Surgical correction of a persistent right aortic arch in a kitten with concurrent mitral and tricuspid valve insufficiency and long-term post-operative management of residual megaoesophagus

Hermien Viljoen[1], Charles Boucher[1], Keagan Boustead[2], Johan P Schoeman[1]

[1]Companion Animal Clinical Studies, University of Pretoria, Pretoria, South Africa

[2]Companion Animal Clinical Studies, Valley Farm Animal Hospital, Anaesthesia and Critical CareService, University of Pretoria, Pretoria, South Africa

*Correspondence: Hermien Viljoen, University of Pretoria, Companion Animal Clinical Studies, Pretoria, South Africa. Email: hermien.viljoen@up.ac.za

Abstract

A Maine Coon kitten was diagnosed with persistent right aortic arch and concurrent mitral and tricuspid valve insufficiency. Diuretic and positive inotropic treatment were administered before surgical treatment was attempted. Surgical ligation of the ligamentum arteriosum was performed successfully. Regurgitation did not resolve despite efforts to slowly introduce solid food to the diet over a period of two months. The kitten suffered from several incidences of aspiration pneumonia that responded to antibiotic treatment. Follow-up computed tomography ruled-out extraluminal constriction of the oesophagus, and oesophagoscopy confirmed a residual megaoesophagus six months post-operatively. The kitten was euthanised seven months after surgery. The importance of a complete cardiac work-up and oesophagoscopy in suspected vascular ring anomaly examinations are highlighted in this case study.

BACKGROUND

Vascular ring anomaly (VRA) refers to the abnormal development of the embryonic great vessels and their associated branches.[1-4] Several different types of VRAs have been described in humans and various animals, including cats and dogs.[1, 2, 4, 5] Some of these anomalies lead to extramural oesophageal constriction and secondary cranial oesophageal dilatation in young dogs and cats. These cases typically show clinical signs of chronic regurgitation when solid foods are ingested.[1, 4, 6]

VRAs that have been reported in cats include persistent right aortic arch (PRAA), PRAA with an aberrant right or left subclavian artery, PRAA with a left brachiocephalic vein, double aortic arch, left aortic arch with a right ligamentum arteriosum and aberrant right or left subclavian artery as sole VRAs.[7-13] PRAA is one the most frequently described and most commonly diagnosed congenital heart abnormalities in the cat.[9] In a recent case series of 20 cats, PRAA was also found to be the most commonly diagnosed VRA in cats.[7] A PRAA develops when the right fourth arch, instead of the left fourth arch, becomes the functional adult aorta and the left ductus arteriosus forms a band between the left pulmonary artery and the anomalous right aorta causing an oesophageal constriction.[2]

The most common clinical sign seen in cats affected by constricting VRA is regurgitation that appears soon after weaning when the animals starts consuming solid food.[2, 6, 7, 14, 15] Owners may report swelling of the cervical region while eating followed by regurgitation.[11, 16-18] Kittens generally present under 6 months of age, underweight for their age and reportedly smaller than the rest of the littermates.[5, 7, 8, 11, 12, 15, 16, 19-28]

Contrast radiography by means of a barium oesophagram is useful to identify VRA resulting in megaoesophagus.[16, 19, 20, 25, 28, 29] However, survey thoracic radiography has been found to be an acceptable modality to diagnose the presence of a compressive VRA without risking aspiration of the contrast medium in a patient that is already predisposed to aspiration pneumonia.[30, 31] Oesophageal enlargement with a narrowed strictured area at the heart base along with tracheal deviation is highly suggestive of a compressive VRA.[30] While survey radiography or contrast radiography is sensitive for diagnosing a VRA, computed tomography with angiographic studies is recommended to identify the type of VRA and aberrant vessels and to assist with surgical planning.[7, 8, 32, 33] Oesophagoscopy is often not used as a diagnostic modality in suspected VRA cases. It could, however, be useful to exclude important differential diagnoses like intraluminal foreign bodies/masses and idiopathic megaoesophagus and furthermore to evaluate oesophageal health prior to performing corrective surgery.[34]

VRAs such a PRAA was previously considered a rare condition in cats.[6, 19, 20] It is however, becoming a more well-recognised condition as reports on cases are increasing.[1, 6, 8, 15] The culmination of case studies provides much needed information regarding the successful treatment, prognosis and long-term prospects of surgically treated patients. This aim of this case study is to report on the long-term management of PRAA in a kitten with a concurrent heart condition, and to provide a brief literature review on VRA cases reported to date.

