

Pylorogastric intussusception in a puppy with a congenital intrahepatic portosystemic shunt – a case report

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Abstract

A sixteen-week-old female Akita-inu puppy was presented for investigation of acute vomiting, diarrhoea, and abdominal distension. Abdominal ultrasound revealed a single congenital right-sided intrahepatic portosystemic shunt and pylorogastric intussusception, which spontaneously resolved during the next three days after initial examination. Owners elected for euthanasia due to the clinical deterioration of the patient that reflected in development of clinical signs associated with liver insufficiency, guarded prognosis and financial concerns for transjugular coil embolisation.

Dog, ascites, proximal duodenum, retrograde invagination

An intussusception is defined as a prolapse of one part of the intestine into the lumen of an immediately adjoining part (Anderson 1994) and has been reported in both humans and animals (Carr et al. 1976; Anderson 1994; Bowersox et al. 1991; Applewhite et al. 2001; Broome et al. 2004; Cole et al. 2005; Barreau 2008; De Brito Galvao et al. 2011; Berent and Tobias 2012; Choi et al. 2012; Bertolini 2017). Intussusceptions are named by the anatomic components of the intussusceptum (the invaginated segment) and the intussusciens (the invaginating segment) (Lewis and Ellison 1987). In dogs and cats, intussusceptions most commonly occur at the ileoceocolic junction (Carr et al. 1976; Marks 1983; Leib and Blass 1984; Lewis and Ellison 1987; Bowersox et al. 1991; Huml et al. 1992; Lamb and Mantis 1998; Applewhite et al. 2001; Patsikas et al. 2003; Barreau 2008; Lideo et al. 2010).

Portosystemic shunts (PSSs) are macroscopic vascular connections between the portal vein and a systemic vein such as the caudal vena cava. These abnormal communications allow the portal blood from the intestine to bypass the liver and enter the systemic circulation. Portosystemic shunts can be classified as congenital or acquired, single or multiple, and intra- or extrahepatic (Lamb 1996; Lamb and Daniel 2002; Tillson et al. 2002; Santilli and Gerboni 2003; Broome et al.; 2004; Cole et al.; 2005). Congenital portosystemic shunts (CPSS) are vascular anomalies that occur secondary to inappropriate closure of different portions of the foetal vasculature, resulting in an intra- or extrahepatic CPSS. Typically, a single CPSS is present, although multiple shunts have been reported (Leeman et al. 2013). This report describes the first case of an intrahepatic PSS and concurrent pylorogastric intussusception in a puppy.

Case presentation

A sixteen-week-old female intact Akita-inu puppy was presented for investigation of acute vomiting, diarrhoea and abdominal distension. On physical examination, the

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puppy was lethargic, mildly ataxic, tachycardic (140 bpm) and had a distended abdomen with dull sounds on abdominal percussion. Serum biochemical abnormalities included hypoproteinaemia (17.8 g/l [reference interval {RI} 55–75 g/l]), hypoalbuminaemia (6.8 g/l [RI 23–34 g/l]), low urea (1.4 mmol/l [RI 3.3–8.3 mmol/l]), hypocholesterolaemia (2.08 mmol/l [RI 3.5–7.8 mmol/l]), hypocalcaemia (1.95 mmol/l [RI 2.30–3.00 mmol/l]), hyperammonaemia (69.19 μ l [RI < 50 μ mol/l]). Haematological abnormalities included moderate nonregenerative anaemia (RBC 4.41×10^{12} [RI $5.5\text{--}8.5 \times 10^{12}$], HCT 0.22 l/l [RI 0.37–0.55 l/l], HGB 66 g/l [RI 120–180 g/l], MCV 49 fl [RI 65–75 fl], MCH 15 pg [RI 22–25 pg], thrombocytopenia (135×10^9 [RI $200\text{--}500 \times 10^9$]) and monocytosis (0.62×10^9 [RI $0.00\text{--}0.50 \times 10^9$]).

