Left cranial lung torsion in a bernese mountain dog: a case report

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ABSTRACT: Lung lobe torsion (LLT) is an uncommon pathology in small animal practice. In large breed dogs LLT effects are localized mainly to the middle lobe of the right lung. This report describes a case of left cranial lung torsion in a Bernese Mountain dog: the patient was referred with a two-day history of asthenia, anorexia, dyspnea and haemoptysis. No trauma was reported. Physical examination showed the presence of bilateral pleural effusion confirmed by radiography and ultrasonography. Broncoscopy revealed that the left cranial lobe appeared to be compressed laterally with complete occlusion of the lumen. Thoracoscopy was performed to exclude other pathologies of the pleural space. The pathological lobe was removed. Histological examination revealed aspects consistent with lung lobe torsion. Clinical follow up carried out after three months showed a normal clinical course.

Keywords: lung; lobe; torsion; Bernese; dog

Lung lobe torsion (LLT) is a rare disorder in veterinary medicine which has been described in dogs and cats, and also in humans (Moses, 1980; Johnston et al., 1984; Fossum et al., 1986; Gallagher 1993; Gelzer et al., 1997; Dye et al., 1998; Neath et al., 2000; Rooney et al., 2001; White and Corzo-Menendez, 2001; Spranklin et al., 2003; Hofeling et al., 2004; Della Santa et al., 2006; Murphy and Brisson, 2006; Fossum, 2007). In LLT a lung lobe is rotated around its long axis, causing physical displacement and the twisting of the lobe around its bronchovascular pedicle (Gallagher, 1993); the consequence of an increase in lobar pressure results in pulmonary consolidation and pleural effusion (Neath et al., 2000).

Trauma, pleural space pathologies, parenchymal disorders and thoracic or abdominal surgery can result in an increase in lobar mobility (Johnston et al., 1984).

In large breed dogs, LLT effects are localized mainly to the middle lobe of the right lung while, in small breeds, the cranial lobe of the left lung is affected (D'anjou et al., 2005; Fossum, 2007).

Clinical features include dyspnea, anorexia, vomiting and diarrhoea (Neath et al., 2000). On physical examination, a decrease in heart and breath lung sounds is observed as a result of pleural effusion.

Radiography and ultrasonography are the initial diagnostic imaging techniques used to make a correct diagnosis (Siems et al., 1998; D'anjou et al, 2005; Della Santa et al., 2006). Radiographic findings, such as lobar consolidation or increased opacity of the thorax, are sometimes non-specific and other diagnostic techniques are required (D'anjou et al., 2005; Seiler et al., 2008). Bronchoscopy allows the visualization of the pathological bronchus which appears twisted and stenosed, containing mucohematic material (Schultz et al., 2009).

Computed tomography permits the visualization of pulmonary structures and any other thoracic pathologies (Seiler et al., 2008). Schultz et al. (2009) compared radiological findings with tomographic and virtual bronchoscopy images in four dogs and two cats affected by LLT. They concluded that the LLT-specific findings of vesicular emphysema and a proximally occluded bronchus were more easily recognised with CT and virtual bronchoscopy than using radiographs.

Surgical treatment is required for treatment of LTT; a thoracotomic approach provides a good visualization of the pathological lobe and facilitates its removal (Neath et al., 2000; White and Corzo-Menendez, 2001; Murphy and Brisson, 2006; Fossum, 2007).

Left cranial lobe torsion is a rare pathology in large breed dogs; for this reason, the aim of this report is to describe the clinical protocol and outcome of rare left lung torsion in a Bernese Mountain dog.

Case description

A seven-year-old male Bernese Mountain dog was referred because of a two-day history of asthenia, anorexia, dyspnea and haemoptysis.

No traumatic events or previous pathologies were reported by the owner.

Physical evaluation revealed a rectal temperature of 39.5 °C, tachycardia (160 beats per minute) and tachypnoea (60 breaths per minute) with an arduous abdominal respiration and the presence of blood in the mouth.

