upon the radiographic findings.[9,10] Historically, treatment decisions for nonsurgical pneumoperitoneum have been based upon the patient's history, serial abdominal examinations, lack of peritoneal signs, presence of a pneumothorax or pneumomediastinum, negative DPL, gastrointestinal swallow series, lack of peritoneal fluid on US and CT scan, the white blood cell count and the temperature of the patient, or combinations thereof.[3,9,10] Emergency laparotomy is performed on hemodynamically unstable trauma patients with the suspicion of intraabdominal injury. In hemodynamically stable human blunt or penetrating trauma patients, the most sensitive and specific diagnostic procedures to detect intraabdominal injury remain controversial and challenging to interpret.[21,22] In blunt trauma patients, CT findings of pneumoperitoneum are not pathognomonic for a ruptured viscus; this was especially true in patients with a pneumothorax.[23–26] Although CT is widely accepted as the gold standard for the diagnosis of intraabdominal injury, sonography (FAST) is often recommended as the first step in the imaging assessment of patients with blunt abdominal trauma; it is even considered the gold standard in some reports.[21,27] In penetrating abdominal injuries, however, the FAST exam is less reliable (variable sensitivity, high specificity) in detecting intraabdominal injury versus blunt trauma (high sensitivity, high specificity).[22,28] Consequently, in hemodynamically stable patients with penetrating trauma and a negative AFAST exam, further investigation for intraabdominal injury via additional diagnostic studies, such as a CT scan, is recommended.[22] A positive FAST exam in penetrating trauma warrants an emergent laparotomy because serious intraabdominal injury is commonly present.[22,28]

Compared to human medicine, there are significantly fewer studies regarding diagnostic modalities for the detection of intrathoracic or intraabdominal injury in traumatized veterinary patients. Human and veterinary reports have shown that physical examination and bloodwork findings in trauma patients are unreliable and insensitive methods for detecting thoracic and intraabdominal injury.[27,29,30] Three recent studies report the clinical utility of sonography via the FAST method in traumatized patients for the early and rapid diagnosis of a pneumothorax and other thoracic injuries (TFAST),[4] as well as free abdominal fluid (AFAST) with an abdominal fluid scoring (AFS) system.[5,6] Lisciandro et al.[4] found that in veterinary trauma patients, TFAST offered a radiographic alternative at triage for the early detection of pneumothorax, and other thoracic injuries, before thoracic radiography can be performed.[4] This expediency is especially useful in respiratory compromised patients that may deteriorate with restraint or transport to radiology. It is also recommended that patients in respiratory distress have TFAST performed in sternal recumbency rather than lateral recumbency; the latter is recommended for the AFAST evaluation. In another report by Lisciandro et al.,[6] an AFS system allows rapid and serial measurements of the volume of free abdominal fluid, which aids in the assessment of the severity of injury and helps to guide case management. This study also revealed that AFAST was more clinically reliable than abdominal radiography for the detection of intraabdominal injury in bluntly traumatized veterinary patients.[6] In nontraumatized veterinary patients, US has been shown to detect free air in 47.8% of patients and free peritoneal fluid in 84.2% of patients with a confirmed gastrointestinal perforation.[31] Computed tomography scanning may be limited in traumatized patients due to availability, cost, and patient safety secondary to the need for general anesthesia.

The clinical course of the patient described in the current report correlates with that of a nonsurgical pneumoperitoneum case in human medicine with a nonsurgical pneumoperitoneum and illustrates a case in which a pneumoperitoneum counters the presumption of a perforated hollow viscus or traumatic penetration of the abdomen with the need for immediate surgical intervention. The source of the pneumoperitoneum in this case was likely thoracic in origin and secondary to blunt trauma to the chest. Increased intrathoracic pressures secondary to a larger dog biting over the patient's thorax and being pinned down may have enabled a mechanism for free air introduction into the abdomen similar to that which occurs in people after CPR. Radiographic signs supporting these mechanisms of injury and a thoracic cause of a pneumoperitoneum include a combination of pulmonary contusions, NCPE, a mild pneumothorax and a mild pneumomediastinum. Noncardiogenic pulmonary edema in people and veterinary medicine has been associated with thoracic trauma and upper airway obstruction.[32–34] Further supporting a thoracic etiology of the pneumoperitoneum, puppies are possibly at increased risk for direct pulmonary parenchymal injury and increased intrathoracic pressures secondary to chest trauma due to the relative flexibility of the chest wall preventing the absorption of the impact.[35]

