

# Balloon-Expandable Aortic Stent-Graft for Treatment of Descending Aortic Rupture Presumably Secondary to *Spiroserca lupi* Larvae Migration

Avner, A.,<sup>1\*</sup> Willenz, U.,<sup>2</sup> Sabbag, I.,<sup>2</sup> Moreshet, A.<sup>3</sup> and Bruckheimer, E.<sup>3</sup>

<sup>1</sup> Department of Veterinary Diagnostic Imaging, Knowledge Farm, Beit Berl, Israel.

<sup>2</sup> Lahav Research Institute, Kibutz Lahav, Israel.

<sup>3</sup> The Rehovot Veterinary Hospital, 4 Bilu Street, Rehovot 7661525, Israel.

<sup>4</sup> Schneider children's Medical Center, Israel.

\*Corresponding author: Dr. Avi Avner, B.Sc., B.V.Sc., CVR, DVDI, MRCVS. Knowledge Farm Veterinary Specialist Referral Centre, Beit Berl Campus, P.O. Box 136 Zichron Yaakov 30900, Israel. Tel: +972-9-7431117. Email: aviavner7@gmail.com

## ABSTRACT

A 4 four-year-old, male, German shepherd dog was presented for acute weakness and respiratory difficulty. Radiographically, there was pleural effusion and subsequent pulmonary atelectasis. Hemothorax was diagnosed based on pleural fluid analysis. Using CT-angiography, a caudal thoracic focal aortic wall defect with surrounding mediastinal hematoma was detected. To prevent further aortic wall exsanguination a percutaneous covered stent was successfully deployed at the level of the aortic lesion under fluoroscopic guidance. The dog recovered uneventfully. This report illustrates the feasibility and therapeutic option of aortic rupture with percutaneous delivery of a covered stent in a dog.

**Keywords:** *Spiroserca lupi*; Aortic Stent; Treatment; Canine.

## INTRODUCTION

*Spiroserca lupi* is a nematode found mainly in tropical and subtropical areas (1). Dogs are the definitive hosts and become infected by ingesting the coprophagous beetle intermediate host (2). After ingestion in the gastric lumen the *S. lupi* larvae migrate through the gastric wall, to the gastric arteries, the celiac artery and then cranially through the abdominal aorta wall to the thoracic aorta wall. From here, they migrate to the esophagus (2).

Parasitic migration in the wall of the aorta can cause extensive intimal damage and aortitis leading to acute rupture or dissection of the aortic wall and subsequent potentially fatal exsanguinating hemorrhage leading to hemomediastinum and hemothorax (3).

In this case report we describe the successful management of aortic rupture with percutaneous delivery of a covered stent

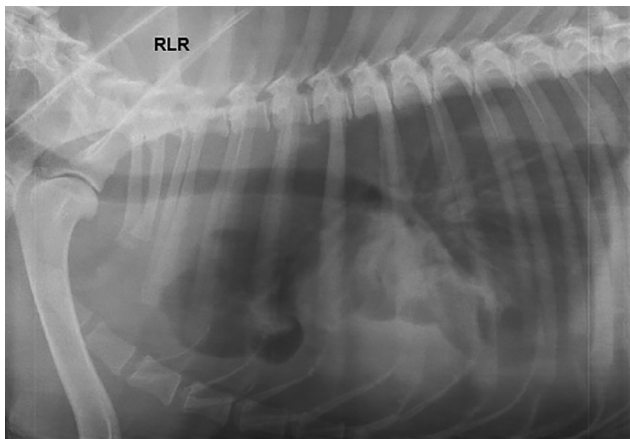
## CASE HISTORY

A four-year-old, male, German Shepherd dog weighing 35 kg, was presented with history of acute onset of weakness, exercise intolerance and respiratory distress. There was no history of trauma or exposure to anticoagulants. There were no records of prophylactic treatment for spirocercosis.

