Persistent Right Aortic Arch and Its Surgical Correction in a Dog

Yılmaz KÖÇ
Department of Surgery, Faculty of Veterinary Medicine, Selçuk University, 42031, Campus, Konya - TURKEY

Kürşat TURGUT, İsmail ŞEN
Department of Internal Medicine, Faculty of Veterinary Medicine, Selçuk University, 42031 Campus, Konya - TURKEY

Fahrettin ALKAN
Department of Surgery, Faculty of Veterinary Medicine, Selçuk University, 42031, Campus, Konya - TURKEY

Fatih M. BÜRDANE
Department of Internal Medicine, Faculty of Veterinary Medicine, Selçuk University, 42031 Campus, Konya - TURKEY

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Abstract: A young dog was examined because of regurgitating at the time of weaning to solid food. The dog was thin, underweight and had a sparse hair coat and distended abdomen. The cervical oesophagus was palpable as a flaccid, air-filled tube in the neck. Cardiac murmurs were not detected on auscultation of the thorax. A test meal of dog food was given and the dog regurgitated masticated, nondigested food a few minutes after eating. Contrast radiography of the oesophagus revealed contrast material accumulating in the oesophagus at the thoracic inlet and cranial to the heart. During surgery, the dilated portion of the oesophagus cranial to the stricture, the right of the aortic arch and the ligamentum arteriosum, over the stricture area was apparent. After surgical correction, the long-term outcome after 9 months was excellent.

Key Words: Persistent right aortic arch, dog.

Case Report

Introduction

Persistent right aortic arch (PRAA) and retention of the left ligamentum arteriosum is the most common vascular ring anomaly in dogs (1-4). PRAA is diagnosed most frequently in young, large-breed dogs (5). Dogs with a vascular ring anomaly usually have histories of postprandial regurgitation of solid foods after weaning. They typically are stunted, thin and unthrifty (3,6-8). A tentative diagnosis is made on the basis of history, physical examination findings, and a contrast oesophagram with megaesophagus and constriction of the oesophagus at the base of the heart. Oesophagram will not distinguish PRAA from less common vascular ring anomalies. Surgical ligation and division of the ligamentum arteriosum is the recommended method of treatment (8-11). Long-term results from studies on PRAA have revealed different outcomes (3,5).

Case history

A 70-day-old female German shepherd dog weighing 4 kg was brought to the Clinic of Internal Medicine, Faculty of Veterinary Medicine, Selçuk University, for evaluation of regurgitation of 20 day duration. The dog began regurgitating 20 days before at the time of weaning to solid food. The dog had no serious medical problems and its vaccination status was current. However, over the previous 20 days, the dog had
persistent regurgitation soon after feeding and retained growth compared with its littermate. Postprandial swelling in the thoracic inlet had been palpated.

Physical examination and diagnostic approach

On admission, the dog was bright and alert. Physical examination revealed a thin dog with a sparse hair coat and distended abdomen. The dog was underweight (4.2 kg) and much smaller than a male intact littermate, which weighed 12 kg. The cervical oesophagus was palpable as a flaccid, air-filled tube in the neck. Rectal temperature was 39.3 °C, pulse rate 140 beats/min, and respiratory rate 50 breaths/min. Cardiac murmurs were not detected on auscultation of the thorax. A test meal of dog food was given and the dog regurgitated masticated, nondigested food a few minutes after eating.

The haematology, serum biochemical analysis, blood gas analysis, urinalysis (sample obtained by cytocentesis) and faecal analysis were performed. Haematologic abnormalities included slightly low PCV (PCV: 36.8%; reference range 37-55%) and low MCV values (MCV: 55.5 µm³; reference range 62-74 µm³). Abnormal serum biochemical findings included hypoproteinaemia (3.1 g/dl; reference range 3.6-5.2 g/dl) and hypoalbuminaemia (0.70 g/dl; reference range 2.3-3.8 g/dl). The blood gas analysis and urinalysis results were normal. Parasites were not detected in the faeces. Contrast radiography of the oesophagus, using a liquid suspension of barium sulphate, revealed contrast material accumulating in the oesophagus at the thoracic inlet and cranial to the heart, consistent with a vascular ring anomaly (Figure 1). Endoscopic examination of the oesophagus revealed a dilated oesophagus that terminated in a blind pouch near the heart base.

According to the age of the dog, and the findings described above a tentative diagnosis of vascular ring anomaly was made.

Surgical correction

Five days after the tentative diagnosis, the dog was anaesthetised for exploratory thoracotomy with atropine sulphate (Vetas®) (0.03 mg/kg, SC) and thiopental

![Figures 1a and b. Lateral and ventrodorsal thoracic radiographs showing contrast material accumulation in the oesophagus at the thoracic inlet and cranial to the heart in dog with PRAA.](image)
sodium (Abbott®) (15 mg/kg, IV). Anaesthesia was maintained with halothane and oxygen delivered through a cuffed endotracheal tube. A left fifth intercostal thoracotomy for the surgical approach to the thoracic cavity was performed. The cranial and caudal portions of the left cranial lung lobe and an adequate view of the base of the heart were packed off caudally using moistened surgical sponges. The dilated portion of the oesophagus cranial to the stricture and the right of the aortic arch was apparent. The aorta was seen dorsal to the oesophagus. The perioesophageal fibrous bands, which are important in the obstruction of the oesophagus by the persistent right aortic arch, and the ligamentum arteriosum over the stricture area were dissected with careful blind dissection.