Abdominal ultrasound (US) examination was performed using a 5–9 MHz microconvex and a 9–15 MHz linear probe (Vivid 7, GE Medical System, Horten, Norway) in order to evaluate the cause of the distended abdomen. The parenchymal organs were pushed to the periphery by hypoechoic free fluid and not well accessible, therefore, ultrasound assisted abdominocentesis was performed and approximately 1000 ml of transudate were removed before thorough ultrasound examination. Additional abnormal ultrasonography findings included a small and diffusely hyperechoic liver without the visualisation of parenchymatous branching of the portal vessels. Moreover, an abnormal tortuous course of the intrahepatic vessel connecting the portal vein and caudal vena cava was identified in the right liver lobes with the maximal diameter at the level of the portal vein of 20 mm. Mean peak systolic velocity in the portal vein was 25 cm/s (a hepatopetal flow) measured by Pulse Wave Doppler. In addition to an intrahepatic portosystemic shunt, pylorogastric intussusception was detected on the ultrasonographic examination. The pylorus was displaced into the body and fundus. Multiple echogenic and echolucent rings were visible on transverse section (Plate I, Figs 1, 2). Colour Doppler ultrasonography revealed blood flow in vessels of the intussuscepted segment of stomach (Plate II, Fig. 3). The small intestines had increased echogenicity of the mucosal layer and decreased distinction between different intestinal layers with pronounced peristalsis on subjective evaluation. There was a small amount of pleural effusion present in the thorax detected by thoracic ultrasound. Ultrasonographic examination was repeated one day later and the pylorogastric invagination was still present.

As owners were considering surgery at this point, portal phase contrast computed tomography (CT) (GE LightSpeed 16-Slice, Milwaukee, Wisconsin, USA; 120 kV, automatic mA, 2.5 mm slice thickness in SD algorithm, retrospective reconstruction 1.25 mm, tube rotation time 0.5 s, and a pitch of 1.375) was performed three days after initial examination with the dog in sternal recumbency under general anaesthesia with butorphanol (0.04 mg/kg IV; Butomidol, Richter Pharma AG, Wels, Austria) and propofol (4 mg/kg IV; Propofol-lipuro 1%, B. Braun, Melsungen, Germany). The scanned area extended from the middle thoracic region to the seventh lumbar vertebrae. A large aberrant vessel originating from the portal vein and leading to the caudal vena cava was detected in the right lateral liver lobe (Plate II, Fig. 4). Pancreaticoduodenal and splenic veins had a normal anatomical localisation and blood flow. The stomach had a normal position, the duodenum and the remaining parts of the gastrointestinal tract had a normal anatomical localisation. There was free fluid in the mesogastrium (11 HU; Hounsfield units). There was a small amount of free fluid, bilaterally, in the pleural space (9 HU), however, no other abnormalities in the thorax were detected.

Due to the high surgical costs of percutaneous transjugular coil embolisation (PTCE), the owners elected medical management and the puppy was treated systemically with amoxicillin clavulanic acid (12.5 mg/kg PO q12h; Synulox, Zoetis, Parsippany-Troy Hills, New Jersey, USA), lactulose (5 ml PO q8h; Dulphalac®, Abbott-Laboratories, Chicago, Illinois, USA), famotidine (0.5 mg/kg PO q24; Famosan, PRO.MED.CS, Prague, Czech

Republic) and probiotics (1 g PO q24; Fortiflora, Purina, St. Louis, Missouri, USA). Three weeks later, an abdominal US revealed a large amount of free anechogenic fluid in the peritoneal cavity. Ultimately, the owners elected for euthanasia due to the clinical deterioration, poor prognosis and financial concerns for PTCE.

Discussion

Intussusceptions may occur at any location within the alimentary tract and most invaginations occur in the direction of normal peristalsis (Marks 1983; Leib and Blass 1984; Lewis and Ellison 1987; Watson 1997). However, there have been few reports of invaginations in the retroperistaltic direction, for example within the small intestine and in other parts of the alimentary tract such as gastro-oesophageal, pylorogastric, gastro-gastric, duodenogastric intussusceptions (Wilson and Burt 1974; Marks 1983; Leib and Blass 1984; Lewis and Ellison 1987; Bowersox et al. 1991; Huml et al. 1992; Lamb and Mantis 1998; Applewhite et al. 2001; Patsikas et al. 2003; Barreau 2008; Lideo et al. 2010). The puppy in this report had a pylorogastric intussusception in which the pylorus and proximal duodenum were invaginated in a retrograde direction into the body of the stomach. Pylorogastric intussusceptions have only been reported in eight cases in dogs (Wilson and Burt 1974; Marks 1983; Applewhite et al. 2001; Heechun et al. 2005; Lee et al. 2005; De Brito Galvao et al. 2011; Choi et al. 2012). Pylorogastric intussusceptions have also been described in one case as gastrogastic intussusception and in two cases as duodenogastric intussusception (Bowersox et al. 1991; Huml et al. 1992; Allman and Pastori 2013). Pylorogastric and gastro-gastric intussusceptions are used as synonyms by some authors but gastro-gastric intussusception not involving the pylorus was also described (Reymond 1971).

