DIAGNOSIS AND SURGICAL CORRECTION OF PYLORIC STENOSIS IN A DOG – A CASE STUDY

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SUMMARY: Pyloric stenosis, which could occur as a congenital or acquired condition, is more common in dogs when compared with other domestic animals. This condition has not been reported in Sri Lanka probably due to unfamiliarity with it and lack of diagnostic facilities. This communication discusses the clinical signs, radiographic and ultrasonographic findings, and treatment of pyloric stenosis in a one-year old female Cocker Spaniel presented to the Veterinary Teaching Hospital (VTH), Peradeniya. The patient developed projectile vomiting and distended, gas filled abdomen after meals as clinical signs. Survey radiographic findings included the 'gravel sign' in the stomach. In ultrasonography, thickened pylorus was observed. Contrast radiographs after oral administration of barium sulphate suspension showed a delay of gastric emptying. All the above findings suggested that the patient had pyloric stenosis. Complete blood count and serum biochemistry demonstrated no abnormalities. Surgical intervention was performed as treatment, using Fredet-Ramstedt pyloromyotomy together with Heineke-Mikulicz pyloroplasty to clear the obstruction in the pyloric canal. Since there were no post-surgical complications, patient was discharged in 4 days following the surgery. The prognosis was satisfactory and the animal has been healthy after the surgery. The pyloric stenosis observed in this case could be congenital, with hypertrophy and subsequent stenosis gradually worsening over time.

INTRODUCTION

The pyloric part of the stomach comprises the pyloric antrum which begins at the angular notch, the short and narrowed pyloric canal and the pylorus which surrounds the opening into the duodenum, and the pyloric sphincter (Evans, 1993). Pyloric stenosis is one of the most significant problems in the canine pyloric region (Khan et al., 2015). In a case report of congenital outflow obstruction in cats, both the queen and two of her offspring, were diagnosed with pyloric stenosis (Twaddle, 1971). Pyloric stenosis or chronic hypertrophic gastropathy refers to the impaired gastric emptying caused by pyloric sphincter dysfunction or pyloric or duodenal stricture. Pyloric stenosis can be congenital or acquired. Due to narrowing of the pathway, the gastric emptying slows down or is blocked, resulting in gastric dilatation, chronic vomiting and regurgitation. Brachycephalic breeds are considered to be more prone to develop the congenital form (Jubb, 2007; Poncet et al., 2005; Washabau, 2003). In the congenital form, clinical signs will be apparent just after introducing solid food to pups and the acquired form is often seen in older animals (Grezeory et al., 2010). Causes of congenital pyloric outflow obstructions in dogs are limited to smooth muscle hypertrophy resulting in pyloric stenosis and gastric polyps (Happe et al., 1977; Pearson 1979; Abel et al., 2002; Diana et al., 2009; Kuan et al., 2009). Acquired pyloric stenosis is technically referred to as chronic hypertrophic gastropathy.
pyloric gastropathy (CHPG) and occurs due to muscular hypertrophy or mucosal hyperplasia (antral mucosal hypertrophy) in the pylorus (Walter et al., 1993; De Novo, 1989). Based on the involvement of the muscles and mucosae, the CHPG is categorized under three types: type 1 - prominent circular muscle hypertrophy, type 2 - muscular hypertrophy together with mucosal hyperplasia, and type 3 - primary mucosal hyperplasia (Bellenger et al., 1990). Previous studies have shown that the CHPG is common among small purebred dogs including Shih-Tzu, Maltese, Yorkshire, Lhasa Apso, Pekingese, Toy Poodle, Chihuahua and English Bulldog (De Novo, 1989; Halfacree, 2010; Walter et al., 1985).

**CASE REPORT**

A one-year-old female Cocker Spaniel weighing 8.5 kg was presented to the Veterinary Teaching Hospital (VTH), Faculty of Veterinary Medicine and Animal Science, University of Peradeniya with the complaint of abdominal distension within 2-3 hours after meals and vomiting undigested food. History revealed that the patient had abdominal distension and geophagic pica over two months and had started vomiting on the day prior to presentation to the VTH. Appetite and defaecation were reported to be normal. The patient was properly vaccinated and dewormed.

On general clinical examination Body Condition Score was 2.0 (emaciated), and there was abdominal distension, tympanic sounds on percussion, absence of pain on abdominal palpation, and dyspnoea due to abdominal distension. The patient was hospitalised and kept under observation. Intravenous fluid therapy was continued using normal saline, 25% dextrose and lactated Ringer’s solution. Percussion of the enlarged abdomen evoked a tympanic sound with simultaneous dyspnoea and projectile vomiting 2-3 hours post feeding. Feeding of solid food was replaced with blended semi-solid food and the response of the patient was observed. The projectile vomiting and abdominal distension persisted similar to when the solid food was given. The distension subsided after vomiting and no signs of pain were detected on abdominal palpation.