The initial clinical examination revealed normal body temperature, weak rapid femoral pulse (140 bpm) and tachypnea (46 bpm). The mucous membranes were pale-pink and capillary refill time mildly increased (2-3 sec).

Hematology showed moderate normocytic anemia (RBC:  $4 \times 10^6/\mu\text{l}$ ) [normal  $6-9 \times 10^6/\mu\text{l}$ ] mild leukocytosis and mild thrombocytopenia ( $170 \times 10^9/\mu\text{l}$ ) [normal  $200-500 \times 10^9/\mu\text{l}$ ]. Results from routine serum chemistry testing, urinalysis and coagulation profile evaluation were normal.

Thoracic radiographs (Fig. 1) taken at the referring veteri-

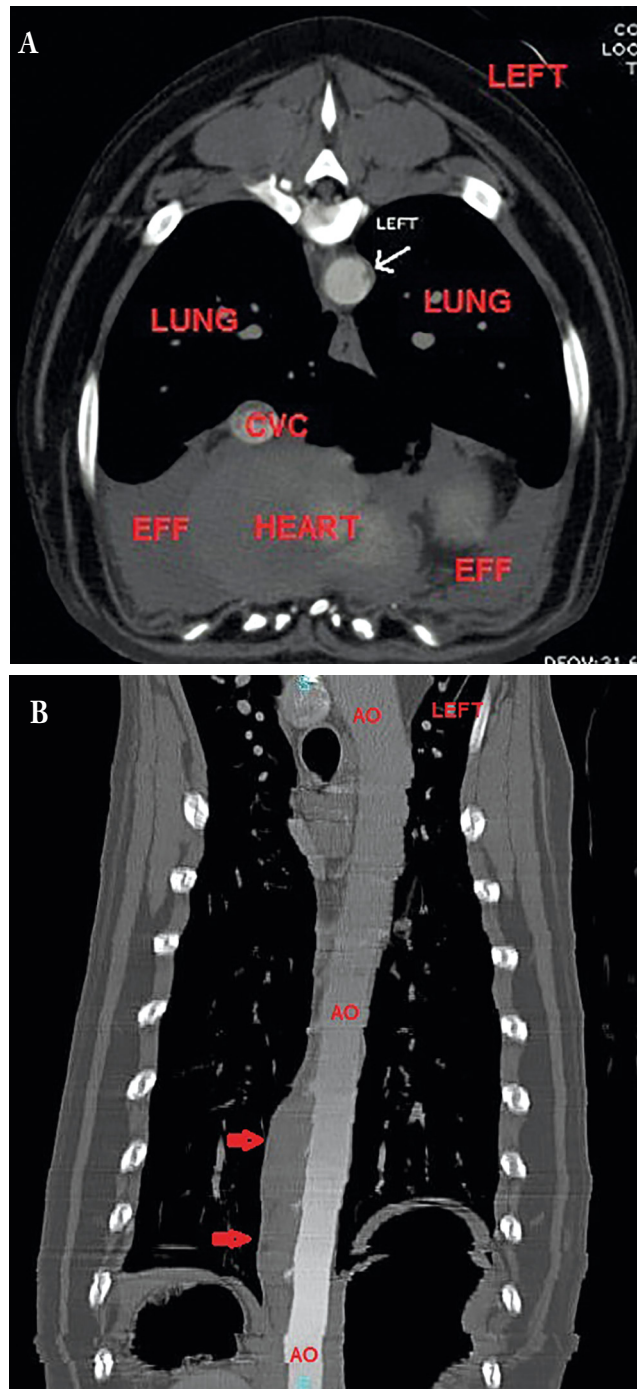


**Figure 1:** Right lateral projection (RLR) of the thorax showing moderate amount of pleural and mediastinal effusion leading to effacement of the cardiac silhouette and mediastinal structures. There is partial atelectasis of the lungs and widening of the pleural fissures by the pleural fluid. There is increased radio opacity of the caudodorsal aspect of the thorax.