Intestinal intussusceptions are most commonly seen in young dogs (Wilson and Burt 1974; Weaver 1977; Lewis and Ellison 1987; Levitt and Bauer 1992). The aetiology of the majority of intussusceptions is unknown, however, in young dogs, enteritis (e.g. viral, parasitic, or immune-mediated), foreign bodies, neoplasia and general anaesthesia followed by or without an abdominal surgery, have been identified as possible predisposing factors (Reymond 1971; Reymond 1972; Lewis and Ellison 1987; Levitt and Bauer 1992; Kipnis 1977). Other conditions causing severe vomiting, such as kidney failure or uraemia, can also be a predisposing factor for intussusceptions. Moreover, there have been reports in human medicine of intussusceptions with chronic renal failure (Carr et al. 1976; Long et al. 2010). The aetiology of a pylorogastric intussusception, as in other intussusception types, is unclear. Pylorogastric intussusception is a retrograde invagination and retroperistalsis most likely plays a major role in its aetiology. Retroperistalsis usually occurs as a precursor to vomiting, which results in gastrointestinal contents to move in the opposite direction. All previously published cases regarding intussusceptions described a presentation of vomiting (Marks 1983; Huml et al. 1992; Applewhite et al. 2001; Heechun et al. 2005; De Brito Galvao et al. 2011; Choi et al. 2012). However, it is not clear whether vomiting is the cause of the pylorogastric intussusception or its consequence. In this case, the puppy also had a congenital PSS which can cause vomiting and therefore it is likely that this contributed to the occurrence of a pylorogastric intussusception. There was a spontaneous resolution of the intussusception in this case between the recheck ultrasound examination and the following CT scan two days later, which has also been reported in other cases (Heechun et al. 2005; De Brito Galvao et al. 2011). The reason for spontaneous resolution of pylorogastric intussusceptions in a high number of cases is unclear, however, one of the reasons could be the large difference of the lumen size between the gastric fundus (the intussusciens) and pylorus (the intussusceptum). The hydrostatic pressure of the fluid accumulating in the stomach due to the obstruction of the pylorus can

also be one of the reasons for the spontaneous resolution of pylorogastric intussusceptions.

There have been reports of diagnosing pylorogastric intussusceptions based on ultrasonographic findings of the abdominal cavity (Bowersox et al. 1991; Applewhite et al. 2001; Heechun et al. 2005; Pennick 2008; Choi et al. 2012). The pylorogastric intussusception in this case has also been diagnosed on abdominal ultrasound. Other diagnostic techniques could be implemented as well. One case report used endoscopy after unspecific findings of abdominal radiographs that revealed a mechanical obstruction of the pylorus (De Brito Galvao et al. 2011). Alternatively, two cases described that the diagnosis was made during exploratory laparotomy after unspecific findings of abdominal radiographs (Bowersox et al. 1991; Watson 1997). Moreover, the puppy also presented with severe ascites, which did not resolve throughout the follow-up. Ascites is not a typical finding in animals with CPSS and is usually an indication of portal hypertension and subsequent multiple acquired shunts but in this case hepatopetal flow was detected in portal vein by pulse-Doppler. It may also occur with severe hypoalbuminaemia (Berent and Tobias 2012). In this case, the puppy had unresolved hypoproteinaemia and hypoalbuminaemia, which was the most likely cause of the abdominal effusion. On abdominal ultrasound, the small intestines had increased echogenicity of the mucosal layer and there was decreased distinction between the different intestinal layers with pronounced peristalsis which could have been the cause or consequence of hypoalbuminaemia. Hypoalbuminaemia was most likely the result of liver insufficiency and the cause of decreased distinction between the intestinal layers.

