Acute aortic rupture in a dog with spirocercosis following the administration of medetomidine

K E Joubert[a*], M J McReynolds[a] and F Strydom[a]

ABSTRACT

Spirocercosis is an emerging disease in veterinary medicine. A strong suspicion of spirocercosis is usually evident after a thorough clinical examination and radiography of the chest has been performed. Lesions seen on radiography include an oesophageal mass, spondylitis and oesophageal air. Unfortunately, radiography is not diagnostic and additional diagnostic procedures are required to confirm the diagnosis. Endoscopy is commonly performed to diagnose the condition. The dog presented in this study had radiographic and clinical signs consistent with spirocercosis and definitive diagnosis was required. Shortly after sedation with medetomidine, the dog went into cardiac arrest and failed to respond to resuscitative measures. On *post mortem,* the diagnosis of spirocercosis was confirmed and the cause of death was identified as acute aortic rupture. Aortic aneurysms are not an uncommon finding and cause of acute death in dogs with spirocercosis. The acute rupture of the aorta in this case is most probably the result of cardiovascular changes associated with the administration of medetomidine. Medetomidine causes an acute rise in systemic vascular resistance with hypertension. The increase in shear stress across the weakened aortic wall resulted in rupture. Caution with the use of medetomidine in patients with spirocercosis is advised.

Key words: aneurysm, aortic rupture, dog, medetomidine, *Spirocerca lupi,* spirocercosis.

Joubert K E, McReynolds M J, Strydom F **Acute aortic rupture in a dog with spirocercosis following the administration of medetomidine**. *Journal of the South African Veterinary Association* (2005) 76(3): 159–162 (En.). Fourways Veterinary Hospital, PO Box 68159, Bryanston, 2021 South Africa.

INTRODUCTION

Spirocerca lupi is primarily a nematode parasite of dogs but may affect other animals, particularly carnivores[10]. The adult is usually found in a nodular mass in the oesophagus[2,10]. The female lays eggs into the oesophagus that travel through the gastrointestinal tract to be passed out in the faeces[2,10]. Coprophagic beetles eat the third stage larva which then develop into infective larva[2,10]. The beetle is then consumed by the host[2,10]. The larva penetrate the gastric mucosa and migrate along the gastric arteries to the aorta[2,10]. Three months later the larva migrate through the aortic wall to the oesophagus to form a granuloma[2,10]. The most common lesions created are oesophageal masses, oesophageal granulomas, aortic scars and aortic aneurysms[10]. Spondylitis of the thoracic vertebrae are commonly seen[3,10]. Oesophageal masses can transform to neoplastic lesions (fibrosarcomas or osteo-sarcomas)[3,10]. Hypertrophic osteopathy can be seen as a result of thoracic masses[3,10]. Granulomatous masses have been reported in the heart, lungs, gastrointestinal tract, urinary system and subcutaneous tissues[10].

Clinical signs are usually seen in dogs of more than 6 months of age[3]. Clinical signs associated with oesophageal lesions include vomition, regurgitation, pyrexia, weakness, anorexia, weight loss, salivation and maleana[3,9,10,14]. In endemic areas it has been suggested that dyspnoea, chronic cough and hypertrophic osteopathy should be considered as signs of spirocercosis[3]. Sudden death has been reported following rupture of aortic aneurisms[3,10]. A definitive diagnosis can be made based on the finding of *S. lupi* eggs in a faecal float[10]. Survey radiographs will usually demonstrate the oesophageal mass which is then confirmed with endoscopy where the nodules and often the adult nematodes can be seen[3,10]. Radiographs are less sensitive than endoscopy for the diagnosis of spirocercosis[2,10,14]. Endoscopy is usually performed under general anaesthesia.

Aortic aneurysms are considered rare findings in veterinary medicine[17]. Most cases in living animals have been described as asymptomatic with the exception of spirocercosis and a case with posterior paresis[17].

Acute rupture of the aorta as a cause of sudden death has been described in dogs with spirocercosis[3,5].

Medetomidine is used for sedation or in combination with general anaesthetics for routine procedures such as radiographs, abdominal ultrasound, endoscopy and minor surgical procedures[6].

This case study describes the acute death of a dog following the administration of medetomidine in a dog suffering from spirocercosis. This case is interesting as a potential pathophysiological link exists between the acute decompensation following the administration of medetomidine and the aortitis and aneurysm from spirocercosis.