Thoracic ascultation and percussion suggested the presence of bilateral diffuse pleural effusion and revealed muffled respiratory and cardiac sounds.

A complete blood count (CBC) revealed neutrophilia (12.1 × 10^3/mm^3) thrombocytopenia (139 × 10^3/mm^3) and lymphopenia (387/mm^3). Serum biochemical analyses revealed slight hypokalemia (3.8 mEq/l), hypoproteinemia (5.33 mg/dl) and hypoalbuminemia (2.44 mg/dl). An increase in AST (69 IU/l) alkaline phosphates (349 IU/l) and CK (732 IU/l) was also noted.

Inflammatory markers, such as C-reactive protein (9.75 mg/dl) and fibrinogen (10.66 μg/ml) were elevated.

A complete coagulation profile was likewise determined: PT (6.1 s) and aPTT (10.9 s) were within the reference ranges.

D-dimers were increased (0.44 μg/ml), while ATIII was 74%.

X-ray examination, performed in lateral (right recumbency) and ventrodorsal view, showed diffused opacification of both hemithoraces and non-visualization of the heart; the lung lobes were dislocated in a dorso-caudal direction (Figure 1). Radiological differential diagnosis included pneumonia, neoplasia, pleural hemorrage, pyotohorax, foreign body, and an abscess.

The radiological findings were indicative of bilateral pleural effusion.

Ultrasonographic evaluation confirmed the presence of pleural effusion; the left cranial lobe showed areas of lobar atelectasis, and the presence of residual air was noted. No superficial nodular lesions were observed.

The ultrasonographic findings were indicative of cranial lobar pneumopathy of the left lung associated with bilateral pleural effusion with a corpuscular aspect and associated with the presence of fibrous bands.

Thoracocentesis in the 7th intercostal space, in the ventral 3rd of the height between the vertebrae and sternum, confirmed the presence of serosanguineous pleural effusion fluid. Cytological analysis of the fluid revealed the presence of red blood cells and numerous intact neutrophils.

The patient was hospitalized and stabilized using broad spectrum antibiotics: Ampicillin Sulbactam

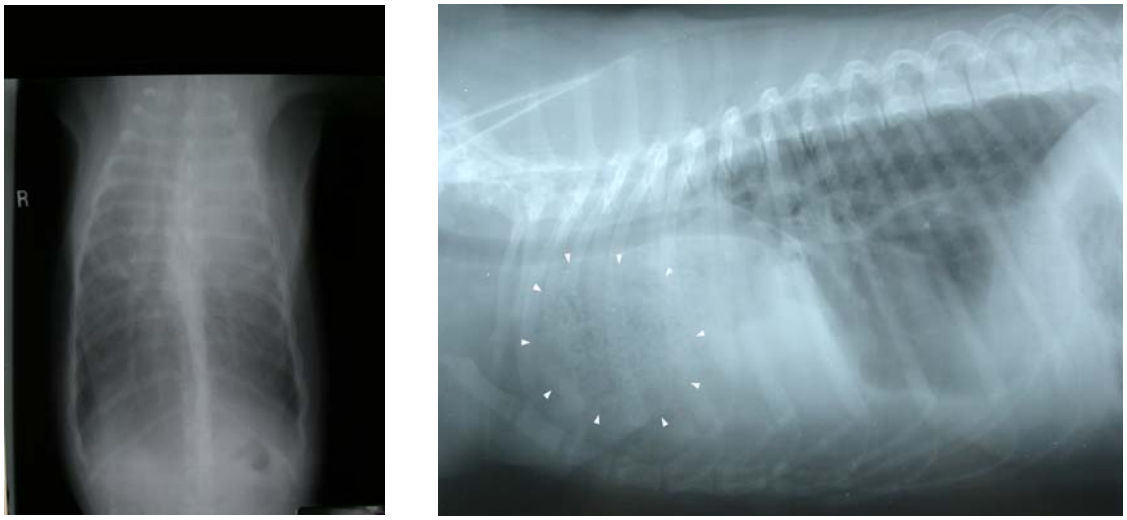


Figure 1. Ventro-dorsal and lateral (right recumbency) thoracic radiographs showing – severe pleural effusion – dorsally displaced and curved trachea – extensive lobar vesicular gas pattern in cranial pulmonary areas (arrows) – lobar consolidation – dorso-caudal displacement of the diaframmatic lobes

