Combined surgical and endoscopic approach for the reduction of a congenital hiatal hernia in a cat: a case report

L. Pisoni, S. Del Magno, F. Cinti, M. Baron Toaldo, M. Joechler, M. Pietra

School of Agriculture and Veterinary Medicine, University of Bologna, Bologna, Italy

ABSTRACT: A case of surgical resolution of type I or "sliding" hiatal hernia is reported. A seven-month-old kitten was presented because of abdominal discomfort, accelerated breathing after eating and chronic vomiting. The clinical examination was unremarkable. Thoracic radiographs and gastroscopy led to the diagnosis of type I hiatal hernia. The surgical resolution consisted of hiatal plication, oesophagopexy and left-flank incisional gastropexy. All procedures were carried out using a 6 mm videoendoscope positioned in the stomach to evaluate the right oesophago-gastric junction reduction. One week after surgery there was a recurrence of symptoms and a second laparotomy was performed. During the second surgery additional hiatal plication was necessary and an oesophagopexy was repeated after dissection of the phrenico-oesophageal ligament. Moreover, a new incisional gastropexy was carried out after resolution of the first one. The cat recovered without complications and at one-year follow-up did show no signs related to the hiatal hernia. This communication reports on possible additional surgical techniques in cases of type I hiatal hernia and contributes to an understanding of the importance of oesophagopexy in cases of hiatus malformation.

Keywords: cat; surgery; endoscopy; hiatal hernia

List of abbreviations
HH = hiatal hernia, LES = lower oesophageal sphincter, CRI = constant rate infusion

Congenital hiatal hernia (HH) is considered an unusual disease in dogs and is also rare in cats (Sivacolundhu et al. 2002; Hunt and Johnson 2003). Different types of HH have been described in veterinary medicine: type I, sliding hiatal hernia, type II or paraoesophageal hernia, type III, which is a combination of types I and II, and type IV, a mix of I and II complicated by the herniation of other abdominal organs (Miles et al. 1988; Williams 1990; Auger and Riley 1997; Rahal et al. 2003; Kirkby et al. 2005; Owen et al. 2005; Baig et al. 2006). Type I or "sliding" HH, in which the gastro-oesophageal junction moves cranially to the diaphragm, is the most common form both in humans and animals (Ellison et al. 1987; Prymak et al. 1989; White 1993). Different pathogenic mechanisms have been proposed, such as: congenital (Lorinson and Bright 1998), traumatic (Prymak et al. 1989; Brinkley 1990; Lorinson and Bright 1998), neuromuscular (Hardie et al. 1998), and metabolic (Hardie et al. 1998). Respiratory diseases, stenosis of the nasopharynx (Hardie et al. 1998; Arndt et al. 2006; DeSandre-Robinson et al. 2011) and the presence of trichobezoar (Ellison et al. 1987; Owen et al. 2005) can be associated with HH.

The main symptoms are not specific and are represented by ptyalism, anorexia, dysphagia, regurgitation, vomiting, hematemesis, coughing, dyspnoea and exercise-intolerance. These symptoms are considered to be connected with the frequent gastro-oesophageal reflux leading from the hernia (Hunt and Johnson 2003). The reflux is probably caused by the reduction in muscular tone of the lower oesophageal sphincter (LES) that moves from the abdomen to the thorax through the diaphragm (Sivacolundhu et al. 2002).

Positive-contrast oesophagography, fluoroscopy and gastroscopy are the diagnostic modalities of

choice in the diagnosis of HH. Thoracic radiographs can show soft tissue opacity dorsal to the caudal vena cava, absence of the right crus of the diaphragm border, megaoesophagus and lung lobe consolidation consequent to aspiration pneumonia. Plane radiographs can also not be diagnostic if the gastric protrusion is not permanent (Hunt and Johnson 2003).

Different surgical techniques are described to resolve HH: antireflux procedures, hiatal plication, oesophagopexy and incisional left-side or tube gastropexy (Ellison et al. 1987; Prymak et al. 1989; Bright et al. 1990; Williams 1990; Callan et al. 1993; White 1993; Lorinson and Bright; 1998). No specific guidelines about treatment protocols are present in the literature, particularly about which type of surgical procedure gives greater success in relation to the different types of HH. Clearer indications can be deduced about surgical intervention in types II, III and IV, where surgical therapy is advised even in the absence of clinical signs, but, as already reported, the incidence of these types of hernia is low (Miles et al. 1988). Usually for symptomatic type I hernia medical therapy should be attempted, even if the success rate is low (Ellison et al. 1986), and surgery is usually proposed only after failure of the medical therapy (Lorinson et al. 1998; Sivacolundhu et al. 2002).

