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TITLE: Surgical management of a cat with hepatic arterioportal fistula

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1 **Surgical management of a cat with hepatic arterioportal fistula**

2

3 **Summary**

4 A 9 month old domestic short haired cat presented with stunted growth and chronic
5 gastrointestinal signs. Tachypnea, a heart murmur, and cranial abdominal bruit were present
6 on physical examination. Echocardiography revealed volume overload in all heart chambers.
7 Computed tomographic angiography (CTA) confirmed the presence of an abnormal
8 communication between the hepatic arterial circulation and the portal vein, along with
9 multiple acquired shunts (MAS). The abnormal vascular communication was surgically
10 ligated. Echocardiography documented improvement in cardiac parameters following
11 surgery and the cat continues to have no clinical signs 39 months post operatively. This
12 report describes successful surgical management of feline hepatic arterioportal fistula (APF)
13 for the first time.

14

15 **Introduction**

16 An arteriovenous fistula (AVF) is a direct communication between an artery and vein
17 bypassing capillary beds (Hosgood 1989). Congenital fistulas can occur at any stage of
18 embryogenesis (Hosgood 1989) and join systemic arteries with either systemic veins (AVF)
19 or the portal vein (APF). Acquired fistulas may be secondary to trauma, aneurysm rupture,
20 neoplasia, infection or created surgically (Hosgood 1989, Schöniger *et al.* 2008, Phillips &
21 Aronson 2012, Adin *et al.* 2002). There are few reports of feline APF or hepatic AVF (HAVF)
22 (McConnell *et al.* 2006), where HAVF represent an intrahepatic communication between the
23 systemic arterial and portal venous system. There are two previous reports of AVF and
24 HAVF in the cat, in both cats, an attempt at surgical management was made, but neither
25 survived (Legendre *et al.* 1976, McConnell *et al.* 2006). Previously reported management
26 techniques for canine HAVF include: hepatic lobectomy with or without caval banding,
27 ligation of the nutrient artery alone and glue embolization (Chanoit *et al.* 2007).

28

29 Case History

30

31 A 9 month old male entire domestic short haired cat, weighing 3.58kg, was presented to the
32 referring veterinarian with a 5 month history of reduced appetite, intermittent diarrhoea,
33 vomiting and stunted growth. The cat remained of normal mentation except during episodes of
34 diarrhoea when depressed mentation was noted. Physical examination revealed a grade I-
35 II/VI left, systolic heart murmur, the remainder of the clinical examination at the referring
36 veterinary practice was reported as unremarkable. Investigations at the referring veterinary
37 practice included haematology, biochemistry, thoracic radiography, echocardiography and
38 abdominal ultrasound. Abnormalities identified were as follows: hypercholesterolaemia
39 7.74mmol/L [reference interval (RI) 0.00 to 5.00mmol/L], and high fasting serum bile acids
40 (SBA) 112µmol/L (RI 0 to 15µmol/L), cardiac silhouette enlargement; vertebral heart score
41 9.1 (RI <8.1; Litster & Buchanan 2000), volume overload with dilation of all cardiac
42 chambers. Abdominal ultrasound revealed an abnormal vessel within the hepatic
43 parenchyma. Doses of 10 mg furosemide twice daily (Millpledge Veterinary), 10 mg
44 spironolactone once daily (Prilactone; Ceva) and 2.5 mg benazepril once daily (Fortekor;
45 Novartis) were prescribed. Following deterioration in respiratory rate and effort the cat was
46 referred.

47

48 On presentation to Queen Mother Hospital for Animals at the Royal Veterinary College, the
49 cat was quiet but alert with a respiratory rate of 64 breaths per minute and heart rate of 132
50 beats per minute. Cardiac auscultation identified a grade II-III/IV left sternal systolic murmur.
51 Lung sounds were normal. Abdominal auscultation revealed a cranial ventral abdominal
52 bruit.

53

54 Echocardiography confirmed the previous findings with left ventricular internal diameter in
55 diastole (LVIDd) 21.5mm (RI 12.0 – 19.8), left atrium to aorta ratio (La:Ao)1.45 (RI 0.95 –
56 1.65) and maximum left atrial diameter on long-axis view (LAD) 16.2mm (RI 9.3 – 15.1)

57 (Jacobs and Knight 1985). No structural heart defects were determined; it was assumed the
58 murmur was a flow murmur secondary to high cardiac output. Abdominal ultrasound
59 identified multiple large blood vessels in and adjacent to the liver containing high-velocity
60 turbulent flow, confirmed with colour Doppler. There was subjective microhepatica. Several
61 enlarged arteries were visible caudal to the liver and multiple small blood vessels in the
62 retro-peritoneal space adjacent to the left kidney (consistent with APF and multiple acquired
63 shunts (MAS)). Medical management for suspected hepatic insufficiency was initiated with
64 doses of 62.5 mg amoxicillin-clavulanic acid orally twice daily (Kesium; Alstoe) and 1.3 mL
65 lactulose orally three times daily (Sandoz). Benazepril and spironolactone were continued
66 unchanged and the furosemide dose was reduced (5 mg orally twice daily).