narian showed moderate amount of pleural effusion and subsequent pulmonary atelectasis leading to border effacement of the cardiac silhouette. The caudal thoracic mediastinum was increased in width and opacity. Reverse fissure lines extending from the lateral border of the mediastinum were suggestive of mediastinal fluid. Thoracocentesis yield blood (PCV 25%)

CT examination (MxTwin Dual (2 slice) helical CT, Manufacturer Picker, Israel) of the thorax was performed with the patient in sternal recumbence and under general anesthesia. Pre- and post-contrast images were reviewed in soft tissue (WL 32, WW 349) and lung (WL -400, WW 1600) windows, sagittal and dorsal 2-mm thick reconstructions were made as required.

There was evidence of pleural effusion, settling at the dependent part of the thorax leading to partial atelectasis of the lung lobes. The mediastinum was distended with iso-attenuating unstructured density particularly adjacent to the descending aorta. Periosteal reaction on the ventral aspect of the caudal thoracic vertebral bodies (L7, L8) were characterized by mid-body, ventral thin brush-like periosteal reaction, consistent with early stage spondylitis. Post IV contrast administration the aortic intima was delineated by the hyper-attenuating blood revealing slightly irregular intima associated with mild thickening of the wall and focal slit like discontinuity of the wall (Fig. 2) associated with peri-wall mediastinal iso-attenuating plaque like structure (hematoma). There was no evidence of active extravasation



**Figure 2:** Post contrast transverse CT image at the level of T9-10 (A) showing mild aortic wall thickening with a hypoattenuating contrast filling defect [arrow] suggestive of aortic wall defect. There is evidence of pleural effusion (EFF) settling at the dependent part of the thorax leading to partial atelectasis of the lung lobes. There is no evidence of aneurysmal dilation or mineralization of the aortic wall. Post contrast dorsal reconstruction at the level of the thoracic AO (B). Note the widening of the caudal thoracic mediastinum (red arrows) due to peri-aorta mediastinal hematoma. Abbreviations: Caudal vena cava (CVC), Aorta (AO)

of the contrasted blood nor aneurysmal dilation or mineralization of the aortic wall. The esophagus was not distended and there was no evidence of intramural parasitic nodules. A diagnosis of hemothorax/ mediastinum with peri-aorta blood clot associated with aortitis and focally contained aortic rupture was made.

Due to the visible silt like defect in the aortic wall, it was suspected that the likelihood of recurring exsanguination into the mediastinal space was high. Therefore it was decided to attempt treatment with percutaneous delivery of a covered stent. An intravenous catheter was placed and fluids (Ringer's lactate, Teva Medical, Ashdod, Israel) were administered at twice maintenance rate. Antibiotic cover was started with amoxicillin and clavulanic acid (Synulox, Pfizer, Sandwich, Kent, UK) (10mg/kg) administered intramuscularly.

The patient was premedicated with 0.2 mg/kg of morphine (Morphine sulphate; Antigen Pharmaceuticals) administered intramuscularly. Intravenous propofol (Rapinivet; Schering Plough Animal Health), at 4 mg/kg, enabled tracheal intubation and general anaesthesia was maintained using isoflurane (Forane, Abbott Laboratories, Queenborough, Kent, UK) in 100 percent oxygen administered through a circle breathing system.

Following general anesthesia a 6Fr sheath was inserted percutaneously to the right femoral artery and a 5Fr Marker pigtail catheter was advanced to the thoracic aorta and an aortogram was performed (Fig. 3). There was unevenness at one site of the aortic wall at the level of vertebrae T9-10. There was no extravasation of contrast media but since this finding was similar to the findings on the pre-procedure CT scan, this was presumed to be the site of aortic wall disruption, and therefore it was decided to place a covered stent at that site. The aortic diameter was ~16mm and therefore we a 45mm ePTFE c was hand crimped over the Cheatham-Platinum stent (NuMed, The Netherlands) on to a 16mm Maxi LD balloon (Cordis, Johnson and Johnson, New Brunswick, NJ, USA). A long Mullins 12Fr sheath was advanced on a stiff guide wire to a point above the site of implantation and the stent was advanced on the balloon. Hand injection of contrast media through the sheath confirmed the site of stent position. Once advanced fully out of the sheath, the stent was gradually inflated until it was fully opposed to the aortic wall above and below the lesion. The balloon was deflated and withdrawn in to the sheath. The stent appeared to be well positioned and stable and a follow up aortogram

