IMAGING DIAGNOSIS-A CASE OF SPONTANEOUS HEPATIC PORTAL VEIN GAS IN AN 11-MONTH-OLD WEST HIGHLAND WHITE TERRIER

A Case of Spontaneous Hepatic Portal Vein Gas in an Eleven Month Old West Highland White Terrier

Jennifer A Cartwright

Craig Breheny

Alison C Major

Tracy L Hill

Adam G Gow

Royal (Dick) School of Veterinary Studies and The Roslin Institute, Division of Veterinary Clinical Studies, The University of Edinburgh, Hospital for Small Animals, Easter Bush Veterinary Centre, Roslin, Midlothian EH25 9RG

Resident: Jennifer Ann Cartwright Jennifer.Cartwright@ed.ac.uk

Corresponding Author: adam.gow@ed.ac.uk

Keywords: Canine, necrotizing colitis, colonic ulceration, mucosal gas

Running Head: Portal Vein and Hepatic Gas in a Young Dog

Funding Sources: none

Previous presentations or abstracts: This case has been presented at BSAVA Scottish Congress 2015

Abstract:

An 11-month old female entire West Highland White Terrier presented for chronic diarrhoea with acute deterioration in demeanour and progression to systemic inflammatory response syndrome. Transcutaneous abdominal ultrasonography identified colonic ulceration and secondary mucosal gas. Suspected hepatic portal vein gas and hepatic parenchyma gas were also visualised. The patient was stabilised and managed for ulcerative colitis. Based on endoscopic biopsies, the dog was diagnosed with severe, chronic, pyogranumolatous colitis. On repeat ultrasonographic evaluation the portal vein and hepatic gas had resolved but the patient deteriorated and was ultimately euthanised due to sepsis.

Signalment, History, and Clinical Findings

An 11 month old, female neutered, West Highland White Terrier was presented with a 3 week history of diarrhoea and progressive anorexia. She was up to date with vaccinations and worming prophylaxis. In the 3 weeks prior to presentation the diarrhoea had progressed to haematochezia with tenesmus and had increased in frequency. She also began to vomit undigested food and had lost 10% body weight. The dog deteriorated acutely in the 12 hours prior to presentation, with development of abdominal pain, abdominal distension and extreme weakness. In the 3 weeks prior to referral, she had been previously treated with Maropitant, intravenous fluid therapy and probiotics but clinical signs persisted.

She was presented with a sinus tachycardia of 240 beats/minute with weak pulses, hypothermia at 36.4°C, hypotension with a systolic blood pressure of 80mmHg, and her respiratory rate was 40 breaths/minute. The combination of these clinical signs are consistent with systemic inflammatory response syndrome (SIRS). She was in poor body condition (3/9) and was estimated to be 7% dehydrated, based upon the loss of skin turgor and presence of a skin tent. Her mucous membranes were pink, tacky and had a capillary refill time of <2 seconds. Thoracic auscultation was unremarkable. The abdomen was distended and markedly painful on palpation cranially.

Initial treatment included fluid resuscitation with lactated ringers and colloid support intravenously (6% hydroxyethyl starch 130/0.4 in 0.9% sodium chloride). She was warmed, given 0.1mg/kg methadone intravenously and inspired air was supplemented with oxygen during stabilisation. Both the dog's blood pressure and demeanour improved in response to treatment and continued to do so over the following days.

Serum biochemistry revealed hypoalbuminaemia, hypokalaemia, mild total hypocalcaemia with ionised calcium within reference range. There were moderate elevations in alkaline phosphatase, bile acids and alanine transferase. There was also a marked elevation in total bilirubin. Haematology revealed a mild leucocytosis of 16.7 $x10^9$/l (reference range 6 – 15 x 10^9/l) mainly composed of a monocytosis of 2.34 x 10^9/l (reference range 0 – 1.5 x 10^9/l). Urinalysis revealed bilirubinuria and haematuria with a specific gravity of 1.008. SNAP® cPL™ Test (Idexx Laboratories, Inc: North Grafton, MA) and SNAP® Parvo Test (Idexx Laboratories, Inc: North Grafton, MA) snap tests were both negative.

Prothrombin time was within normal limits, while activated partial thromboplastin time was moderately prolonged (Table 1). Faecal analysis was negative for parasites including *Giardia*, *Salmonella* and *Campylobacter*. Baseline cortisol excluded hypoadrenocorticism.