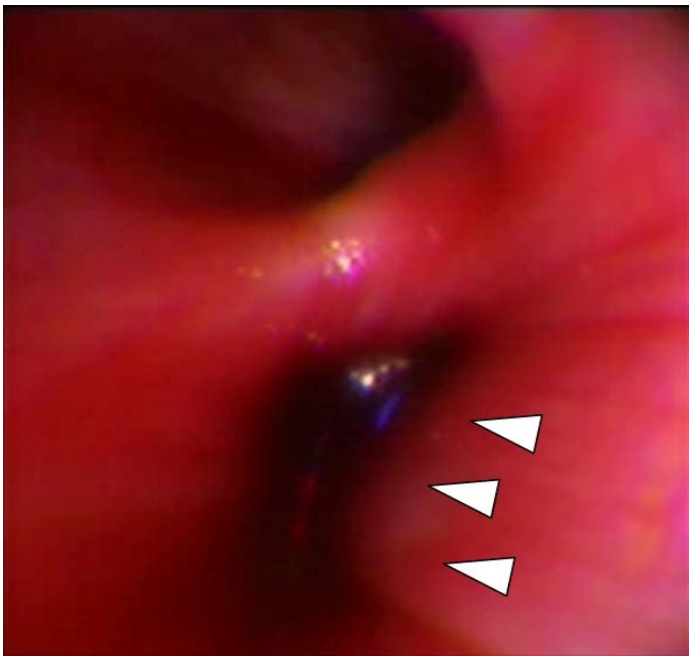


Figure 2. Broncoscophic evaluation; the left cranial lobe appeared compressed laterally with complete occlusion of the lumen (arrows)

20 mg/kg *i.v.* bid (Unasyn®, Pfizer, Italy) and Marbofloxacin 2 mg/kg *i.v.* sid (Marbocyl FD®, ATI, Italy) analgesia by methadone 0.2 mg/kg *i.m.* every four hours (Eptadone® Molteni Farmaceutici, Italy) and intravenous fluids.

On the second day, broncoscopy, a monolateral thorascopy and a thoracotomy were performed.

After premedication with constant-rate infusion of Fentanyl 2–4 µg/kg/h (Fentanest®, Pfizer, Italy) and Midazolam 0.1 mg/kg *i.m.* (Midazolam IBI, Lorenzini, Italy) general anaesthesia was induced with Propofol 2 mg/kg *i.v.* (Rapinovet®, Intervet, Italy)and maintained with isoflurane in oxygen. Lactated Ringer's solution was administered at 5 ml/kg/h during anaesthesia.

A tidal volume of 10 to 15 ml/kg at a frequency of 10 to 12 inspirations/minute was controlled with a volumetric ventilator.

The patient was monitored with an electrocardiogram, spirometer, pulse oximeter, capnograph and non invasive pressure.

A warm water blanket was used to maintain body temperature.

The broncoscopy (Pentax EG 1840 6 mm × 1050 mm) was performed in sternal recumbency after patient extubation; total intravenous anaesthesia was mantained using Propofol 2 mg/kg *i.v.* (Rapinovet®, Intervet, Italy) and oxygen was administered.