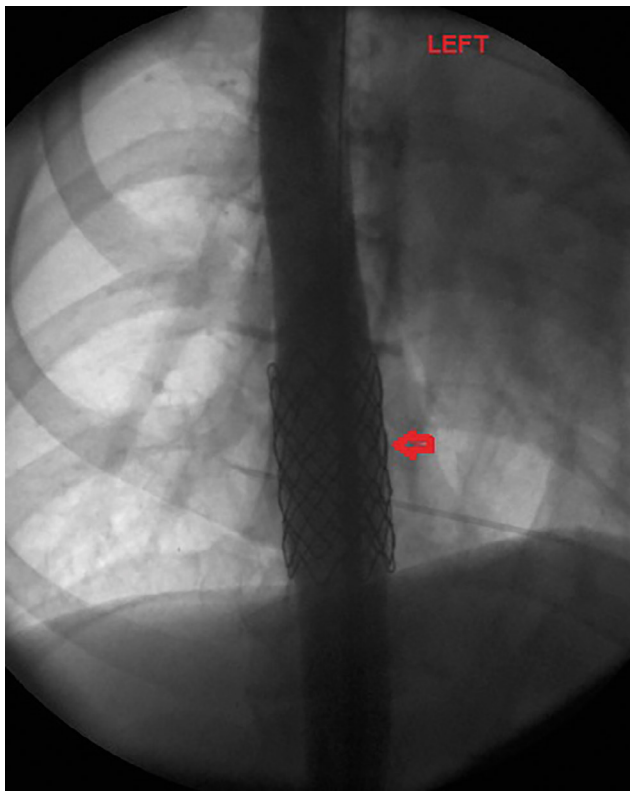


**Figure 3:** A caudal thoracic aortogram (level of L9-10). There is an unevenness of the aortic wall seen as contrast filling defect at one site (red arrow). There was no extravasation of contrast media.

confirmed attachment and position of the stent (Fig 4). The stent expanded completely and there was no apparent damage to the aortic wall. The sheath was removed and the bleeding was stopped by manual compression.

No anticoagulants were administered throughout the procedure. Post-operative analgesia was achieved with Buprenex (Buprenorphine hydrochloride; Norwich Eaton Pharmaceuticals, Norwich, NY) at 20 µg/kg administered intravenously every six hours. Following the procedure the dog was closely monitored over a period of 24 hours and recovered uneventfully. The following day the dog was discharged with a ten day course of antibiotics (Augmentin, Amoxicillin/ Clavulanic acid, GSK, Petach Tikva, Israel;





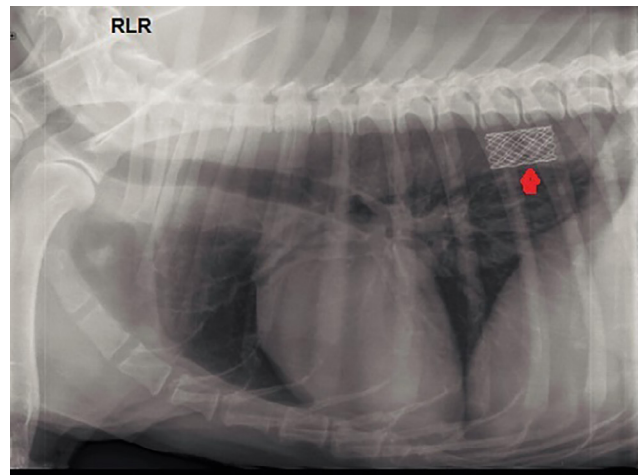
**Figure 4:** A caudal thoracic aortogram obtained after deployment of the stent-graft. The stent (red arrow) appear to be well positioned and stable. The stent expanded completely and there is no damage to the aortic wall.