Imaging, Diagnosis and Outcome:

Transcutaneous ultrasonography of the abdomen was performed, on the day of presentation, using the Logiq 9 General electric (GE Healthcare, Little Chalfont, Buckinghamshire, United Kingdom) with a linear 9-12MHz transducer and a microconvex 5-8MHz transducer. On the day of presentation, abdominal ultrasonography identified that the colon wall was markedly thickened, especially within the descending colon (up to 5mm in places) with complete loss of layering. The presence of luminal gas precluded accurate assessment of overall colon diameter. There was a large volume of fluid and gas within the colonic lumen There were also multiple areas of hyperechoic foci with associated comet-tail artefact within the colonic mucosa, likely representing ulceration and gas infiltration into the colon wall. (Fig. 1). The remaining gastrointestinal tract wall thickness was within accepted limits with normal layering. The small intestine was fluid filled throughout its length. The left medial iliac and jejunal lymph nodes were mildly enlarged, at 6mm and 8mm respectively. There was a small volume of anechoic peritoneal effusion visible throughout the abdomen There were multiple mobile hyperechoic foci peripherally within the liver parenchyma, which demonstrated associated dirty acoustic shadowing and comet tail artefact and they were mobile against gravity (Fig. 2 and video 1). These same mobile foci were also identified within the portal vein moving hepatopetaly (video 2). The presence of hyperechoic foci within the portal vein, hepatic parenchyma and colonic wall was thought

to be due to ulceration of the colonic wall. Differentials for this ulceration would include; marked inflammatory bowel disease with resultant ulcerative colitis, infectious process such as E. *coli* or a neoplastic process. The portal vein and hepatic parenchymal mobile hyperechoic foci were most likely gas from the colon. Other possible differentials for hyperechoic foci within the liver, such as mineralisation or pneumobilia, were considered unlikely due to their appearance and location (1). The foci were mobile, which would not be consistent with mineralisation and were present within the hepatic associated vasculature rather than the biliary system. In addition the presence of dirty acoustic shadowing and comet-tail artefact is more typical of gas. Radiographs of the abdomen, taken the day after presentation, were unremarkable apart from some loss of serosal detail consistent with the mild peritoneal effusion and gas could not be visualized within the liver.

Fresh frozen plasma was given as treatment for prolonged activated partial thromboplastin time. Medications included maropitant (Cerenia®), metoclopramide (Emeprid®) and omeprazole (Losec®). Intravenous metronidazole and enrofloxacin (Baytril®) were administered. Nutrition was provided through a naso-oesophageal feeding tube initially and subsequently an oesophageal feeding tube.

Following stabilisation of the patient, gastrointestinal endoscopy identified ulcerative lesions within the colon (Fig. 3). Biopsies were taken and sent for histopathology[f]. These revealed diffuse ulceration and heavy infiltration of macrophages and neutrophils throughout all sections, consistent with severe, chronic, diffuse pyogranumolatous colitis.

Forty-eight hours after admission a second ultrasound revealed an improvement in the intrahepatic and colonic mucosa hyperechoic foci and no hyperechoic foci were detected in the portal vein. Seven days after initial presentation, ultrasonography revealed resolution of the hyperechoic foci in the hepatic parenchyma and portal vein, although these were still present within the colonic mucosa.

After 3 weeks of treatment, and despite initial improvement, the dog deteriorated acutely. She developed signs of septic shock alongside the development of an antebrachial abscess and infection of the oesophageal feeding tube stoma site. She was still receiving antibiotic therapy, so a multidrug resistant bacterial infection was suspected. At this stage the owners decided against further treatment and elected for euthanasia without a post mortem.

Discussion:

Portal vein gas has been described in one dog, iatrogenically, after oral hydrogen peroxide administration, which is also reported as a cause in the human literature (2, 3). Spontaneous Hepatic portal vein gas in a dog as a result of underlying gastrointestinal disease has not been previously reported.

HPVG was first described in infants secondary to necrotising enterocolitis and was associated with a poor prognosis (4). It is also associated with hypertrophic pyloric stenosis, blunt force abdominal trauma, gastric dilatation, secondary to gastrointestinal endoscopy and hepatic biopsy, ulcerative colitis and abdominal abscessation in people (5-8).

Hepatic portal vein gas was initially thought to be associated with a high mortality and an indication for surgery in people (9). Following the increased use of ultrasound in patient evaluation, hepatic portal vein gas has been more commonly identified giving rise to the question of whether the condition is benign or noxious (10, 11). In recent years, with advances in imaging capabilities, the detection of portal vein gas at an earlier stage in disease process has resulted in a better prognosis and is no longer a direct indication for surgery unless a suitable underlying cause is identified. Computed tomography (CT) is now routinely used in people to diagnose and confirm the presence of intestinal pneumatosis and hepatic portal vein gas (12). Pathogenesis of this clinical finding is reported to be multifactorial in people, including increased mucosal permeability, increased colonic luminal pressure, sepsis and immune compromise (10).