CASE PRESENTATION

A 13-week-old, intact, male Maine Coon kitten weighing 1.2 kg was referred for evaluation of a suspected VRA and concurrent mega-oesophagus. The kitten was presented to the referring veterinarian with a history of regurgitation following solid food meals, since weaning. A barium swallow study was performed at the referring veterinarian and revealed an oesophageal constriction at the heart base and severe dilation of the oesophagus cranial to the constriction. The referring veterinarian recommended elevated feedings with home-made gruel until such time of a definite diagnosis had been obtained.

On presentation the kitten was alert, responsive and had a ravenous appetite. The kitten was moderately underweight (BCS 2/9)[35, 41], and the owner reported that it was smaller compared to litter mates. A bulging oesophagus could be palpated just cranial to the thoracic inlet. Particular attention was paid to auscultation of heart and lung sounds but no abnormalities could be detected. Cryptorchidism was also diagnosed in the cat. The remainder of the physical examination was unremarkable. Vaccination and deworming status were up to date at the time of presentation.

INVESTIGATIONS

Diagnostic testing performed included a blood smear, a free catch urinalysis, complete blood count and serum biochemistry. Blood smear and urinalysis were unremarkable. Haematology showed a mild segmented neutropenia (0.73×10^9/l, reference range 2.5–15.37×10^9/l), band

neutrophilia (0.38×10^9/l, reference range $0.0-0.3 \times 10^9$/l) and moderate neutrophil toxicity that was suggestive of an inflammatory leukogram. Mild hypoalbuminemia (24.1 g/l, reference range 27.0−38.0 g/l) was found, and mildly elevated levels of alkaline phosphatase (84 U/l, reference range 3–50 U/l). During hospitalisation, blood glucose levels were measured intermittently, and no abnormal levels were noted.

Diagnostic imaging performed included survey radiographs and computed tomography angiography. Survey radiographs of the thorax showed the oesophagus cranial to the heart dilated with air, food and ingesta. No signs of aspiration pneumonia could be detected clinically or radiographically. On the ventro-dorsal view, the descending aorta on the right side of the oesophagus could be visualised. Tracheal deviation with a focal leftward curvature of the trachea near the cranial border of the heart was also noted.

LEARNING POINTS/TAKE-HOME MESSAGES

- Although PRAA is the most commonly diagnosed VRA, other variations are sometimes diagnosed in cats, and specialised imaging techniques should be performed to define abnormalities is advised to assist in surgical planning
- Concurrent congenital heart disease occurs often, and echocardiography should be considered as part of the pre-operative work-up even when no murmur is auscultated.
- Prognosis for improvement after surgical correction is good, but persistence of clinical signs is likely and long-term management of such cases should be discussed with the owner.
- Oesophagoscopy at a follow-up visit will allow for evaluation of oesophageal motility, identify residual dilatation of the oesophagus and rule-out foreign bodies that may impede recovery.

Computed tomography was performed with an angiogram that revealed a PRAA without any additional variants of usual aortic branches. The location of the oesophageal constriction was determined from both the thoracic radiographs to be at the level of the 5th intercostal space. An echocardiogram was done to identify possible concurrent congenital anomalies of the heart. Tricuspid and mitral valve regurgitation was diagnosed due to tricuspid and mitral valve dysplasia. Mild right atrial enlargement was found but no myocardial changes or systemic hypertension was noted at the time of examination.

DIFFERENTIAL DIAGNOSIS

Congenital megaoesophagus

Oesophageal stricture

Oesophageal foreign body

Gastro-oesophageal intussusception

Oesophageal soft tissue mass (neoplasia, granuloma)

TREATMENT

Due to the cardiovascular abnormalities found on echocardiography, treatment with furosemide (2 mg/kg, OID, PO, Mylan, South Africa) and pimobendane (0.25 mg/kg, OID, PO, Ingelhein Pharmaceuticals, South Africa) was started five days prior to surgery. The patient was starved for 12 hours prior to surgery, and in-house blood glucose levels were measured every 6 hours (normal 3.3–5.5 mmol/l). On the morning of surgery, an over-the-needle catheter was placed in the left cephalic vein (22G Jelco, Smith Medical, UK) and general anaesthesia induced with intravenous administration of diazepam (0.2 mg/kg, Valium, Roche, South Africa) and alfaxalone (2 mg/kg, Alfaxan, AfriVet, South Africa). Following induction of general anaesthesia, the trachea was intubated using a cuffed 3.0 mm internal diameter polyvinyl chloride endotracheal tube (ETT) (Surgivet, Smith Medical, Pm Inc., USA). Throughout the induction process, the cat's head was kept elevated to avoid passive regurgitation. The cuff of the ETT was subsequently inflated using the minimal occlusive volume technique while the airway pressure was raised to 20cmH_2O. A second over-the-needle catheter (22G Jelco, Smith Medical, UK) was inserted into the left medial saphenous vein to allow for additional venous access if required. At this point the following medication was given: intravenous morphine sulphate (0.2 mg/kg, Fresenius Kabi, South Africa), subcutaneous meloxicam (0.1 mg/kg, Metacam, Boehringham Ingelheim, South Africa) and intravenous cefazolin (20 mg/kg, Zefkol, Litha Pharma, South Africa). Intravenous fluid administration with a lactated Ringers' solution (Fresenius Kabi, South Africa) at 3 ml/kg/hour was also initiated and maintained throughout the duration of anaesthesia.