CASE HISTORY

A 10.5-year-old, 40.5 kg, male Labrador was presented with a history of listlessness and anorexia for 2 days. The dog had killed 2 snakes 4 days ago and this was considered an incidental event. The initial clinical examination revealed a fever (39.7 °C), a normal pulse (112 bpm) and tachypnoea (46 bpm). The mucous membranes were pink and moist and the capillary refill time was 1 second. Some signs of discomfort were evident when the cranial abdomen was palpated but no masses or foreign bodies were felt. Clinically the dog was depressed and reluctant to move. Rectal palpation showed an enlarged prostate. Auscultation of the chest, palpation of lymph nodes and examination of the eyes, mouth and skin revealed no abnormalities.

A peripheral blood smear showed a neutrophilia, no blood-borne parasites were detected and the remainder of the blood components appeared normal. A cystocentesis was performed and a urine analysis done. The urine SG was 1.019 and on dipstick (Combur–9, Roche Diagnostics, Isando) the pH 7, proteinuria 1+ and haemoglobin 1+. Faeces were normal in consistency and colour and faecal floatation revealed no eggs. Total protein, albumin and globulins were

[a]Fourways Veterinary Hospital, PO Box 68159, Bryanston, 2021 South Africa.

*Author for correspondence.
E-mail: kenneth@fourwaysvet.co.za

Received: August 2005. Accepted: September 2005.

Table 1: **Serum chemistry, electrolytes and haematology.**

Parameter	Units	Day 1	Day 2		Reference values
Electrolytes					
Na^+	mmol/ℓ	158	152		144–160
K^+	mmol/ℓ	5.2	4.5		3.5–5.8
Ca^+	mmol/ℓ	2.4	2.4		1.95–2.83
Cl^-	mmol/ℓ	112	120		109–122
Serum chemistry					
ALT	U/ℓ	34	75		8–80
ALP	U/ℓ	92	218		30–400
Urea	mmol/ℓ	2.7	2.2		2.5–9.6
Creatinine	umol/ℓ	45	78		44–159
Glucose	mmol/ℓ	6.4	5.2		4.2–6.9
Total protein	g/ℓ	105	75		53–78
Albumin	g/ℓ	47	26		27–38
Globulins	g/ℓ	58	49		26–46
Lipase	U/ℓ		231		100–900
Haematology			**Absolute**	**Relative**	
White cell count	$\times10^9$/ℓ		13.98		6–17
Lymphocytes	$\times10^9$/ℓ		1.6	11.5 %	1–4.8
Monocytes	$\times10^9$/ℓ		1.54	11 %	0.15–1.35
Neutrophils	$\times10^9$/ℓ		10.32	73.8 %	3–11.4
Eosinophils	$\times10^9$/ℓ		0.44	3.2 %	0.1–0.75
Basophils	$\times10^9$/ℓ		0.08	0.5 %	<0.1
PCV	%		27.3		37–55
Red cell count	$\times10^{12}$/ℓ		4		5.5–8.5
Haemoglobin	g/dl		10.3		12–18
Mean cell volume	fl		68.7		60–77
Mean corpuscular haemoglobin concentration	g/dℓ		37.7		32–36
Mean corpuscular haemaglobin	g/dℓ		37.7		19.5–24.5
Reticulocytes	%		10.4		0–1.5
Platelets	$\times10^{12}$/ℓ		175		200–900

elevated. Serum electrolytes and serum chemistry was normal (Table 1).

Initially a diagnosis of pyrexia of unknown origin was made and the dog was admitted to the hospital for further tests the next day. An intravenous catheter was placed and fluids (Ringer's lactate, Fresenius Kabi, Midrand) were administered at twice maintenance rates. Antibiotic cover was started with amoxicillin and clavulanic acid (Synulox RTU, Pfizer, Sandton) (10 mg/kg) administered intramuscularly.

Clinical examination the next morning showed a fever (39.4 °C), normal heart rate (120 bpm) and tachypnoea (48 bpm). The mucous membranes, capillary refill time and auscultation remained unchanged. The cranial abdomen was still tender on palpation. A mild bilateral ocular discharge was evident. Otoscopic examination of the ears revealed mild inflammation with a small quantity of black wax. A complete neurological examination revealed no abnormalities. The serum electrolytes and serum chemistry were repeated and a full haematology was done. A mild normochromic normocytic appropriate regenerative anaemia, a mild thrombocytopaenia and an increase in alkaline phosphatase was present (Table 1). Urine analysis of free flow collected urine revealed an SG of 1011, pH 7+ and haemoglobin 1+. A repeat faecal float and peripheral blood smear did not give any additional information.