PO 20/mg/kg BID) and NSAIDS (Carprofen; Rimadyl Zoetis LLC, Lincoln, NE, USA) PO 4/mg/kg SID). The referring veterinarian was instructed to continue with the anthelmintic treatment as advocated by Lavy *et al.* 2002 (4).

Thoracic radiographs were repeated by the referring veterinarian two weeks post procedure (Fig 5). At that time there was no evidence of pleural or mediastinal effusion. The stent was clearly visible lodged within the lumen against the AO wall without evidence of migration or distortion. Follow-up clinical examination six-month post procedure was unremarkable.

## DISCUSSION

The clinical presentation of *spirocercosis* varies; with symptoms being mostly associated with esophageal lesions include vomiting, regurgitation, pyrexia, weakness, anorexia, weight loss, salivation and melena. The migration of the larvae initially creates extensive intimal damage and aortitis, which may lead to aortic wall rupture or dissection and exsanguination. Furthermore,



**Figure 5:** Follow up right lateral projection (RLR) of the thorax (taken by the referring veterinarian one month after the stenting procedure). The lungs are fully aerated and adequately radiolucent. There is no evidence of residual free fluid or mediastinal widening. The stent (red arrow) is in the correct position with normal size and shape. There is no evidence of migration, distortion or aortic wall dissection.

in many of these cases the aortitis progress to wall scarring, dystrophic mineralization and possibly aneurysm formation which may then lead to rupture and acute death (5).

The diagnosis of early *S. lupi* infection when esophageal nodules are absent is challenging. Computed tomography may detect early spondylitis and aortic lesions as well as aberrant migration. It was established that in endemic areas spondylitis of the thoracic vertebrae is an almost consistent finding in spirocercosis (6). Other diagnostic modalities such as endoscopy and fecal examination depend on the presence of esophageal nodules or eggs in the feces, respectively, therefore are not helpful in the very early stages of infection. Serology might aid in detecting early infection (7).

The CT findings of spondylitis and aortic lesions and the fact the dog lives in an endemic area for *S. lupi* were highly suggestive of early larval migration leading to the aortic lesion and subsequent exsanguination. Therefore was decided to commence with conventional doramectin treatment (4).

It was previously assumed that aortic rupture with consequent hemothorax is almost always fatal (3), however at our facility we have observed such cases with hemothorax or hemoabdomen in which the initial aortic exsanguination has been contained by a large blood clot within the dorsal mediastinum (8). Obviously, these cases are still at risk of recurring exsanguination and death (9). In one experimental study 12% of dogs died 12-102 days post-infection due to aortic rupture (2).

In humans, traumatic or spontaneous acute aortic rupture is often treated successfully with a covered stent (9). In this case report we demonstrated that deployment of covered stent within the damaged aorta could be considered as a fairly simple and viable treatment for impending recurring aortic exsanguination. However, the procedure requires basic knowledge in endovascular access and catheterization and the availability of image intensified fluoroscopy.

Complications such as paraplegia, cerebral vascular accident, onset of fever that is not related to infection were described in human cases (10), however, were not noted in this case and despite occlusion of two pairs of intercostal arteries (Fig. 4), the dog did not show any clinical abnormality to suggest spinal cord injury.

In clinical patients where the aortic wall is thickened, irregular and scarred, one should be careful not to further rupture the wall while deploying the covered stent. Therefore the site of rupture was allocated on the angio-CT exam prior to the procedure and the caliber of the AO was measured on each side of the lesion. Subsequently we have chosen to inflate the stent with a balloon with the same size as the aorta in order to prevent over distension and possible rupture.

To the best of our knowledge, this is the first report of successful treatment of pathologic aortic rupture with the percutaneous delivery of a covered stent in a dog.

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