This report is the first in veterinary literature that describes the occurrence of an intrahepatic PSS and concurrent pylorogastric intussusception. Pylorogastric intussusception is a rare type of intussusceptions of the gastrointestinal tract causing a mechanical obstruction of the stomach. Pylorogastric intussusceptions can resolve spontaneously, however, they can be life threatening and the diagnosis can be made based on typical ultrasonographical findings of the abdominal cavity.

Conflict of Interest

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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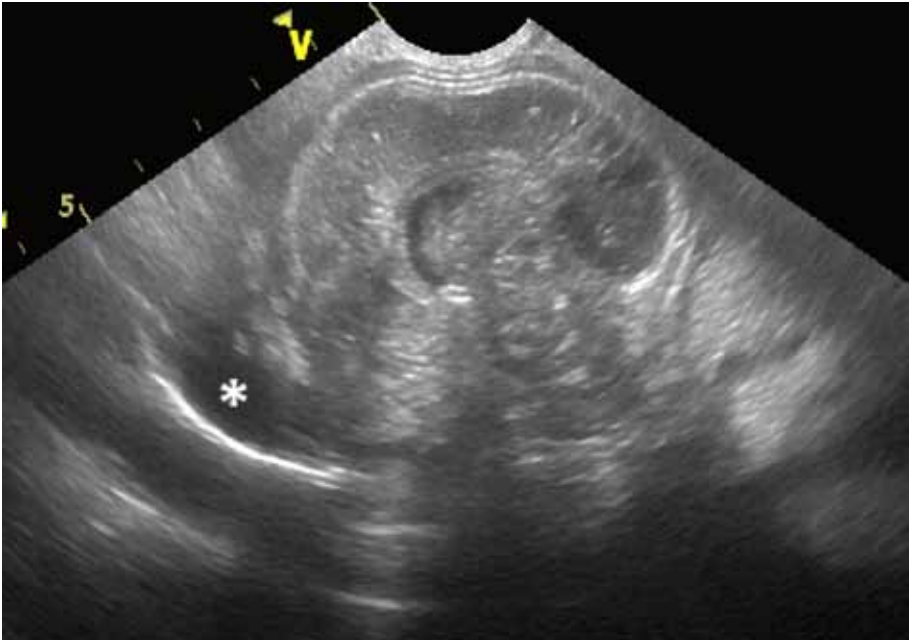


Fig. 1. Ultrasonographic examination of the pylorogastric intussusception. Oblique transverse image of the pyloric region. Multiple echogenic and echolucent rings were visible. There was also evidence of free abdominal fluid (*).

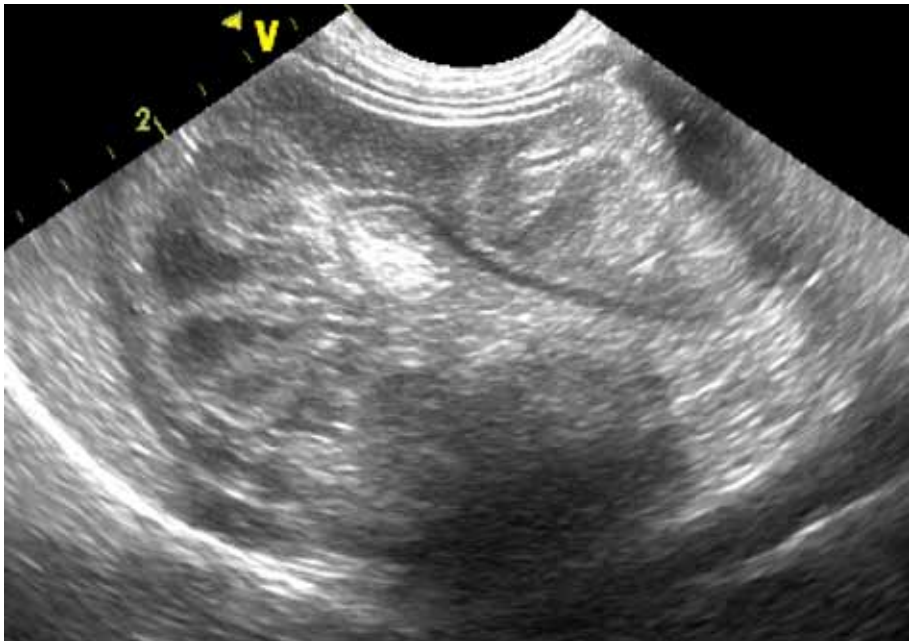


Fig. 2. Ultrasonographic examination of the pylorogastric intussusception. Sagittal image of the intussusception. Pylorus was displaced into the fundus.