In this case report we describe the surgical therapy of a case of congenital "sliding" HH in a cat. We wish to emphasise that hiatal plication and gastropexy may not be sufficient to prevent recurrence if the anatomy of the diaphragm is altered. In this case, phreno-oesophageal ligament dissection and an oesophagopexy were necessary.

Case description

A seven-month-old cat was referred to the Veterinary Teaching Hospital of the University of Bologna for nausea, chronic vomiting, abdominal discomfort and a transient increase in respiratory rate after eating. Physical examination revealed values within normal limits. Standard survey radiographs of the thorax were taken with right laterolateral and ventrodorsal views. An oval, poorly marginated soft-tissue structure was observed in the caudodorsal thoracic space, in the area of projection of the oesophagus (Figure 1).

An oesophagogastroscopy with a flexible endoscope 6 mm in diameter and 1050 mm in length (Pentax EG-1840; Pentax, USA) showed the presence of an area of wide dilation at the gastro-oesophageal junction, with gastric rugal folds into the caudodorsal thorax associated with superficial ulcers in the distal oesophagus.

The retroverted endoscopic view showed a widened hiatus and a loose fit around the insertion tube of the endoscope (Figure 2). Functional oesophageal distension due to nasopharyngeal stenosis was excluded and a diagnosis of type I hiatal hernia and concurrent gastro-oesophageal reflux was made.

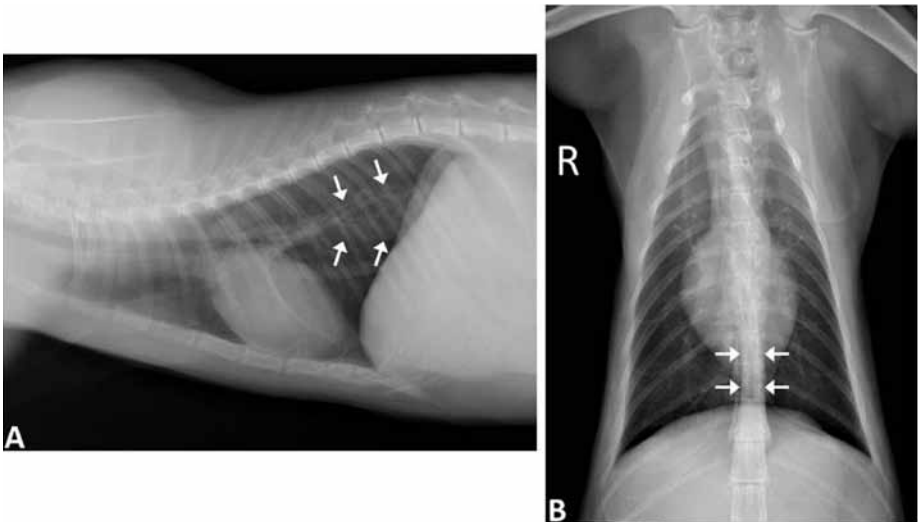

Figure 1. Survey thoracic radiograms taken in two perpendicular projections (A = left to right lateral; B = ventrodorsal; R = indicates the right side of the animal)) from a kitten with hiatal hernia (HH). A soft tissue opacity is evident in the caudal-dorsal aspect of the lung fields in the lateral view and superimposed on the thoracic vertebrae in the sagittal view (white arrows). These aspects are consistent with a fluid-filled mildly dilated caudal oesophagus and suggestive of the possible presence of HH