At the level of the trachea a streak of blood was noted coming from the left cranial lobe which ap-

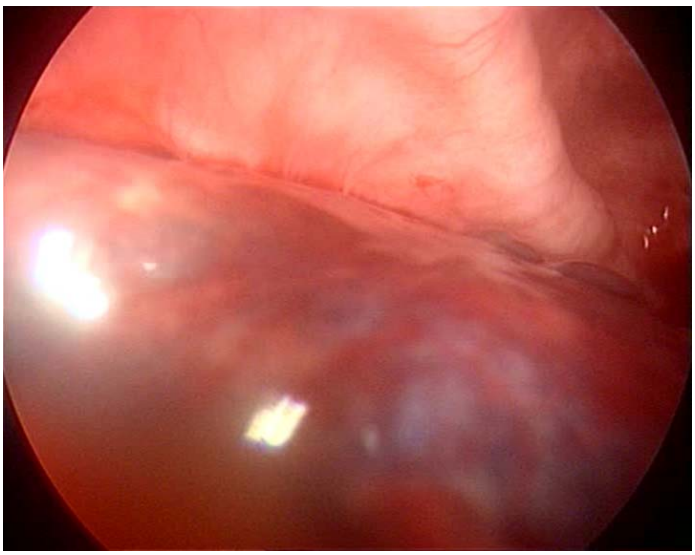


Figure 3. Thoracoscopy showed the cranial lobe of the left lung to be atelectastic and brownish

peared compressed laterally with complete occlusion of the lumen (Figure 2).

Bronchoscopic evaluation extended to the main right bronchus in right lateral recumbency. The endoscopic image of the remaining bronchi, with the exception of moderate hyperaemia of the mucosa, was within normal limits.

Thoracoscopy was performed in dorsal recumbency; a 10 mm 30° thoracoscope was introduced in the ninth intercostal space(IC), while the instrumental port was placed in the sixth IC space. The cranial lobe of the left lung appeared atelectasic and congested, and instrumental palpation showed a consolidation in comparison with the other lobes (Figure 3); pathologies of the pleural space or parenchyma were not seen. Minimal pleural effusion was observed.

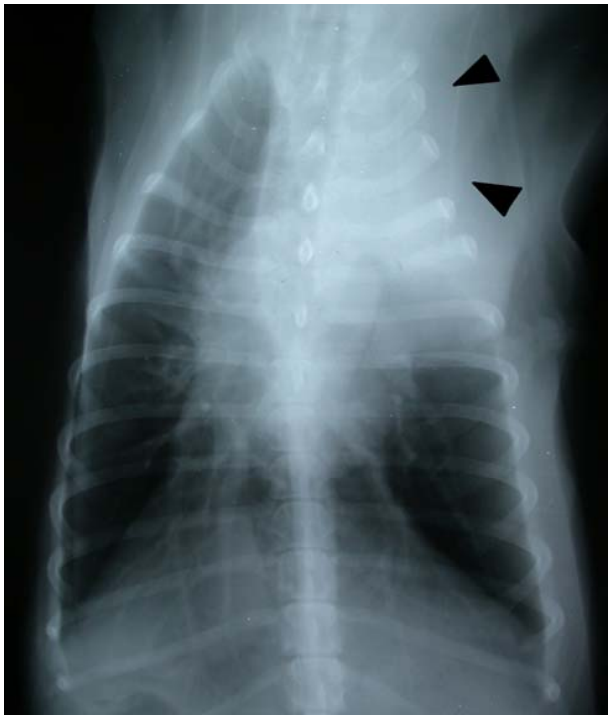
A fourth left intercostal thoracotomy was then performed. The damaged cranial lobe was retracted dorsally to visualize the untwisted pedicle; the pulmonary vessels supplying the affected lobe were ligated (2–0 absorbable monofilament suture) and transected; the main bronchus supplying the lobe was transected and sutured with preplaced interrupted horizontal mattress sutures (2–0 absorbable monofilament suture). The lobe was removed without detersing it and was submitted for histopathological evaluation. The bronchial suture line was tested for air leaks by flooding the thorax with warm saline solution.

A local anaesthetic intercostal nerve block (bupivacaine 1 mg/kg) was carried out and the thoracic wall was routinely closed. In addition, an active chest drain was placed.