In this case, it is suspected that the gas within the colonic mucosa, due to severe ulcerative colitis, translocated into the colonic veins before being transported through the hepatic portal vein, where it became trapped within the hepatic parenchyma. The pyogranulomatous inflammation in this case could be consistent with a response to pathogenic bacteria or fungi. While Histoplasmosis would be a leading cause in other regions of the world it is not a common pathogen in the United Kingdom, favouring warm and humid environments. The patient was treated with Enrofloxacin for potential E. *coli* infection from the outset, although subsequently developed an oesophageal feeding tube stoma site infection, from which a resistant bacteria, an extended spectrum beta-lactamases (ESBL) E. coli, was isolated, thus remaining a potential differential diagnosis. Severe immune-mediated, inflammatory bowel disease localised to the colon is also a possibility as no pathogens were identified in the diagnostics performed. The full

significance of finding hepatic portal vein gas at presentation was unknown due to the previous lack of literature in dogs. It did highlight to us a need for aggressive support and serial patient evaluations, given the previously identified poor prognosis in some human cases (10). Broad spectrum antibiotic therapy beyond correction of the SIRS was continued, as we felt that it was likely that colonic bacteria would also be entering the vasculature and hepatic parenchyma if the colonic wall was compromised to the extent that gas could translocate (13, 14).

The significance of hepatic portal vein gas in this dog along with the rapid resolution is unknown, but transient hepatic portal vein gas has been identified in critically ill people after management of shock (15). Transient hepatic portal vein gas has also been reported in cases of ulcerative colitis in humans, often following endoscopy, which is likely a reflection of poor mucosal integrity, as in this case. This is most often a benign finding in these cases requiring only conservative management, only one out of a total of nine patients required surgical intervention, a case in which the patient had Crohn's disease (16). It may be possible that more critically unwell dogs or those with severe gastrointestinal disease have transient undiagnosed hepatic portal vein gas. CT was not used in this case, and could be useful for future suspected cases to investigate this condition. Whilst ultrasound has comparable sensitivity and accuracy, in people CT is considered the gold standard as it also allows early detection of associated pathology (17) such as intraluminal gas (Pseudo-pneumatosis), abdominal abscess and localized pneumoperitoneum (18). Hepatic portal vein gas is more frequently recognised in human medicine in various disease states and subsequently the management of these patients is being tailored rather than decisions being made solely on the discovery of gas. With the increased availability and capability of

advanced veterinary diagnostic imaging there is every possibility we will be able to identify these changes in our patients and go on to identify their relative significance. This dog did have changes consistent with HPVG though it is not clear whether the presence of the hepatic portal vein gas was correlated with the poor outcome in this case.

Acknowledgements

The authors would like to thank Elizabeth Munro and Evi Pecceu.

Footnote

Gastrointestinal Laboratory, Department of Small Animal Clinical Sciences, Texas A&M University

References:

1. Yarze JC, Markowitz DM. Distinguishing between hepatic portal vein gas and pneumo(aero)bilia. Liver Transplantation2007;13(10):1476-.
2. Faverzani S, Lodi M, Pagetti S. Imaging diagnosis--portal venous gas in a dog. Vet Radiol Ultrasound2009 May-Jun;50(3):312-3.
3. Lewin M, Pocard M, Caplin S, Blain A, Tubiana JM, Parc R. Benign hepatic portal venous gas following caustic ingestion. Eur Radiol2002 Dec;12 Suppl 3:S59-61.
4. Stevenson JK, Graham CB, Oliver Jr TK, Goldenberg VE. Neonatal necrotizing enterocolitis: A report of twenty-one cases with fourteen survivors. The American Journal of Surgery1969 8//;118(2):260-72.
5. Sarti J, Kennedy A. Portal venous gas in hypertrophic pyloric stenosis. Journal of pediatric surgery2006 Nov;41(11):1935-6.
6. Gosink BB. Intrahepatic gas: differential diagnosis. AJR American journal of roentgenology1981 Oct;137(4):763-7.
7. Dill-Macky MJ. Benign hepatic portal venous gas following blunt abdominal trauma. Australasian Radiology1997;41(2):166-8.
8. Brown MA, Hauschildt JP, Casola G, Gosink BB, Hoyt DB. Intravascular Gas as an Incidental Finding at US after Blunt Abdominal Trauma. Radiology1999;210(2):405-8.
9. Berne TV, Meyers HI, Donovan AJ. Gas in the portal vein of adults with necrotizing enteropathy. The American Journal of Surgery1970 8//;120(2):203-9.
10. Abboud B, El Hachem J, Yazbeck T, Doumit C. Hepatic portal venous gas: physiopathology, etiology, prognosis and treatment. World J Gastroenterol2009 Aug 7;15(29):3585-90.