A lateral thoracic radiography showed marked spondylitis of several vertebrae (T6–T9). The oesophagus was dilated 3–5 mm with air from the thoracic inlet to the level of T6. A poorly marginated 20 × 15 mm soft tissue opacity was evident caudal to the heart. On the dorsoventral radiograph deviation of the mediastinum to the right was present. The lesions seen were strongly suspicious for spirocercosis. There was no radiographic evidence of any pulmonary pattern.

Based on the clinical findings, an ultrasound was required to evaluate the painful cranial abdomen and the prostate and endoscopy was required to confirm spirocercosis. The dog was sedated with medetomidine (Domitor, Novartis, Isando) (0.01 mg/kg) given intravenously to allow for an abdominal ultrasound. The dog went into cardiac arrest 2 minutes after the administration of medetomidine. Cardiopulmonary resuscitation was instituted and atipamezole (Antisedan, Novartis) (0.4 mℓ) was administered intravenously. The dog failed to respond to standard resuscitation measures (cardiac compressions, positive pressure ventilation, adrenalin and fluids) and the resuscitation was stopped after 10 minutes. The owner was informed of what had happened. With the owner's consent the dog was submitted for a *post mortem*.

A severe bilateral haemothorax with moderate lung atelectasis was seen. Two 20 mm exophytic granulomas were evident in the distal oesophagus. On sectioning the granulomas, *Spirocerca lupi* worms were recovered. A fibrous granuloma was present between the oesophagus and the overlying aorta. Multifocal, uneven bulging granulomas associated with haemorrhage were present in the overlying aorta. The liver revealed multiple small nodules throughout the surface which were histopathologically confirmed as micronodular cirrhosis. The severe hepatic cirrhosis would have led to hepatic failure but was unrelated to the spirocercosis. Mild protastic enlargement was also found. The spleen was normal and the stomach was empty. The *post mortem* concluded that acute rupture of the aorta led to hypovolaemia (bilateral haemothorax), shock and death.

DISCUSSION

Spirocerca lupi has been reported to occur in all provinces of South Africa with the highest incidences found in KwaZulu-Natal and Gauteng[9]. Twenty-eight per cent of veterinarians reported seeing cases in South Africa[9]. Thirteen per cent of dogs from a resource-limited area in the Free State Province, South Africa had *S. lupi* recovered from their gastrointestinal tract[11]. Spirocercosis is described as an emerging disease with a significant increase in the number of cases seen[4,9,10]. A higher incidence has been reported in large-breed dogs[9,10,14].

The most common clinical sign of spirocercosis have been reported as vomition and regurgitation[10,14]. In a smaller prospective case series, ptyalism and sialoadenopathy were reported as common clinical signs[2,14]. In a retrospective study of spirocercosis, 40 % presented with only 1 clinical sign and 4 % presented with no clinical signs but had eggs present on faecal floatation[10]. In a survey amongst South African veterinarians, 20 % of practitioners reported asymptomatic cases[9]. Thirty-four per cent of cases present with signs not associated with the gastrointestinal tract[10]. Other common clinical signs are pyrexia, weakness and anorexia[3,10]. Respiratory signs have been described in 77 % of dogs with spirocercosis[3]. This case did not present with vomition and regurgitation but did present with a tachypnoea, pyrexia, an-

orexia and listlessness. These signs are not typical of spirocercosis and further investigations are required to confirm the diagnosis[10]. The clinical signs of spirocercosis are therefore non-specific[2].

Thoracic radiographs were taken in this case to exclude pulmonary disease (tachypnoea) and as part of the work up to pyrexia of unknown origin. The abnormalities identified were an oesophageal mass, spondylitis and air in the cranial oesophagus. Thoracic radiographs of dogs with spirocercosis usually show a caudal oesophageal mass in most cases and an oesophageal mass in a non-typical location in a few dogs[2,3,10]. An incidence of oesophageal masses of between 53 % and 91 % have been reported[3,10,14]. Most studies report an incidence of spondylitis or pseudospondylosis of the vertebrae of between 33–39 %[2,3,10]. A higher incidence of spondylosis has been reported (80 %) in one study[14]. Not all spondylosis is associated with an oesophageal mass[3,10]. No oesophageal masses have been found in 15–47 % of cases[3,10]. A moderate amount of air in the oesophagus cranial to the lesion can often be seen[3,10]. Seven per cent of dogs had no radiographic evidence of spirocercosis with diagnosis being made on endoscopy, faecal flotation or necropsy[9,10]. The aorta usually shows minimal radiographic changes[3]. Computed tomography of spirocercosis has demonstrated mineralisation of the aorta that was not evident on radiographs[3]. This may make computed tomography more sensitive for aortic lesions.