Intensive care included oxygen support for two days, analgesia by methadone (Eptadone®, Zambon, Italy) 0.2 mg/kg *i.m.* every four hours for four days followed by Tramadol hydrochloride (Contrama®, Prodotti Formenti Srl, Italy) 3 mg/kg *i.m.* tid for seven days, antibiotic therapy with Ampicillin Sulbactam (Unasyn®, Pfizer. Italy) 20 mg/kg tid and anti-coagulant therapy (Eparina Vister®, Marvesc Pharma Services Srl Italy) 100 IU/kg bid.

Oxygen was removed 48 hours postoperatively and the thoracic drain 72 hours postoperatively. No radiographic abnormalities were noted except for a left fibrothorax (Figure 4); therefore, the dog was discharged one week after surgery.

Histological examination of the tissue removed revealed pulmonary necrosis with acute diffuse haemorrhage. Numerous red blood cells were present in the alveolar and interstitial spaces associated with acute oedema and, in some areas, with moderate fibrin formation (Figure 5). The other findings were focal vascular thrombosis and granulation tissue formation characterized by an inflammatory response (infiltration of macrophages and neutrophils) to necrosis-induced cellular damage (Figure 6).



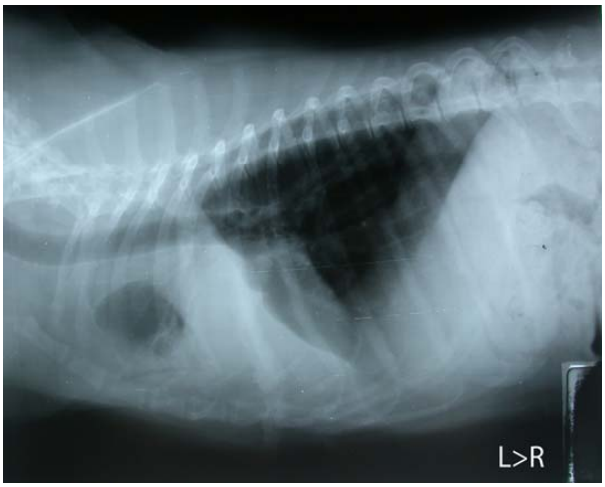


Figure 4. Ventro-dorsal and lateral (right recumbency) thoracic radiographs (seven days after surgery) showing: increased radiopacity of the cranial areas of the thorax – normal lobar radiopacity of the other lobes. The radiographic finding was indicative of a left fibrotorax

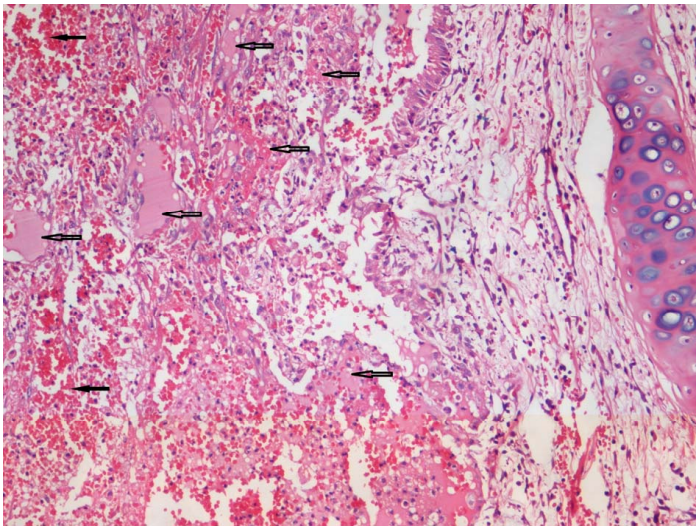


Figure 5. Lung. Histological section: pulmonary necrosis with acute diffuse haemorrhage (black arrows). Numerous red blood cells were present in the alveolar and interstitial spaces associated with acute oedema and, in some areas, with moderate fibrin formation (empty arrows); haematoxylin and eosin, ×100

Clinical follow up was carried out after three and 12 months; clinical, haematological and radiographic evaluation (Figure 7) showed a normal clinical course.