Noncardiogenic pulmonary edema is due to an increase in endothelial permeability causing leakage of a high protein fluid into the pulmonary interstitium. This is in contrast to edema formation secondary to an increased hydrostatic pressure, as is observed in conditions of fluid overload and left-sided heart failure.[36] Mechanisms leading to the development of NCPE include barotrauma, inflammation, drowning, smoke inhalation,

pulmonary thromboembolism, and cisplatin administration in cats.[37] The use of diuretic administration, such as furosemide, for cases of increased permeability edema remains controversial. However, some authors note that increased permeability edema may be coupled with hemodynamic changes that increase transpulmonary blood volume and the subsequent increase in hydrostatic pressure will affect net fluid flux.[34,37] Although the clinical benefits are not well established, some human and veterinary authors recommend considering furosemide administration in patients affected with NCPE.[32,38–40]

The nondiuretic benefits of furosemide have been studied as an adjunct therapy for acute lung injury (ALI) and include improvements in pulmonary gas exchange, intrapulmonary shunt fraction, and lung injury scores.[41] In addition, human and veterinary studies have also shown increased diuresis with furosemide administration as a CRI versus intermittent boluses, presumably from less rebound sodium and water retention.[41–44] Due to the severity of respiratory distress secondary to the NCPE and pulmonary contusions, this dog was placed on a low-dose furosemide CRI. The furosemide was discontinued after about 12 hours when the tachypnea improved and the puppy seemed more comfortable.

Although this dog demonstrated mild abdominal pain at presentation, which would provide evidence for the decision to perform a laparotomy, the patient lacked obvious puncture wounds or outward evidence of trauma to the abdominal cavity, such as abdominal herniation or persistent abdominal pain. In addition, his respiratory distress and pulmonary pathology made him a poor surgical candidate. The abdominal pain resolved by the next morning and the puppy was improving clinically. The dog did not develop a fever and the CBC did not show any significant changes. Serial ultrasonographic evaluations of the abdomen did not reveal any free air or peritoneal fluid. Radiographs obtained 12 hours after presentation revealed resolution of the pneumoperitoneum. This correlates with human studies in which resolution of pneumoperitoneum that is thoracic in origin occurs within a few days.[14]

The dog's ALT and AST were significantly increased above reference interval. Dogs with a higher AFS were more likely to have marked increases in the ALT concentrations.[6] In hemodynamically stable children with blunt trauma, reports recommend performing a CT to detect intraabdominal injury only in negative FAST patients with predetermined cut-off ALT and AST values to avoid unnecessary radiation exposure.[45,46] The patient in this report would have met this criteria in favor of performing a CT. However, the serum biochemistry results were not known until about 16 hours after presentation and the pneumoperitoneum had resolved.

In conclusion, in the majority of veterinary patients, free abdominal air with no penetrating wounds and no recent history of surgery suggests a ruptured viscus as the source of air. The standard recommendation for treatment of a pneumoperitoneum is immediate surgical intervention to locate the origin of free air and reduce the amount of peritoneal contamination. In patients without historical or physical evidence of penetrating trauma, or in ones in which there is a low index of suspicion for a spontaneous pneumoperitoneum, a nonsurgical condition may be considered. Numerous studies have established that physical exam and bloodwork results are insensitive diagnostic tools to detect intraabdominal injury. Before concluding a case has a nonsurgical pneumoperitoneum, imaging, such as abdominal radiography, the utilization of AFAST with the AFS, a formal diagnostic US, and where available, a CT, should ideally be performed. A DPL can also be performed, however, this test adds free abdominal fluid and potentially introduces free air, which will confound the clinical picture and may negate the utility of imaging via an AFAST, a formal diagnostic US or CT. A DPL may also overlook retroperitoneal injuries. When possible, the utilization of serial AFAST exams versus a DPL will better aid in the treatment decisions of hemodynamically stable bluntly traumatized patients.

If these diagnostic tools are not available to the veterinary practitioner, the risk of a negative abdominal explore will lead to less morbidity than incorrectly selecting conservative management. However, a greater awareness of the potential for a nonsurgical pneumoperitoneum in veterinary patients and subsequent conservative management may help avoid unwarranted surgical exploration and the associated increased morbidity or mortality, particularly when other significant abnormalities are present in other body systems. In the future, the development of an algorithm, similar those used in human medicine, may guide treatment decisions in blunt trauma veterinary patients with a pneumoperitoneum.

Footnotes

[a] Equi-Phar Equine 7 HSS, Vedco Inc, St. Joseph, MO.
[b] Hespan, Braun Medical Inc, Irvine, CA.
[c] Furosemide, Butler Animal Health Supply, Dublin, OH.
[d] Critical Care Xpress, Nova Biomedical Corporation, Waltham, MA.
[e] Hydromorphone, Baxter Healthcare Corporation, Deerfield, IL.

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