Two homeostatic forceps spaced 1 cm apart were placed proximal and distal to the isolated ligamentum arteriosum (Figure 2a) and it was divided with a blade between the forceps (Figure 2b). Both ends of the divided ligamentum arteriosum were ligatured with 2.0 surgical silk (Figure 2c). After division of the ligament, releasing the oesophagus from the vascular ring, the oesophagus does not immediately re-expand at the stenosis. There was no haemorrhage when the homeostatic forceps were subsequently removed. The ligature ends were cut short.

Closure was accomplished by placing interrupted circumcostal sutures of 1- polyglycolic acid (Ethicon®, Dexon) around the ribs immediately cranial and caudal to the incision. Before the final 1 or 2 interrupted circumcostal sutures were tightened and tied, pneumothorax was eliminated by inflation and expansion of the lung to re-establish negative intrathoracic pressure. The ventral serratus, latissimus dorsi, scalene, external abdominal oblique, and cutaneous transversus muscles and skin were closed using standard techniques. A chest tube was not placed.
Results

Postoperatively, the dog was maintained on a balanced electrolyte solution with 5% dextrose (Eczacıbaşı®) 2 ml/kg/h, IV for 6 hours. Penicillin G procaine (Pfizer®) (20,000 U/kg, IM, daily for 5 days) was administered. The dog was fed a commercial dry food (Hills p/d) pre-soaked in milk while in a vertical position (Figure 3) and there were no episodes of regurgitation. Twelve days after surgical correction, contrast radiography revealed that the constriction around the oesophagus was almost three times as big as its presurgical size. There was an easy passage of material into the stomach and a decrease in the size of the dilated cranial oesophagus was seen. At the same time, a dilation of the oesophagus caudal to the stenosis was present (Figure 4). The dog was discharged the following day and the owner was instructed to feed the dog frequent, small meals in a vertical position for 2 weeks. Follow-up information was obtained by telephoning the owner and we were informed that the dog was eating well in a normal position without any subsequent regurgitation.

Two months after discharge, the dog was re-examined. It had gained 2.3 kg in this time. Radiography revealed a more extended constriction around the oesophagus and a large decrement in the size of the dilated cranial oesophagus. Dilation of the oesophagus caudal to the stenosis was still present (Figure 5). The dog is currently eating in a normal position and has gained weight gradually. Nine months after surgery, the dog weighed 24 kg.

Discussion

PRAA and retention of the left ligamentum arteriosum is the most common vascular ring anomaly in both dogs and cats (1-4,12). Other vascular ring anomalies, including double aortic arch, left aortic arch and right ligamentum arteriosum, persistent left or right subclavian arteries, ductus arteriosus with normal aortic arch, persistent right dorsal aorta, and aberrant intercostal arteries, have been reported rarely (1,4,12-16). PRAA is diagnosed most frequently in young, large-breed dogs (5). Dogs with a vascular ring anomaly usually have histories of postprandial regurgitation of solid foods after weaning. They typically are stunted, thin and unthrifty. The breed, age, signalment, history and clinical signs of the dog in this case were consistent with reports in the literature (1,3,8,9).

Microcytosis usually indicates the presence of iron deficiency. Chronic haemorrhage, prolonged copper
deficiency, chronic diseases in adult animals, and portosystemic shunts are the causes of microcytosis. Iron deficiency without blood loss is common in nursing animals because milk is low in iron and there is increased demand for iron in these rapidly growing animals (17). Hypoproteinaemia and hypoalbuminaemia in vascular ring anomalies are common and as in this case, they could be associated with malnutrition (17). A tentative diagnosis is made on the basis of history, physical examination findings, and a contrast oesophagram with megaoesophagus and constriction of the oesophagus at the base of the heart. The results of oesophagraphy will not distinguish PRAA from less common vascular ring anomalies.

Endoscopic examination of the oesophagus is usually considered unnecessary when evaluating vascular ring anomalies (2). However, as reported by Hurley et al. (1), the oesophagus of dogs that have PRAA can be characteristically seen endoscopically as a terminating blind pouch near the heart base. Medical treatment of PRAA (e.g. liquid diets and supportive care) has been shown to be unrewarding (8,9). Long-term results are poor because the oesophageal constriction remains and oesophageal dilation worsens with time (9). Thus, surgical ligation and division of the ligamentum arteriosum is the recommended method of treatment. The ultimate goal of surgical treatment is alleviation of the obstruction and resultant clinical signs (8-11). In this case, the ligamentum arteriosum was identified, ligated and transacted, and the underlying oesophagus was freed of any residual extramural fibrous bands. Some authors have suggested that age at the time of surgical correction of PRAA is an important factor in long-term prognosis (3,7,9,18). Early surgical intervention has been recommended, because it was thought that oesophageal dilation and motility disorders would worsen and possibly become irreversible if surgery were delayed (3). However, Shires and Liu (5) stated that dogs <2 months old at the time of surgical correction had a lower survival rate than did older dogs. In the present case, the dog was 75 days old at the time of surgical correction and the long-term results were satisfactory. Long-term results of studies on PRAA have indicated different outcomes. Shires and Liu (5) stated that only 9% dogs did not have any long-term clinical abnormalities or complications. Muldoom et al. (3) have, however, reported that 92% dogs did not regurgitate at the time of long-term follow-up. Our dog also responded well to surgical correction, and did not regurgitate in a normal position, and gained weight gradually. This could be the result of recognising PRAA more easily than previously, treating the cases earlier before the development of preoperative complications, and giving more attention to the dissection of perioesophageal fibrous bands.

References