DISCUSSION AND CONCLUSIONS

LLT can be either a primary disorder leading to pleural effusion, or a secondary disorder caused by a predisposing condition (Neath et al., 2000).

The anatomic contiguity of the intrathoracic structures is responsible for lobar stability (Rooney et al., 2001).

For these reasons, torsion is always associated with the increased mobility of a lung lobe (Fossum, 2007).

Any intrathoracic disorder can cause an increase in lobar mobility and any thoracic pathology or trauma can change the spatial relationship of the organs and predispose to torsion.

Mobility alteration is also associated with iatrogenic causes such as surgical manipulation during thoracic or abdominal surgery leading to lobar torsion.

In large breeds it has been described that the primary localization of lung torsion is the middle right lobe, particularly in the Afghan hound, where the interlobar fissures are more pronounced, causing a greater possibility of torsion (D'anjou et al., 2005; Fossum, 2007).

In this report, it was shown that lung lobe torsion involved the cranial lobe of the left lung, which is typically seen in small breed dogs (D'anjou et al., 2005; Fossum, 2007).

The subject showed no signs of trauma; in addition no thoracic or abdominal surgery was performed; in addition, no abnormality was present to account for the lesions. For these reasons, we

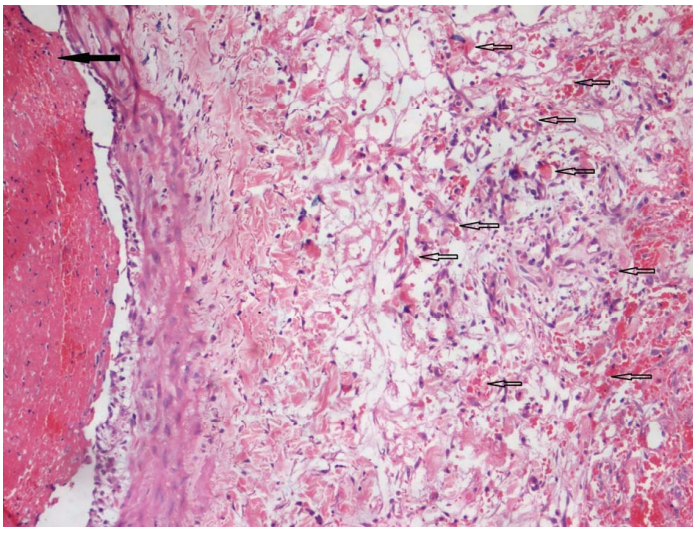


Figure 6. Lung. Histological section: focal vascular thrombosis (black arrow) and granulation tissue formation (empty arrows) characterized by an inflammatory response (infiltration of macrophages and neutrophils) to sites of necrosis-induced cellular damage; haematoxylin and eosin, ×100