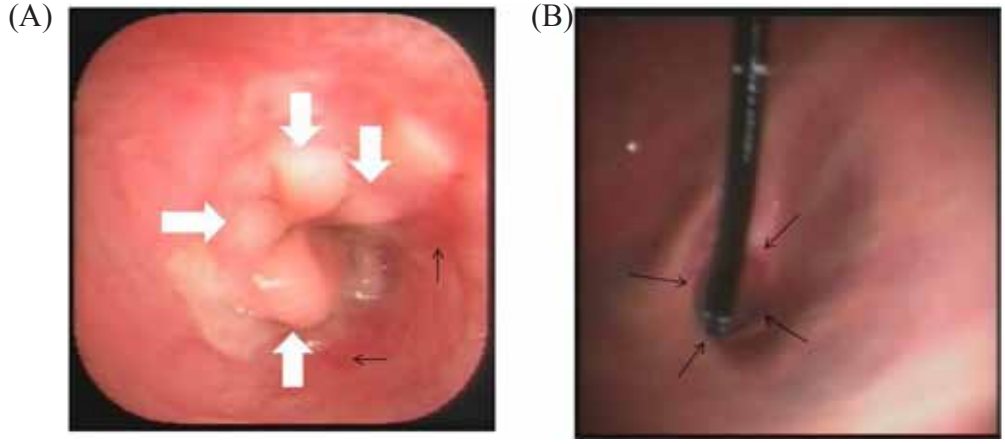

Figure 2. Endoscopic images of the oesophagogastric junction of a kitten with type I hiatal hernia. A = orthograde view: some longitudinal stomach folds are visible in the distal portion of the oesophagus (white arrows) and superficial oesophageal erosions are evident (black arrows). B = retroverse view from the stomach toward the cardia: note the abnormally enlarged distal oesophageal sphincter around the 6 mm endoscope (black arrows)

Medical therapy was instituted with an antacid syrup (Maalox plus, Sanofi-aventis SpA 2ml/kg q8) for 10 days and the owners were advised to feed the cat with a low-fat diet four/five times daily and to keep him in an elevated position during feeding and for up to five minutes after feeding. No significant amelioration of the symptoms was observed; therefore, the owner opted for surgical treatment.

The cat was sedated with ketamine (3 mg/kg *i.m.*), midazolam (0.2 mg/kg *i.m.*) and methadone (0.2 mg/kg *i.m.*); induction was obtained using propofol (4 mg/kg *i.v.*); anaesthesia was maintained using isoflurane in pure oxygen after oro-tracheal intubation; analgesia was induced during surgery and during the first 6 h post-operatively with a constant rate infusion (CRI) of fentanyl (0.002 mg/kg *i.v.* as induction bolus, then CRI 0.005 mg/kg/h *i.v.*), then by buprenorphine (0.015 mg/kg q8 *i.v.*) for two more days. Antibiotics (ampicillin and sulbactam, 20 mg/kg *i.v.*) were administered 2 h before induction of anaesthesia. Before surgery, a further oesophagoscopy confirmed the previous diagnosis of HH, and revealed severe oesophagitis and that the upper portion of the stomach was herniated into the caudal mediastinum through the oesophageal hiatus. A laparotomy was performed: after the incision of the hepato-gastric ligament, the liver was retracted on the right side of the abdomen, and the hiatus was plicated in its dorsal and left lateral part with interrupted horizontal mattress sutures to reduce the width of the hiatus. During plication the endoscope was maintained in the stomach through the gastro-oesophageal junction to avoid an excessive reduction of the hiatus and to directly confirm the resolution of the HH. Three stitches were applied from the diaphragm and the seromuscular layer of the oesophagus. Left flank fundic incisional gastropexy was performed to prevent sliding of the gastro-oesophageal junction in the thorax. Before closure of the laparotomy, endoscopic images of the hiatus were consistent with resolution of the hernia (Figure 3).

The recovery was uneventful and post-operative medications constituted antibiotics (ampicillin and sulbactam 20 mg/kg q12 *i.v.* until oral nutrition started, then *p.o.*), ranitidine (1 mg/kg q12 *i.v.*, slowly), sucralfate (0.5 g/kg q8 *p.o.*), and a low-fat soft diet in small amounts five times daily followed by maintaining the cat in a vertical position for five minutes after eating. On the second day after surgery the cat vomited after oral administration of the antibiotic drug. Assuming that the vomiting was the result of gastritis induced by the antibiotics, we decided to change from oral to subcutaneous administration, and an abdominal ultrasound was planned. Ultrasound examination revealed an abnormal motility of the stomach, while gastropexy was normal in aspect and no signs of HH were present. After two days the respiratory rate appeared to increase and violent vomiting started again. Consequently, it was decided to include maropitant citrate in the therapy as an antiemetic (1 mg/kg q24 *s.c.*) and to re-check the abdomen using ultrasound. Ultrasound examination was consistent with a recurrence of the HH by visualisation of the gastric wall through the diaphragmatic hiatus. A new oesophagogastroscopy