Anaesthesia was maintained for the duration of the anaesthetic period with isoflurane (Isofor, Safeline Pharmaceuticals, South Africa) in 100% oxygen at a flow rate of 2 L/min, through a circle breathing system. The patient was allowed to breath spontaneously until the start of surgery. Once the left hemithorax had been clipped and the skin prepared aseptically, the patient was moved to a surgical theatre, placed in right lateral recumbency, instrumented with anaesthetic monitoring equipment, a final skin preparation performed and aseptically draped prior to initiation of surgery. The following parameters were monitored: non-invasive doppler blood pressure (Ultrasound Blood Flow Detector MD4, Mano Medical, Poland), pulse oximetry, three lead electrocardiogram (RA, LA, RH; Lead II), oesophageal temperature, end-tidal carbon dioxide, end-tidal isoflurane, tidal volume, minute volume, peak inspiratory pressure (PIP)and level of positive end expiratory pressure (PEEP) (Datex Ohmeda CardioCap 5, GE Health care, USA). Parameters were monitored continuously but recorded every 5 minutes. Blood glucose concentration was also measured every hour during the peri-anaesthetic period using a portable glucometer (AlphaTRAK2, China). All anaesthetic parameters remained within acceptable limits throughout the anaesthetic period. At one point during the surgery, the heart rate dropped below 120 beats per minute while the doppler blood pressure read 60 mm Hg. At this point glycopyrrolate (10 ug/kg, Robinul, Pharmacare Limited, South Africa) was given intravenously. Intravenous administration of morphine sulphate (0.2 mg/kg) and cefazolin(20 mg/kg) were repeated once intraoperatively. The patient was heated intraoperatively by means of forced air warming (Bair hugger warming unit – model 505, 3 M, South Africa).

Before the start of surgery, the ventilation strategy was changed from spontaneous breathing to continuous mandatory ventilation by means of pressure controlled ventilation (9100c Anaesthesia workstation, GE Medical Systems, China). The following setting were used: PIP set at 10 cmH_2O, PEEP set at 4cmH_2O and inspiratory to expiratory ratio set at 1:2. The

respiratory rate was adjusted to keep the end tidal CO_2 below 50 mm Hg. Once the thorax was opened, the PIP was lowered to keep the tidal volume around 10 ml/kg.

The thoracic cavity was approached via a left fifth intercostal thoracotomy. Once the pleura was identified, it was carefully penetrated with scissors during exhalation, creating a pneumothorax. Small Finochietto retractors were positioned to gently spread the ribs, and the lungs were isolated from the surgical field using moist laparotomy sponges. The mediastinum was incised just dorsal to the vagus nerve. The vagus nerve was gently pulled ventrally with two stay sutures placed in the mediastinum. At that point, the ligamentum arteriosum could not be visualised but was identified by careful digital palpation. Stay sutures were placed in the dilated oesophagus to assist with exposure of the ligamentum arteriosum. The ligamentum arteriosum was dissected bluntly off the underlying oesophagus using curved mosquito haemostatic forceps and was double ligated with 3/0 silk before it was divided. Once the band was resected, a stomach tube was passed through the oesophagus to allow for identification of remnant circular fibrous bands that could still be constricting the oesophagus. Some circular fibrous bands were identified and subsequently resected. The mediastinal incision was closed with 4/0 PDS in a simple continuous suture pattern, and the thoracotomy was closed routinely. Negative pressure was re-established with a three-way catheter attached to a syringe until negative pressure was maintained. Once the negative pressure was re-established, the tube was removed.

The thoracotomy incision site and two intercostal sites cranial and caudal to the incision site were blocked with 1 mg/kg bupivacaine (Macaine 0.5%; Adcock Ingram Ltd., South Africa). Post-operative pain was managed with 0.02 mg/kg buprenorphine (Temgesic; Adcock Ingram Ltd., South Africa) every 8 hours for 48 hours. Post-operative feeding regimen consisted of multiple small feedings per day (every four hours) of liquified canned food (Hills Prescription Diet a/d). The kitten was discharged four days after surgery. The owner was advised to continue with the feeding regimen for three months before attempting to introduce solid food to the diet and to continue administration of furosemide and pimobendan.

