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1 **A Case of Spontaneous Hepatic Portal Vein Gas in an Eleven Month Old West**

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16

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

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20

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23

24

25

26 **Abstract:**

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

36 **Signalment, History, and Clinical Findings**

37

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

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

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

60 Serum biochemistry revealed hypoalbuminaemia, hypokalaemia, mild total hypocalcaemia
61 with ionised calcium within reference range. There were moderate elevations in alkaline
62 phosphatase, bile acids and alanine transferase. There was also a marked elevation in total
63 bilirubin. Haematology revealed a mild leucocytosis of $16.7 \times 10^9/l$ (reference range 6 – 15 x
64 $10^9/l$) mainly composed of a monocytosis of $2.34 \times 10^9/l$ (reference range 0 – 1.5 x $10^9/l$).

65 Urinalysis revealed bilirubinuria and haematuria with a specific gravity of 1.008. SNAP® cPL™
66 Test (Idexx Laboratories, Inc: North Grafton, MA) and SNAP® Parvo Test (Idexx Laboratories,
67 Inc: North Grafton, MA) snap tests were both negative.

68

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

72

73 **Imaging, Diagnosis and Outcome:**

74

75 Transcutaneous ultrasonography of the abdomen was performed, on the day of
76 presentation, using the Logiq 9 General electric (GE Healthcare, Little Chalfont,
77 Buckinghamshire, United Kingdom) with a linear 9-12MHz transducer and a microconvex 5-
78 8MHz transducer. On the day of presentation, abdominal ultrasonography identified that
79 the colon wall was markedly thickened, especially within the descending colon (up to 5mm
80 in places) with complete loss of layering. The presence of luminal gas precluded accurate
81 assessment of overall colon diameter. There was a large volume of fluid and gas within the
82 colonic lumen There were also multiple areas of hyperechoic foci with associated comet-tail
83 artefact within the colonic mucosa, likely representing ulceration and gas infiltration into
84 the colon wall. (Fig. 1). The remaining gastrointestinal tract wall thickness was within
85 accepted limits with normal layering. The small intestine was fluid filled throughout its
86 length. The left medial iliac and jejunal lymph nodes were mildly enlarged, at 6mm and
87 8mm respectively. There was a small volume of anechoic peritoneal effusion visible
88 throughout the abdomen There were multiple mobile hyperechoic foci peripherally within
89 the liver parenchyma, which demonstrated associated dirty acoustic shadowing and comet
90 tail artefact and they were mobile against gravity (Fig. 2 and video 1). These same mobile
91 foci were also identified within the portal vein moving hepatopetaly (video 2). The presence
92 of hyperechoic foci within the portal vein, hepatic parenchyma and colonic wall was thought

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

104

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

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

114

115

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

121

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

127

128 **Discussion:**

129

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

134

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

139

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

151

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

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

171

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

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

192

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195

196 Footnote

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198 University

199

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201 References:

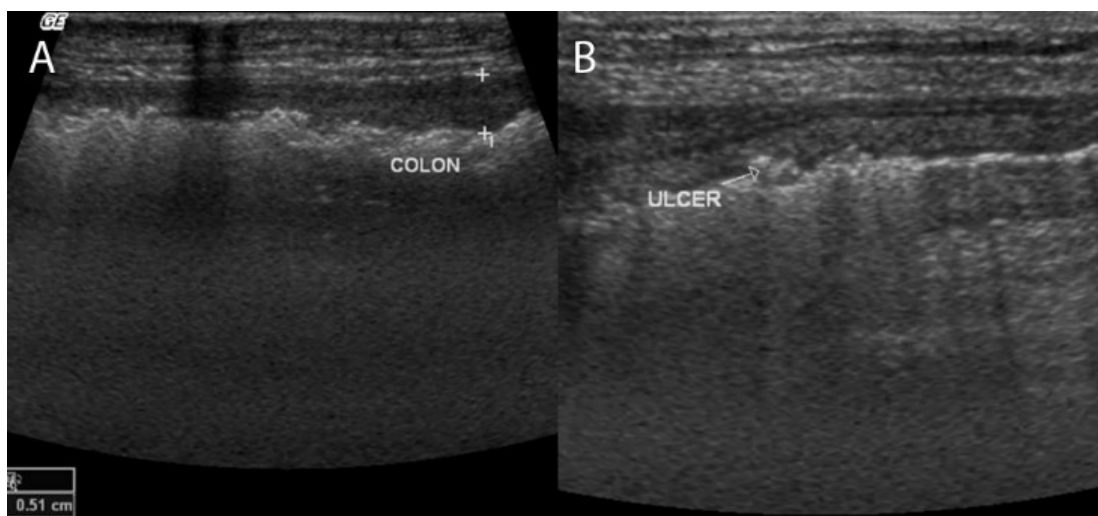
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243 Figures