![Figure 1: Survey radiographs of the abdomen - A: lateral projection; B: dorso-ventral (DV) projection.](image)

Note the severely distended stomach with ingested materials visible as radio opaque foci “gravel sign” (black arrow) on a soft tissue background. The DV projection shows that the stomach occupies almost entire abdomen while intestines are displaced towards the pelvis (white arrow).
Vomiting is the predominant sign associated with delayed gastric emptying which is a prominent feature in pyloric stenosis (Pazzi et al., 2013). The differential diagnosis (DD) included pyloric stenosis, pyloric obstruction due to foreign body, gastric neoplasia, eosinophilic granuloma, polyps and fungal infections of the gastric mucosa, uremia, hypercalcemia, hepatic insufficiency, pancreatitis, gastritis, and inflammatory bowel disease (Bellenger et al., 1990; Mott and Morrison, 2019).

In order to rule out the diseases in the DD list, survey and contrast radiography, ultrasonography of the abdomen, Full Blood Count (FBC), measurement of Blood Urea Nitrogen (BUN) and creatinine, and urine analysis were carried out. Survey abdominal radiography revealed the “gravel sign”, the presence of radiopaque materials of various sizes in the distended stomach with gas filled intestines.

Barium contrast radiographs were taken at the intervals of 0, 5, 15 and 30 minutes, and 1, 2 and 3 hours. Contrast agent had travelled to the stomach within five minutes. Only a small amount of contrast agent had travelled from pylorus to duodenum over a period of two hours.

In order to examine the gastrointestinal tract, an abdominal ultrasonography was performed and hyperechoic pyloric antrum was observed. Also single circular hypo echoic muscle layer of small intestines surrounding hyperechoic bowel content (mostly gas) were observed. Thus, hypertrophied pyloric antrum with the stenosis of pyloric canal was identified. All the values in FBC, BUN and creatinine were within the normal range. Urinalysis findings were straw coloured clear urine of pH 8.0 (alkaline), mild proteinuria, mild leucocytosis, and specific gravity 1.000. In urine sediment WBC 3/high power field and epithelial cells were present.

The definitive diagnosis was made as pyloric stenosis due to pyloric antral hypertrophy and surgical intervention was decided as the corrective measure. Intravenous fluids such as lactated Ringer’s solution, 25% dextrose and normal saline were administered to prevent metabolic imbalances and dehydration prior to the surgery. Food was withheld for 12 hours. Premedication was administered using diazepam 0.2 mg/kg IV followed by propofol for induction and maintenance of anaesthesia at 4-6 mg/kg IV. After intubation and preparing the ventral abdomen for operation aseptically, laparotomy was performed through ventral midline incision.

Figure 2: Contrast abdominal radiographs - A: immediately after giving barium meal; B: five minutes later.
Figure 3: A: Contrast radiography of the right lateral abdomen after three hours showing the stomach distended with radiopaque material mixed with ingesta. B: Abdominal ultra-sonogram with arrow showing hyperechoic (hypertrophied) pyloric antrum and narrowed pyloric canal.

from xiphoid to midway between the umbilicus and pubis. Then the distended stomach was located and partially exteriorised and isolated using sterile abdominal packs. The gastrotomy was done with a stab incision of 2 cm long in a hypo-vascular area of the ventral aspect of the stomach between the greater and lesser curvatures away from the pylorus. The stomach which was filled with a large volume of fluid (1,250 ml) and small stones and mineral particles was emptied using suction and a scoop.

The pylorus was found to be severely thickened in comparison with the consistency of a normal

Figure 4: A: Application of suction to remove fluid and particles in the stomach. B: Suturing the gastrotomy incision, which was done leaving a space to insert a pair of artery forceps.
pylorus. It was held between index finger and thumb in the non-dominant hand and a longitudinal incision was made through serosa and muscle layers while preserving the mucosa.

**Figure 5:** Making an incision excluding the mucosa of pylorus

Then a pair of artery forceps was inserted through the partially closed gastrotomy incision to make sure that there was no obstruction in the pylorus. Following few unsuccessful attempts to clear the passage of the pyloric canal by inserting the artery forceps, a full thickness incision (pyloromyotomy) was made in the pylorus. A thickened mucosa was obvious through the incision and pyloroplasty was planned. Two traction sutures were placed at the two centres of the incision, incision was oriented transversely and simple interrupted sutures were placed using 3/0 vicryl at <3 mm intervals. A complete seal was ensured when there was a leak noticed by placing additional sutures at the places of leaks. Then abdominal lavage and suction were performed, and the laparotomy incision was closed routinely.

Post-operative care was followed with analgesia (tramadol HCl 2 mg/kg IV), antibiotic (cefuroxime, 20 mg/kg IV), and antiemetic (promethazine, 0.2 mg/kg IV) 12 hourly. The patient was kept Nil Per Os (N.P.O.) for 48 hours and fluid therapy (lactated Ringer's solution, 25% dextrose, normal saline) was administered to prevent metabolic imbalances and dehydration. The patient did not show vomiting post-surgically and after 48 hours of N.P.O. Even after the gradual introduction of solid food, the vomiting was not observed. Prognosis of the patient was good after the surgery and therefore animal was discharged in four days post-surgically.
DISCUSSION

Pyloric stenosis of both congenital and acquired origins has been reported in dogs from other countries, but no such case studies have been published in Sri Lanka. Clinical signs of congenital pyloric stenosis could appear as late as one year of age (Mott and Morrison, 2019). The age of the dog which was presented in this case was 11 months. Thus, the pyloric stenosis observed in this case could be congenital because hypertrophy and subsequent stenosis gradually worsened over time (Mott and Morrison, 2019).