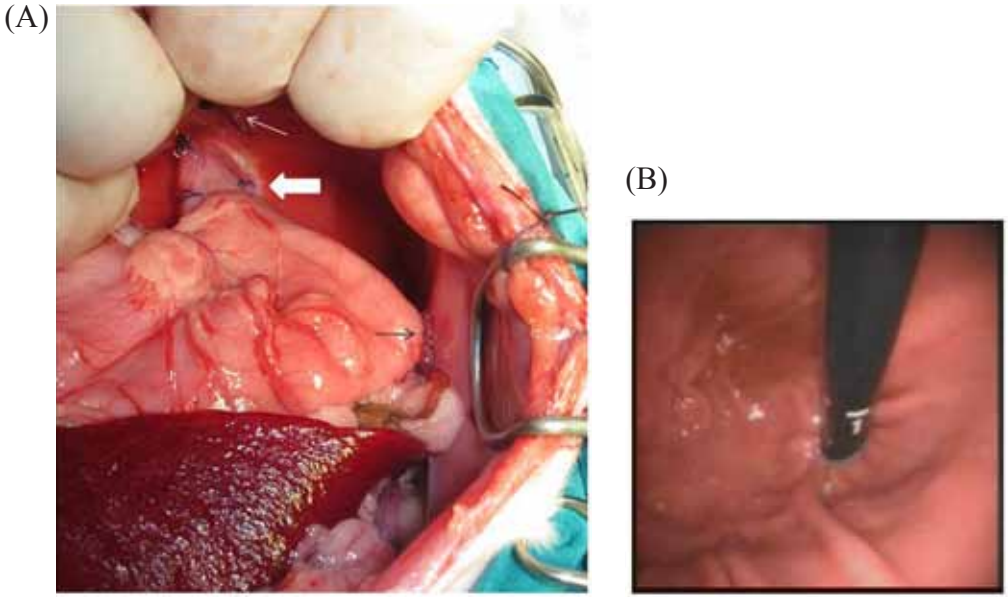

Figure 3. A = first surgery, intraoperative image of a kitten with type I hiatal hernia: it is possible to note the simple sutures of the oesophagopexy (wide white arrow point at one of the stitches), the mattress sutures of the hiatal plication (thin white arrow) and the left-flank incisional gastropexy (black arrow). B = retroverse view of the cardia, which appears reduced around the 6 mm endoscope

was performed and a second surgery was planned. The anaesthetic protocol was the same as for the previous surgery. After laparotomy the diaphragm appeared thinner than at the previous procedure, while the hiatus was still enlarged, allowing displacement of the proximal part of the stomach into the thorax. Similar to the previous operative procedure that had already been performed, a surgical approach was made, maintaining the endoscope inside the stomach. The gastropexy was resolved, and a dissection of the phrenico-oesophageal ligament was made dorso-laterally, taking care not to damage the dorsal and ventral vagal trunks. The stomach was pulled back through a vascular loop in the abdomen and an oesophagopexy was carried out using simple interrupted sutures between the diaphragm and the siero-muscular layers of the oesophagus. The pneumothorax induced was drained intraoperatively using thoraco-centesis through the right muscular part of the diaphragm. A new fun-