OUTCOME AND FOLLOW-UP

A follow-up consultation was done two months after surgery where the cat presented for recurrence of regurgitation whenever solid food was consumed. The cat appeared depressed, was still underweight (1.5 kg, BCS 2/9) and presented with an unkept haircoat. On thoracic auscultation, crackles could be heard on inspiration, but the rest of the clinical examination was unremarkable. Thoracic radiographs were taken on admission and again two days later to survey for signs of pneumonia. No signs of pneumonia could be detected on the radiographic images. The kitten was admitted for treatment with intravenous fluid and antibiotic therapy with amoxycillin clavulanic acid (Synulox; Haupt Pharma Latina, Italy) at 20 mg/kg subcutaneously twice a day. Antibiotic therapy was stopped after five days when lung sounds were no longer auscultated, and habitus improved markedly. The kitten was placed on a strict feeding regimen of liquefied canned food every second hour from an elevated height and kept upright for five minutes after feeding. Water was offered every hour from a height. One episode of regurgitation occurred the day after admission. Habitus improved remarkably within three days, and the cat was kept in-hospital for 12 days to monitor for recurrent regurgitation. The patient was discharged with instructions to feed a liquefied diet every four hours.

Four months after surgery the patient presented again for recurrence of regurgitation. The cat was alert, and no abnormalities were found on clinical examination besides poor body condition (2.2 kg, BCS 2/9) and unkept haircoat. The cat was admitted for feeding regimen of Royal Canin Recovery Liquid diet alternated with Hill's A/D every two hours for 12 hours a day and was discharged after 5 days. During hospitalisation, no regurgitation episodes occurred. Follow-up computed tomography was performed. A diffusely dilated oesophagus with diverticulum was present, but no constrictions of oesophagus or evidence of intraluminal obstructive lesions could be observed on computed tomography images.

A follow-up examination was performed again six months after surgery when the owner reported recurrence of regurgitation. Body weight was recorded as 2.5 kg (BCS 2/9). Oesophagoscopy was performed, and a compact trichobezoar and insect parts were removed from the severely dilated oesophagus. A mucosal fold could be visualised separating the saccular component from normal oesophagus. It was possible to advance the endoscope past the mucosal fold to oesophagus that appeared normal (video in supplementary material). The supportive cardiac treatment of furosemide and pimobendan was discontinued by the owner around 4 months after surgery. The owner also declined a follow-up echocardiography examination. The kitten showed no clinical signs of cardiovascular deterioration at that point. A month after the oesophascopy was performed the patient developed severe aspiration pneumonia and was humanely euthanised on request of the owner at another veterinarian (telephonic consultation with owner).

DISCUSSION

In a recent case study of 20 cats diagnosed with VRAs, 85% (17/20) had PRAA as the primary VRA, with four of these cats presenting with concurrent aberrant left subclavian artery.[7] A total of 43 other cases (that the authors are aware of) of VRAs in cats has been described in scientific literature dating as far back as 1960.[1-3, 5, 6, 8-28, 32, 33, 36, 37] Of these cases, 60% (25/43) was diagnosed with a simple PRAA, and a further 12% (5/43) with PRAA and an aberrant left subclavian artery. Although PRAA is the most common reported VRA in cats, it appears to be slightly lower compared to dogs where PRAA is responsible for 95% of clinical VRAs.[30, 38, 39]

Due to the nature of oesophageal constricting VRAs, most reported cases are of cats under the age of 6 months.[3, 5, 8, 10-12, 15-28] However, cats as old as 5 years have been reported to been treated with surgery with acceptable outcomes.[1, 6, 8, 33] There is no apparent sex predilection found in reported case studies (28 males and 26 females). Due to limited numbers of reported cases no breed predilection has been determined so far, but Siamese cats and Persian cats have been thought to be overrepresented.[7, 15] There are seven reports of Siamese cats and three of Persian cats reported to have been affected by VRA.[1, 10-12, 20, 21, 28, 32] Domestic shorthair cats are by far the most commonly affected (34 cases) but other specific breeds that have been reported to be affected include one each of a Ragdoll, Himalyan, Sphinx and Maine Coone.[3, 7] This case report is the second reported Maine Coon cat affected by a VRA.