Depending on the degree of the pyloric narrowing, clinical signs associated with pyloric stenosis can be varied. Even though the reason for acquired pyloric hypertrophy in dogs is not known, it has been suggested that the repeated pylorospasms could lead to acquired pyloric stenosis. In addition, allergic responses, irritation of the pylorus, hyperacidity, and nervous tension may influence the development of this disease (Rhodes et al., 1965). Complete obstruction causes complete retention of ingesta in the stomach and obstruction of its passage into the duodenum, resulting in fluid-filled distended stomach (Khan et al., 2015). In this case, the patient had severely distended stomach which occupied 2/3 of the abdomen within 2-3 hours after a meal.

Survey and contrast radiographic studies can reveal delayed gastric emptying, gastric dilation or enlargement, pyloric mass or filling defect and increased motility (Lieb et al., 1993). In survey radiography, “Gravel sign” (presence of radiopaque material in various sizes in stomach) can be seen (Dennis, 2010). The contrast radiograph could be used to diagnose and determine the severity of the pyloric stenosis (Biller et al., 1994). The barium filled pyloric antrum stops abruptly giving the appearance of a “beak”, “string” or “tit” signs (Shuman et al., 1967). Beak sign is a bulge in the distal antrum with streak of barium pointing towards pyloric canal (Bilodeau, 1971), tit sign is due to the presence of peristalsis compressing the peristaltic pouch against the hypertrophied sphincter (Biller et al., 1971), and string sign is due to long pyloric canal and increased muscle thickness (Rhodes et al., 1965). Besides, double-track sign is the most reliable of all signs. It shows the interposition of folded, thickened mucosa in the pyloric canal (Shuman et al., 1967).

However, the radiograph of this patient did not show above signs, and beak sign could not be observed in contrast radiography. Nevertheless, the movement of minute amount of contrast agent from stomach into the duodenum after 2 hours of consumption of barium meal could be observed. Further, the “gravel sign” was visible in the survey abdominal radiograph.

In abdominal ultrasonography, circumferential thickening of the pylorus in the affected dog irrespective of the breed, should be equal to or more than 9 mm, with the thickness of the muscular layer similar or more than 4 mm in pyloric stenosis (Biller et al., 1994; Dennis, 2010; Penninck et al., 1989). In our patient, pyloric muscular layer thickness was more than 4 mm. In addition to the techniques that were used in this case, fluoroscopy and endoscopy could also be useful diagnostic tools to identify pyloric stenosis (Biller et al., 1994; Rhodes et al., 1965). These techniques mostly help to rule out neoplasias and polyps which can cause similar clinical signs (Leib et al., 1993). Fluoroscopic studies can reveal normal or vigorous gastric contractions with slow passage of
contrast material into the duodenum (Leib et al., 1993; Rhodes et al., 1965).

In events when the contrast radiographic and abdominal ultrasound findings were inconclusive, the definitive diagnosis can be arrived with exploratory laparotomy. During the exploratory surgery, the pyloric stenosis should be confirmed and gastric outflow should be improved after the removal of the thickened folds of mucosa by pyloroplasty or pyloric resection (Leib et al., 1993).

Surgical intervention is necessary in pyloric stenosis due to pyloric antral hypertrophy (Rhodes et al., 1965). Pyloromyotomy and pyloroplasty can increase the diameter of the pylorus and these procedures are used in correcting gastric outflow obstructions in pyloric stenosis. The commonly used pyloromyotomy is called as Fredet–Ramstedt pyloromyotomy (Khan et al., 2015). There are two types of pyloroplasty; they are Heineke–Mikulicz pyloroplasty and Y-U pyloroplasty. Y-U pyloroplasty allows resection of pyloric mucosa with muscular hypertrophy and as the outcome it increases luminal diameter of the outflow tract (Fossum et al., 2002). Fredet–Ramstedt pyloromyotomy is the simplest and easiest of the procedures (Halfacree, 2010). However, the healing process may narrow down the lumen size. Sometimes, duodenal gastric reflux may occur if pyloric function is altered in the surgery. Metoclopramide or cisapride may be beneficial in these cases (Fossum et al., 2002). However, in this patient, aforementioned drugs were not used since the patient did not develop duodenal gastric reflux.

In this patient, Fredet–Ramstedt pyloromyotomy combined with Heineke–Mikulicz pyloroplasty was performed to correct the condition. Pyloroplasty enhances the healing process by prevention of stricture formation which can be a result of pyloromyotomy. The technique pyloroplasty simply augments drainage of the gastric contents by increasing the diameter of the pyloric canal (Khan et al., 2015). The outcome of the surgery of this patient was satisfactory and so far, the patient has not developed vomiting, abdominal distension or any post-surgical complications except diarrhoea for several days within a month after the surgery. The diarrhoea had resolved with the appropriate medication.

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