Oesophageal lesions have been found in 82 % of dogs following endoscopy[10]. Five of 6 dogs in another study that had no visible radiographic mass had oesophageal masses on endocscopy[3] Radiographs are considered less sensitive then endoscopy for the diagnosis of spirocercosis[2,10,14]. The radiographic signs of spirocercosis are not conclusive and additional diagnostic test are required to confirm the diagnosis.

It has been reported that 80 % of dogs will have eggs present if two faecal floats are done with a sugar solution[10]. However, another study reported a positive faecal float in only 2.6 % of cases[3]. It is suggested that a solution with a higher specific gravity is more sensitive for spirocercosis[2,11]. Generally faecal floatation has a poor sensitivity in the diagnosis of spirocercosis in clinical cases[2,3]. No eggs were found on the two faecal floats done on this case. This could have been the result of using a salt solution for the floatation with a lower specific gravity.

The haematological abnormalities reported in dogs with spirocercosis are anaemia, leukocytosis, monocytosis, neutrophilia, eosinophilia and thrombocytopaenia[10,14]. The most common biochemical abnormality was a raised creatinine kinase (54 %) with 30 % being more than 2.5 times greater than top normal[10]. This case showed a mild normochromic normocytic appropriate regenerative anaemia and a mild thrombocytopaenia. These changes are most commonly due to haemorrhage. It is postulated that haemorrhage from the damaged aorta before death cause these changes. Not all cases of spirocercosis will have haematological abnormalities[2]. Haematological and biochemistry are not useful for the diagnosis of spirocercosis but form an important part of a work up for pyrexia of unknown origin.

Based on the clinical findings, an ultrasound was requested to evaluate the cranial abdomen (painful) and the enlarged prostate and endoscopy to confirm spirocercosis. As the ultrasound machine was immediately available this was performed first. The endoscopy was planned for the next day. The dog was sedated with medetomidine. Acute cardiac arrest followed which was not responsive to resuscitation measures. A diagnosis of acute aortic aneurysm rupture was made on *post mortem.* Based on the clinical evaluation and radiographs, an aortic aneurysm or bleeding aorta was not suspected before sedation. Acute aortic ruptured with fatal haemorrhage occurred after the administration of medetomidine.

The usual pathophysiology of an aortic aneurysm involves disruption of the aortic intima and media with the formation of a haematoma[17]. Pulsatile and turbulent blood flow then allows for dissection either cranial or caudally[13,17]. Turbulent blood flow is responsible for aortic aneurysms seen with patent ductus arteriosus (PDA)[13]. The blood velocity in the aorta is high and this promotes turbulent blood flow[13]. Turbulence increases wall shear[13]. Turbulence also increases when the fast moving blood ejected from the heart during systole meets slower moving blood in the aorta[13]. It is plausible that both these mechanisms would lead to the aortic aneurysm seen with spirocercosis following injury to the aortic wall during larval migration. Once an aneurysm has formed, LaPlace's Law (the tension in a cylindrical wall increases as the radius increases) and Bernoulli's Principle (as velocity increases pressure decreases) leads to the aneurysm enlarging[5,13].

Aortic aneurisms have been found in 6 of 14 cases[10] and 8 of 11 cases[14] submitted for necropsy. Thirty per cent of South African practitioners reported acute haemothorax as a complication of spirocercosis[9]. Fatal rupture of the aorta is not uncommon in dogs with spirocercosis[3–5].

Medetomidine binds to peripheral α_2-receptors and increases peripheral vascular resistance[8,12,16]. This results in a compensatory decrease in heart rate, mild hypotension and reduction in sympathetic tone[8,12]. Mean blood pressures as high as 175–210 mmHg have been recorded after the administration of medetomidine[1,6,15]. The increase in afterload through vasoconstriction, would lead to a significant increase in transmural force within the aorta and to turbulent blood flow. An increase in transmural force can lead to rupture of an aortic aneurysm and death. In dogs anaesthetised for endoscopy with spirocercosis, propofol and isoflurane were used without complications[2]. These agents tend to decrease afterload through vasodilatation[7]. The recommended treatment for aortic dissection is usually a reduction in blood pressure through the administration of nitropruside[17]. The administration of medetomidine is contrary to this advise. It is postulated that in this case, that the rapid rise in blood pressure due to vasoconstriction which occurs shortly after the administration of medetomidine resulted in fatal aortic rupture and haemorrhage.