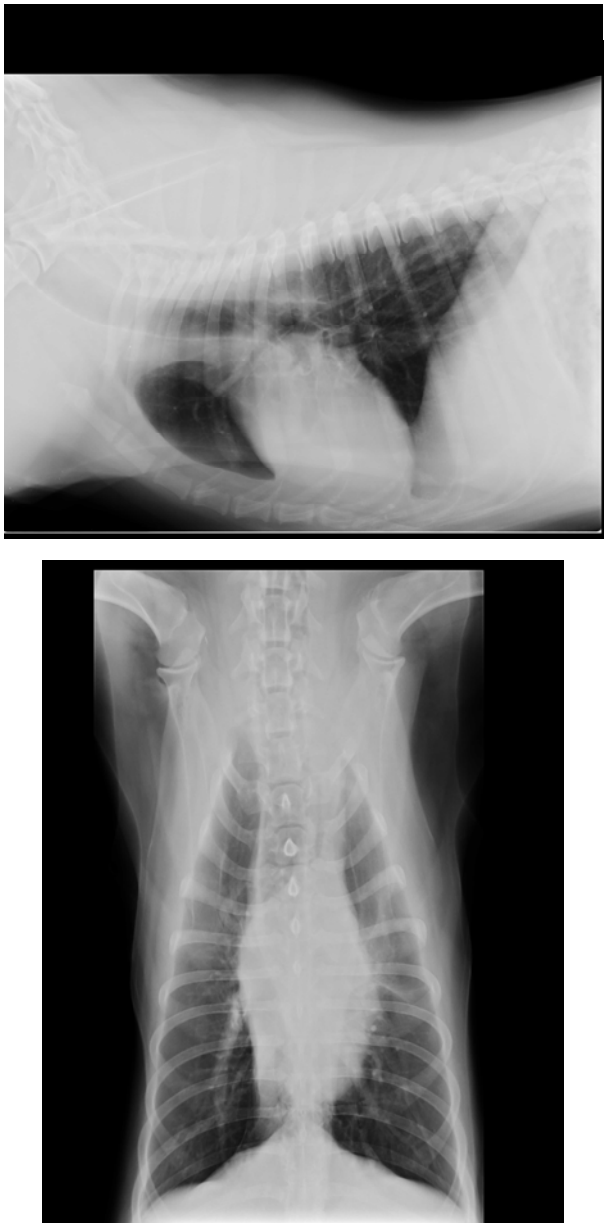


Figure 7. X-ray follow up performed 12 months after surgery confirmed the normal clinical course of the patient

suppose that anatomical abnormalities of the lung tissue (position or structure) could be responsible for the increased mobility of the cranial lobe.

In humans, computed tomography is an indispensable diagnostic tool, and its use is increasing in the field of veterinary medicine (D'anjou et al., 2005; Seiler et al., 2008). The most common CT findings are a broncus with an abrupt ending, as well as with enlargement, consolidation, emphysema, and in an abnormal position (Seiler et al., 2008; Schultz et al., 2009).

In our case, similar to what has been already documented, radiological radiographic and ultrasonographic findings were not specific and broncoscopy was preferred in order to evaluate the hemoptysis which could be associated with other bronchial pathologies (Siems et al., 1998).

The broncoscopic evaluation permitted the visualization of a medio-lateral compression of the left cranial lobe. In addition, the mucosal appearance was clearly altered with the presence of haemorrhage. This diagnostic imaging technique constitutes a specific procedure for visualizing bronchial disorders even if CT examination is probably the more sensitive diagnostic tool.

Exploratory thoracoscopy confirmed the torsion of the lobe and permitted us to exclude other intrathoracic lesions.

The transition of a thoracoscopy into a thoracotomy was carried out immediately and a cranial lobe lobectomy was performed (Fossum et al., 1986; Rooney et al., 2001). Possible complications reported after surgical resections include subacute pleuritis, acute toxaemia and recurring LLT; however, in our case, no complications from surgery were observed.

The lobectomy was performed without detorsion of the affected lobe. In fact, detorsion can result in reperfusion injury to the lungs and the systemic vasculature (such as DIC), which is reflected by a D-dimer increase and an ATIII decrease (Rooney et al., 2001; Lora-Michiels et al., 2003).

Thoracic lavage is important in order to ensure that there is no dispersion or loss of air at the resection site or in other sections of lung tissue (Rooney et al., 2001; Lora-Michiels et al., 2003). In human medicine, fixation of the right middle lobe is suggested after thoracic surgery because LLT may occur in the postoperative period (Rooney et al., 2001).

In conclusion, left cranial lung torsion is a rare pathology in large breed dogs. The clinical signs are not specific; diagnostic aids include X-ray examination, broncoscopy, thoracoscopy and, if necessary, computed tomography. Surgical resection without detorsion of the lobe should be the treatment of choice.

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