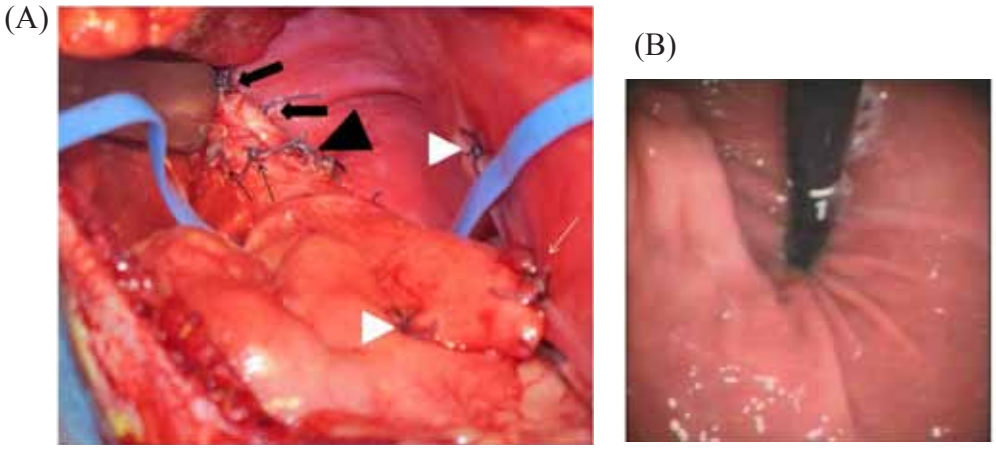

Figure 4. A = second surgery, intraoperative image of a kitten with recurrence of type I hiatal hernia: the white arrowheads indicate the site of the previous gastropexy that was solved; the white arrow points to the new incisional gastropexy; the thin black arrows show the stitches for the oesophagopexy, while the thicker black arrows outline the mattress sutures for the hiatal plication. The black arrowhead indicates the lower oesophageal sphincter that is back in the abdomen. B = retroverse view of the cardia, which appears reduced around the 6 mm endoscope

dus left flank incisional gastropexy was performed (Figure 4). The post-operative therapy consisted of antibiotics (ampicillin and sulbactam 20 mg/kg q12 *i.v.*), ranitidine (1 mg/kg q12 *i.v.* slowly), omeprazole (0.7 mg/kg q24 *i.v.*) and maropitant citrate (1 mg/kg q24 *s.c.*), with a soft low-fat diet with small and frequent meals, and the elevation of the cat for 5 min after eating. The cat was sent home seven days after surgery with ranitidine (1 mg/kg q12 *s.c.*), omeprazole (0.7 mg/kg q24 *o.s.*) and the special food regimen for 14 days.

Twenty days after surgery the cat was in good clinical condition, with increased body score and was presenting only sporadic vomiting with small amounts of food. At this time, an ultrasound control confirmed the normal anatomy of the lower part of the oesophagus through the hiatus in the abdomen.

At one year follow-up the cat was in good condition and no longer showed any signs related to the HH.

DISCUSSION AND CONCLUSIONS

Congenital hiatal hernia, as other congenital malformations (Pisoni et al. 2012; Tremolada et al. 2013) is an uncommon abnormality in dogs and rare in cats, but various reports are available in the literature (Gaskell et al. 1974; Teunissen et al. 1978; Peterson 1983; Ellison et al. 1987; Miles et al. 1988; Prymak et al. 1989; Bright et al. 1990; Williams 1990; Brinkley 1990; White 1993; Callan et al. 1993; Auger and Riley1997; Hardie et al. 1998; Lorinson and Bright 1998; Sivacolundhu et al. 2002; Rahal et al. 2003; Kirkby et al. 2005; Owen et al. 2005; Ardnt et al. 2006; Baig et al. 2006; Keeley et al. 2008; DeSandre-Robinson et al. 2011), most of which are congenital, such as, apparently, the case described in the present report. Despite the several cases reported, the pathogenesis is not completely understood and, similarly, the therapeutic protocol has not been standardised. Different types of hernia are reported in small animals, probably with different aetiologies. Consequently, various medical and surgical therapies have been proposed. For the symptomatic sliding hiatal hernia, or type I HH, medical therapy consists of alleviating the presumed reflux oesophagitis using sucralfate, H1 receptor antagonists like cimetidine or ranitidine, or proton pump inhibitors such as omeprazole. A prokinetic agent can improve the LES pressure and accelerate gastric emptying. The diet is also important; reducing the fat content, changing the consistency of the food, giving small amounts of food frequently and keeping the animal in a vertical position for some minutes after the meal can help oesophageal transit and gastric emptying (Sivacolundhu et al. 2002). Usually surgery is suggested only after failure to alleviate the symptoms by medical therapy. In the cat of the present report medical therapy was carried out only for ten days, due to the lack of improvement and because the owners were favourable to surgery as the cat experienced severe discomfort after eating.