11. Wayne E, Ough M, Wu A, Liao J, Andresen KJ, Kuehn D, et al. Management algorithm for pneumatosis intestinalis and portal venous gas: treatment and outcome of 88 consecutive cases. Journal Gastrointest Surg2010 Mar;14(3):437-48.
12. Berona K, Hardiman K, Mailhot T. Hepatic Portal Venous Gas: Findings on Ultrasound and CT. Western Journal of Emergency Medicine2014;15(7).
13. Kashani A, Chitsazan M, Che K, Garrison RC. Bacteremia in a Patient with Ulcerative Colitis. 2014;2014.
14. Johansson MEV, Gustafsson JK, Holmén-Larsson J, Jabbar KS, Xia L, Xu H, et al. Bacteria penetrate the normally impenetrable inner colon mucus layer in both murine colitis models and patients with ulcerative colitis. Gut2014;63(2):281.
15. Coulier B, Van den Broeck S, Coppens JP. Transient and rapidly resolving intrahepatic portal gas: CT findings. JBR-BTR2008 Sep-Oct;91(5):214-6.
16. Shinagawa T, Hata K, Watanabe T. Hepatic Portal Venous Gas After Colonoscopy for Ulcerative Colitis: A Case Report. Journal Crohns Colitis2015 Jul 17.
17. McElvanna K, Campbell A, Diamond T. Hepatic portal venous gas--three non-fatal cases and review of the literature. Ulster Med Journal2012 May;81(2):74-8.
18. Wang JH, Furlan A, Kaya D, Goshima S, Tublin M, Bae KT. Pneumatosis intestinalis versus pseudo-pneumatosis: review of CT findings and differentiation. Insights Imaging2011 Feb;2(1):85-92.

Figures

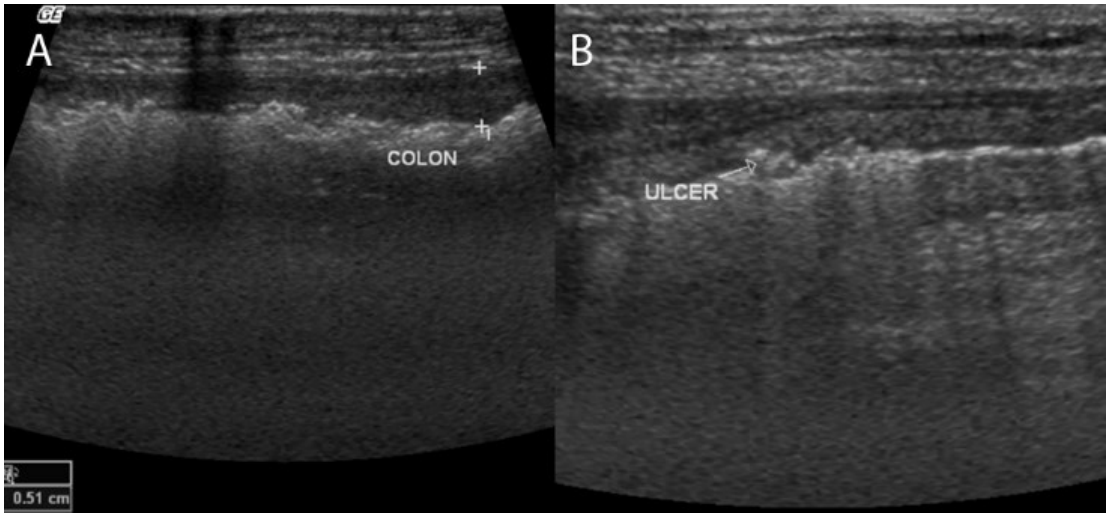

Fig. 1 –Ultrasound of the descending colon at presentation, longitudinal sections from left lateral approach, 9-12MHz linear probe. The colonic wall is markedly thickened (up to 5mm), particularly the descending colon, and there is marked loss of normal layering (1A). There is a large volume of gas within the colonic lumen. There are focal areas of hyperechoic

speckling within the colon wall, with associated comet-tail artefact, suspected to be gas within the mucosa, consistent with colonic ulceration (1B).