244

245 Fig. 1 –Ultrasound of the descending colon at presentation, longitudinal sections from left

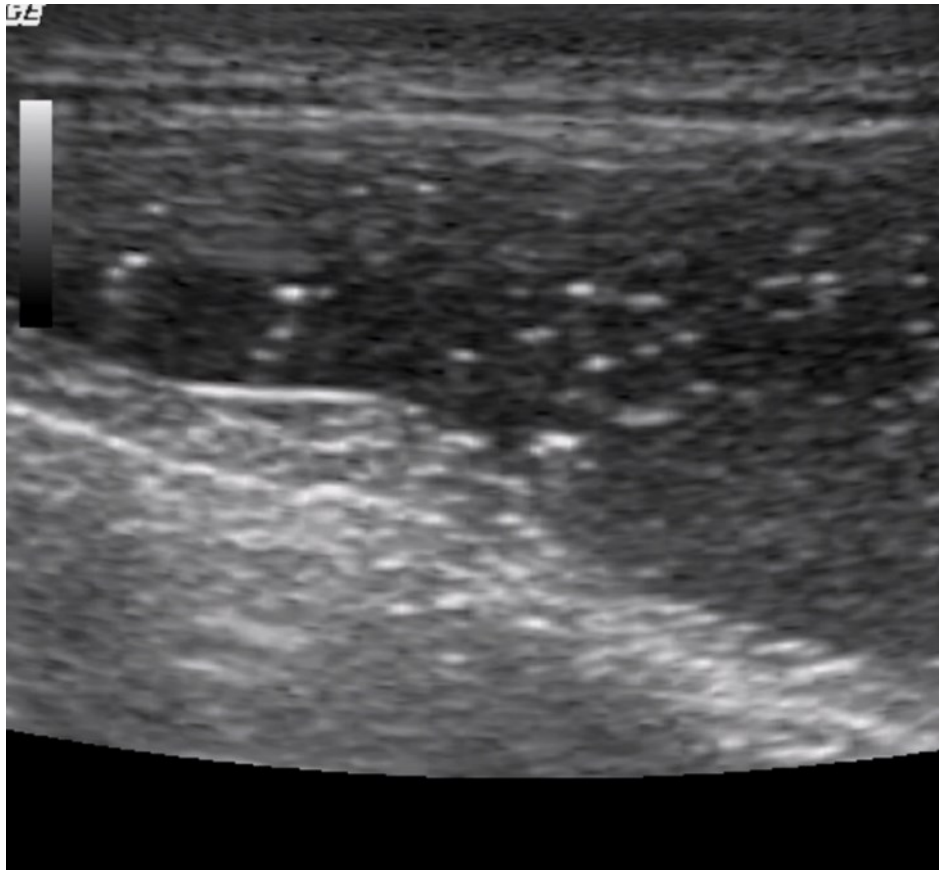
246 lateral approach, 9-12MHz linear probe. The colonic wall is markedly thickened (up to

247 5mm), particularly the descending colon, and there is marked loss of normal layering (1A).

248 There is a large volume of gas within the colonic lumen. There are focal areas of hyperechoic

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

251



252

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

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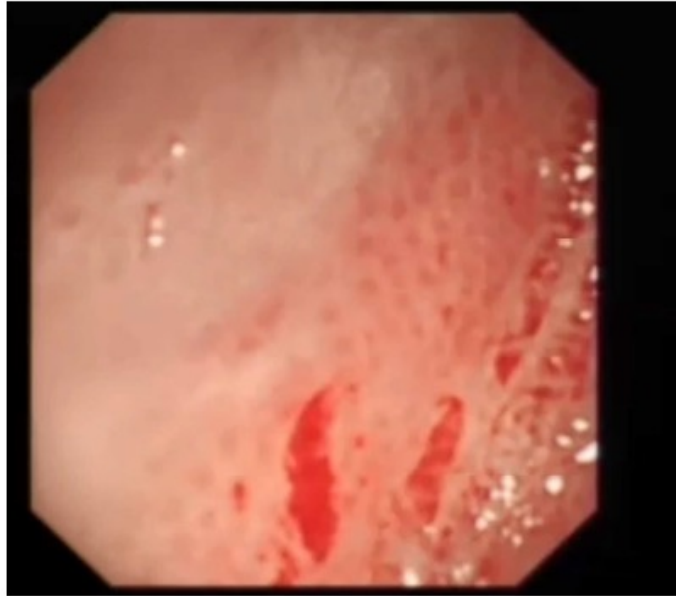


Fig. 3 Colonic mucosa viewed endoscopically demonstrating diffuse mucosal oedema, gross ulceration and focal areas of haemorrhage.
101x88mm (96 x 96 DPI)