The HH was suspected by survey radiography and abdominal ultrasound was helpful in recognising the recurrences. However, a definitive diagnosis was obtained using endoscopy. In our opinion endoscopy is the preferred diagnostic imaging technique, both for its ability to directly visualise the HH and for its suitability for examining the status of the oesophageal mucosa if the stomach is repositioned in the abdomen.

Several procedures are carried out alone or in combination to treat HH surgically: hiatal plication, oesophagopexy, several types of gastropexy and antireflux procedures. Hiatal plication is necessary to reduce an excessively wide opening in the diaphragm for passage of the oesophagus; oesophagopexy is essential to avoid sliding of the oesophagus; gastropexy helps in maintaining the stomach in a correct position; the antireflux procedures prevent gastro-oesophageal reflux from increasing the pressure of the LES (Sivacolundhu et al. 2002). Different techniques have been proposed to avoid gastro-oesophageal reflux (Leonardi et al. 1977), but the Nissen fundoplication is the most frequent technique performed (Bright et al. 1990; Sivacolundhu et al. 2002). In the case reported here the Nissen fundoplication could be indicated but, with reference to the literature, we decided not to perform it (Callan et al. 1993; White 1993; Lorinson and Bright 1998; Sivacolundhu et al. 2002), to avoid the severe complications reported: dysphagia, inability to belch, "gas bloat" and recurrence of stomach herniation with possible strangulation (Gaskell et al. 1974; Callan et al. 1993).

Thus, we opted for hiatal plication that could reduce the excessive widening of the oesophageal hiatus present. Thanks to the endoscope positioned in the stomach, it was possible to assess the effectiveness of the reduction of the hiatus and avoid an excessive constriction. The left-side gastropexy

helped in maintaining the position of the oesophagogastric junction, but a strong oesophagopexy was necessary to reposition the lower part of the oesophagus in the abdominal cavity. To maintain the oesophagogastric junction in position, during the second surgery it was necessary to dissect the oesophagophrenic ligament, pull back the lower part of the oesophagus in the abdomen and suture the ligament more cranially to the seromuscolar layer of the oesophagus. This procedure was necessary because of the malformation of the hiatus, which was anchored to the gastric wall instead of the oesophageal wall, or because it was too loose and abnormally wide.

It has been stated that hiatal plication and gastropexy are important for resolving hernias (Sivacolundhu et al. 2002), but oesophagopexy can also be essential in the surgical management of a sliding HH.

The trend in human medicine in recent years is to increase the use of minimally invasive surgery with the aim of a faster recovery even with the largest hiatal hernias, and to practice fundoplication to prevent gastro-esophageal reflux. In these cases a mesh is laparoscopically positioned to reduce the dimension of the hiatus (De Moor et al. 2012; Bell et al. 2013). To our knowledge, in veterinary medicine the laparoscopic approach has not been performed up to now, probably because of the low frequency of the pathology in dogs and cats and the lack of a standard procedure for the surgical therapy. In fact, it is advisable to choose a therapy for HH individually (Sivacolundhu et al. 2002). Moreover, data are not available about the use of a mesh in veterinary medicine to treat a recurrence of HH, so we decided first to implement the oesophagopexy. The pathogenesis is different in humans and dogs and cats, because HH in humans are frequently paraoesophageal and linked to age, while in small animals they are usually sliding and congenital (Sivaconundhu et al. 2002).

The post-operative management of patients is also an important consideration for success in treating hiatal hernia. This management is aimed at preventing vomiting and promoting a normal gastric flow, and at the same time at treating the oesophagitis.

With this report we wish to contribute to the discussion of the most suitable type of surgery to perform in cases of HH. Reduction of the oesophageal hiatus, oesophagopexy and gastropexy, performed simultaneously under endoscopic control, could be a good choice with minimal occurrence of surgical complications.

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Received: 2013–06–05
Accepted after corrections: 2014–03–29

Corresponding Author:

Luciano Pisoni, University of Bologna, School of Agriculture and Veterinary Medicine, via Tolara di Sopra, 50, 40064 Ozzano dell'Emilia, Bologna, Italy

Tel. +39 051 209 7537, E-mail: luciano.pisoni@unibo.it