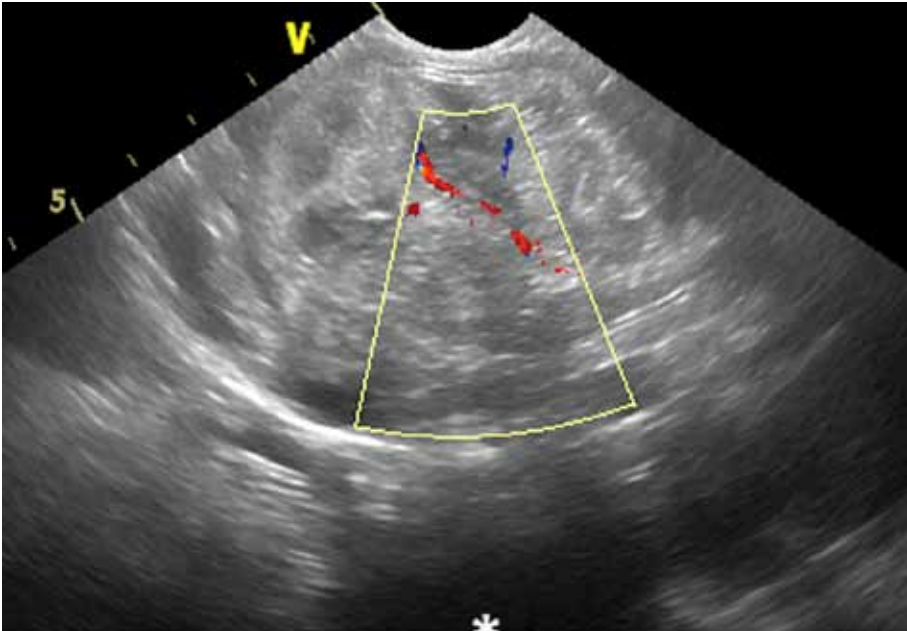


Fig. 3. Colour Doppler examination of the pylorogastric intussusception confirmed blood flow in vessels of the intussuscepted segment of the stomach.

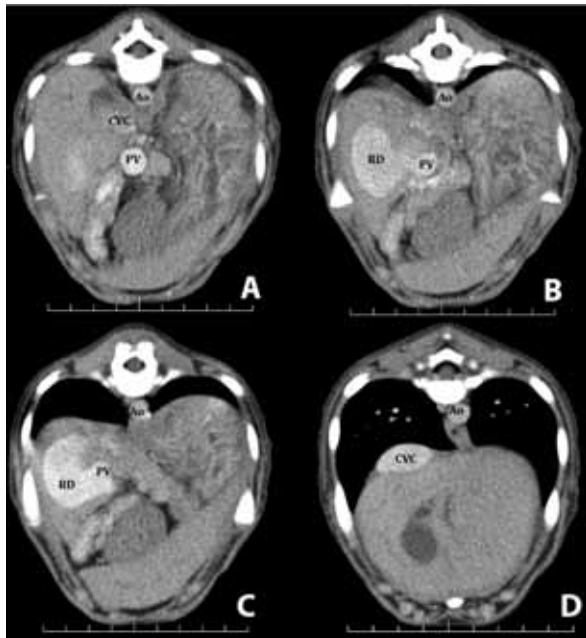


Fig. 4. Contrast-enhanced computed tomographic images of the liver obtained during the portal phase (A–D). From caudal to cranial. A right-sided intrahepatic portosystemic shunt (RD) originates from the right hepatic portal vein (PV) and loops into the right liver, forming a wide loop before entering the caudal vena cava (CVC).