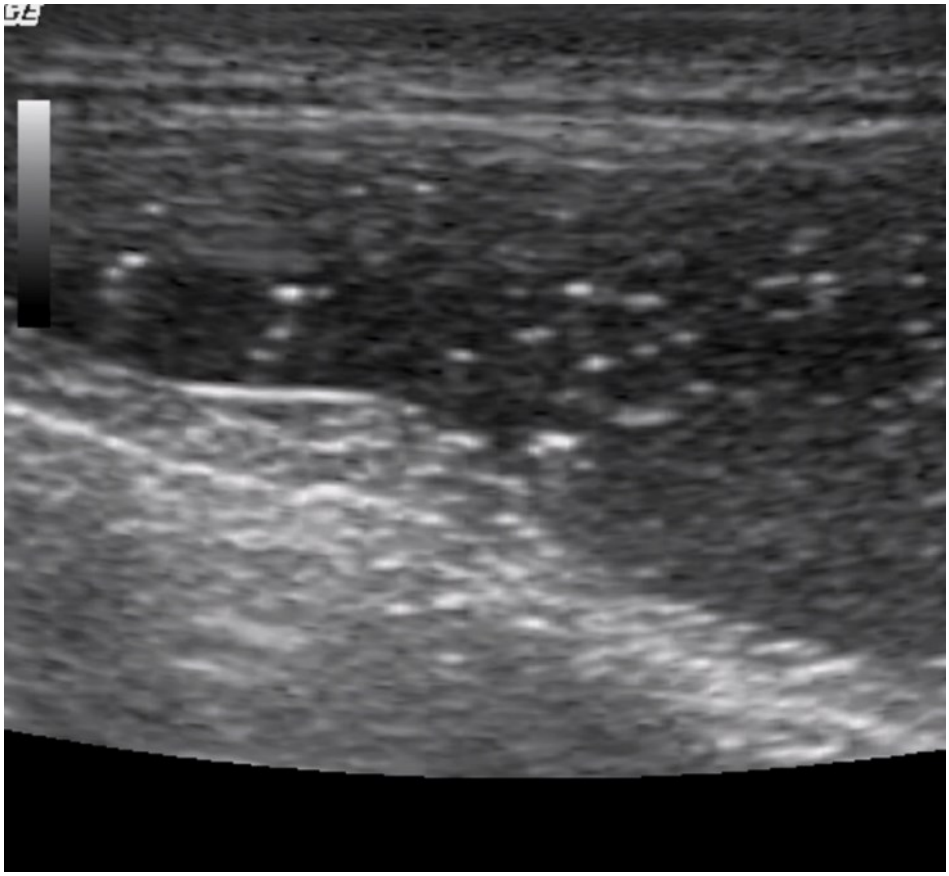

Fig. 2 – Ultrasound of the liver at presentation, parasagittal from ventral midline, 9-12MHz linear probe. There are multiple hyperechoic foci within the liver parenchyma which have associated comet-tail artefact and are mobile, this mobility was not related to the dogs breathing. Additional hyperechoic foci are seen moving within the portal vein moving hepatopetally (see video).

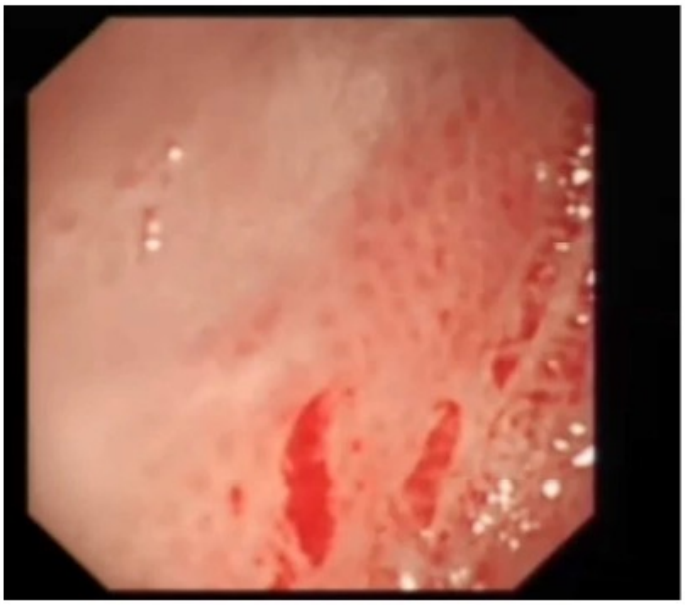

Fig. 3 Colonic mucosa viewed endoscopically demonstrating diffuse mucosal oedema, gross ulceration and focal areas of haemorrhage.
101x88mm (96 x 96 DPI)