Caution should be exercised when using medetomidine to sedate dogs suspected of or known to have spirocercosis and in all dogs with aortic aneurysms.

ACKNOWLEDGEMENTS

The nurses and support staff of Fourways Veterinary Hospital are gratefully thanked for their passion to care for their patients and their compassion shown to patients.

REFERENCES

1. Bergström K 1988 Cardiovascular and pulmonary effects of a new sedative/analgesic (medetomidine) as a preanaesthetic drug in the dog. *Acta Veterinaria Scandanavia* 29: 109–116
2. Berry W L 2000 *Spirocerca lupi* esophageal granulomas in 7 dogs: resolution after treatment with doramectin. *Journal of Veterinary Internal Medicine* 14: 609–612
3. Dvir E, Kirberger R M, Malleczek D 2001 Radiographic and computed tomographic changes and clinical presentation of spirocercosis. *Veterinary Radiology and Ultrasound* 42(2): 119–129
4. Hamir A N 1984 Perforation of thoracic aorta in a dog associated with *Spirocerca lupi* infection. *Australian Veterinary Journal* 61(2): 64
5. Ivoghli B 1977 Fatal aortic aneurysm and rupture caused by *Spirocerca lupi* in a dog. *Journal of the American Veterinary Medical Association* 170(8): 834
6. Joubert K E, Lobetti R G 2002 The cardiovas-

cular and respiratory effects of medetomidine and thiopentone anaesthesia in dogs breathing spontaneously at an altitude of 1486 m. *Journal of the South African Veterinary Association* 73(3): 104–110
7. Keegan R D, Greene S A 1993 Cardiovascular effects of a continuous two-hour propofol infusion in dogs: comparison with isoflurane anesthesia. *Veterinary Surgery* 22(6): 537–543
8. Lawrence C J, Prinzen F W, de Lange S 1996 The effect of dexmedetomidine on the balance of myocardial energy requirement and oxygen supply and demand. *Anesthesia and Analgesia* 82: 544–550
9. Lobetti R G 2000 Survey of the incidence, diagnosis, clinical manifestations and treatment of *Spirocerca lupi* in South Africa. *Journal of the South African Veterinary Association* 71(1): 43–46
10. Mazaki-Tovi M, Baneth G, Aroch I, Harrus S, Kass P H, Ben-Ari T, Zur G, Aizenberg I, Bark H, Lavy E 2002 Canine spirocercosis: clinical, diagnostic, pathologic, and epidemiologic characteristics. *Veterinary Parasitology* 107: 235–250
11. Minnaar W N, Krecek, R C, Fourie L J 2002 Helminths in dogs from a peri-urban resource-limited community in Free State Province, South Africa. *Veterinary Parasitology* 107: 343–349
12. Murrell J C, Hellebrekers L J 2005 Medetomidine and dexmedetomidine: a review of cardiovascular effects and antinociceptive properties in the dog. *Veterinary Anaesthesia and Analgesia* 32: 117–127
13. Olsen D, Harkin K R, Banwell M N, Andrews G A 2002 Postoperative rupture of an aortic aneurysmal dilation associated with a patent ductus arteriosus in a dog. *Veterinary Surgery* 31: 259–265
14. Ranen E, Lavy E, Aizenberg I, Perl S, Harrus S 2004 Spirocercosis-associated esophageal sarcomas in dogs a retrospective study of 17 cases (1997–2003). *Veterinary Parasitology* 119: 209–221
15. Sap R, Hellebrekers L J 1993 Medetomidine/propofol anaesthesia for gastroduodenal endoscopy in dogs. *Journal of Veterinary Anaesthesiology* 20: 100–102
16. Vainio O, Palmu L 1989 Cardiovascular and respiratory effects of medetomidine in dogs and influence of anticholinergics. *Acta Veterinaria Scandanavia* 30: 401–408
17. Waldrop J E, Stoneham A E, Tidwell A S, Jakowski R M, Rozanski E A, Rush J E 2003 Aortic dissection associated with aortic aneurysms and posterior paresis in a dog. *Journal of Veterinary Internal Medicine* 17: 223–229