Chromosomal anomalies are generally accepted to result in vascular anomalies, and affected animals may show a higher frequency of other concurrent congenital conditions.[3, 11, 12] Concurrent congenital heart defects reported in cats affected with VRAs include a double chambered right ventricle, ventricular septal aneurysm, cardiomegaly, septal defect.[7, 8, 13, 23, 25, 32, 33] The high incidence of concurrent heart defects diagnosed with VRAs highlight the importance of performing echocardiography in affected animals prior to surgical intervention

to rule out concurrent conditions and plan treatment accordingly. A high incidence of axial skeletal malformations in a case series of six cats affected with PRAA but was not able to establish a genetic link.[3] The case presented here was diagnosed with concurrent congenital tricuspid and mitral valve dysplasia and cryptorchidism.

Aberrant vessels occur frequently in cats and some VRAs may require a right intercostal approach.[32] In dogs a PRAA with an aberrant artery has been reported in between 33% to 85% of cases.[7, 33, 38] Advanced diagnostic modalities, specifically computed tomography with angiography, are important to specifically identify the nature of the VRA, identify aberrant arteries and give an accurate location of the constriction and plan surgery accordingly.[7, 8, 32, 33]

Medical therapy to treat the condition leads to poor outcomes and surgical ligation and transection of the ligamentum arteriosum is currently the treatment of choice.[1, 8, 7, 33] Once the condition is diagnosed, surgical correction should be performed as early as possible to reduce the likelihood of aspiration pneumonia occurring and to prevent excessive oesophageal stretching and potential damage to the myenteric ganglion cells responsible for oesophageal motility.[7, 8, 15, 17, 19, 33]

Medical treatment alone is associated with poor long-term prognosis and typically leads to recurring aspiration pneumonia.[7, 17] Short-term medical management may be necessary to stabilize debilitated and malnourished animals and treat patients with concurrent conditions prior to surgery.[15, 19, 21] Gastrotomy tubes offer a long-term feeding solution but require the patient to undergo general anaesthesia and disadvantages include blockage and infection.[15, 16, 22] Cats with constricting VRAs are also at high risk for suffering from concurrent aspiration pneumonia, and patients should be carefully evaluated for any signs of respiratory compromise.[8-11, 13, 17, 23, 28] Arterial blood gas analysis is very sensitive in detecting pulmonary pathology and it may be wise to perform arterial blood gas on all patients prior to general anaesthesia when there are any clinical signs of respiratory compromise even in the light of normal diagnostic imaging findings.[40]

Corrective surgery is often performed on very young animals and although the procedure is not technically difficult, it remains a very invasive procedure that requires careful consideration of anaesthetic and analgesic protocols.[7] Reported intra-operative complications include hypotension, hypothermia, tachycardia and cardiac arrest.[7] The most common post-operative complications reported are aspiration pneumonia and persistence of regurgitation.[7, 15] Less common post-operative complications include unilateral Horner's syndrome, chylothorax and front limb neuropraxia.[7, 8, 10] In two reported cases of front limb neuropraxia, the exact cause was not identified, but was most likely as a result of excessive extension of the limbs during positioning, or excessive displacement of the ribs cranial to the surgical site.[8, 10] In the recent case series by Bascuñán et al post-operative survival to discharge was 90%, but more than half of the cases continued to have clinical signs after being discharged.[7] Beside the case series, there are only six case reports of cats surgically treated for VRAs (out of 21) with long-term follow-ups (>2 months) that showed complete resolution of clinical signs.[6, 8, 15, 19, 20, 24] Persistence of clinical signs post-surgery in dogs have been reported to be between 57% and 90%.[4, 37] This suggests that even if some degree of improvement can be expected after surgical correction, complete resolution of the oesophageal dilatation restoration of peristalsis is uncommon in both the dog and cat.[7, 15] It has been suggested that severe oesophageal dilation may cause irreversible nerve degeneration and oesophageal hypomotility and that oesophageal muscle is eventually replaced by scar tissue.[7, 16, 33]

Cats should be fed a diet of small, frequent slurry or liquid meals that are from an upright position.[7] There are no definite guidelines as when owners should start introducing solid food into the diet. Reports vary from introducing solid food a day after surgery, to feeding small, blended diets fed from a height for up to three months after surgery before introducing solid food.[17, 19, 21, 23, 24, 26] It has however been shown, that some cats may need to remain on a gruel diet for the remainder of their lives to manage clinical signs.[7, 10, 17, 21, 23] It is important that owners should be warned of the possibility of failure of surgical treatment and a need for lifelong management of clinical signs. In hindsight, oesophagoscopy should have been performed the first time the cat presented for regurgitation after surgery to rule out intra-luminal obstructions, evaluate motility of the oesophagus and the re-evaluate the prognosis of the patient.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest that could be perceived as prejudicing the impartiality of the research reported.

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