67

68 The cat was returned for CT angiography (CTA) under general anaesthesia five days later.
69 Pre-contrast, time-attenuation curve (TAC) and multiple-phase post-contrast images were
70 acquired (Philips Mx8000 IDT). Time-attenuation curve was acquired during a test bolus of
71 150 mgI/kg of non-ionic iodinated contrast media (iohexol (Omnipaque, GE Healthcare)) at
72 3mL/min. The multiple post-contrast phases images were acquired after administration of
73 600mgI/kg of contrast media at 3mL/min, at 15s, 30s, 60s and 120s post contrast to
74 represent arterial, portal, venous, hepatic and late phases.

75

76 Computed tomographic angiographic examination (figure 1) revealed a single abnormal
77 vessel derived from the main hepatic artery feeding multiple small arteries that wrapped
78 around the intrahepatic central and left divisions of the portal vein and terminated in a single
79 dilated, tortuous portal vein (figure 1C, arrow head) all outside the hepatic parenchyma. The
80 CTA showed almost simultaneous contrast enhancement of the portal system (>100Hu) and
81 the aorta, prior to caudal vena cava (CVC) enhancement. There was also an extensive
82 network of MAS between all major branches of the portal system and CVC, throughout the
83 dorsal abdomen – extending cranially from the level of the first lumbar vertebrae to the
84 caudal aspect of the fourth lumbar vertebrae and laterally extending to the medial aspect of

85 both kidneys.

86

87 Following CTA, a routine celiotomy was performed. The APF was identified adjacent to the
88 left medial and the quadrate liver lobes (figure 2), no ascites was present. Multiple acquired
89 shunts were seen in the mesentery adjacent to both kidneys. Mesenteric pressure (as a
90 surrogate for portal pressure), measured via a cannula in a jejunal vein connected via a
91 three-way tap and an extension set to a transducer, was 30mmHg. Intraoperative mesenteric
92 portovenography demonstrated that the majority of blood flow entered the CVC through the
93 MAS. Based on the pathoanatomy highlighted by the CTA, an abnormal arterial branch
94 feeding the APF was identified and encircled using 2 metric polypropylene (Prolene –
95 Ethicon). Temporary occlusion of this vessel led to an acute reduction in measured
96 mesenteric pressure, to 8mmHg with no detrimental effect on heart rate or systemic blood
97 pressure, this was taken as confirmation that the correct vessel had been identified. The
98 abnormal vessel was ligated with 2 metric polypropylene. Post ligation mesenteric
99 portovenography showed complete occlusion of the APF, there continued to be significant
100 flow through the MAS. Recovery was uneventful; the abdominal bruit disappeared
101 immediately, analgesia was provided by a remifentanyl (Ultiva; GlaxoSmithKline) constant
102 rate infusion initially, titrated to methadone boluses every four hours (Comfortan; Dechra)
103 and buprenorphine every six hours (Vetergesic; Alstoe) as indicated. The cat was discharged
104 three days post-operatively with medications as prescribed preoperatively.

105

106 The cat was re-examined four weeks and six months postoperatively. At four weeks the cat
107 was behaving normally, the heart murmur and gastrointestinal signs had resolved;
108 medications other than furosemide and benazepril were stopped. By six months the cat was
109 reported to have normal activity levels and appetite and physical examination was
110 unremarkable. Haematology and serum biochemistry revealed: pre-prandial SBA 27µmol/l
111 and post-prandial SBA 21.9µmol/l, all other parameters were normal. Echocardiography
112 demonstrated subjectively mild eccentric left ventricular hypertrophy although left atrial and

113 ventricular size were reduced: LVIDd 18.6mm, LAD 16.0mm and La:Ao 1.32 at this point,
114 furosemide and benazepril therapy was discontinued. During telephone interview with the
115 owners 39 months after surgery, the cat was reported to be free of clinical signs.

116

117 **Discussion**

118 The presenting clinical signs in the cat reported here, were similar to previous reports where
119 the age of the cats and the absence of trauma, suggest a congenital aetiology (Legendre *et al.*
120 *al.* 1976, McConnell *et al.* 2006). The clinical features of APF are identical to those
121 associated with congenital portosystemic shunt (CPSS) with the exception of abdominal bruit
122 (Lipscomb *et al.* 2007, Tivers & Lipscomb 2011). Also, the clinicopathological findings
123 were consistent with hepatic insufficiency, as seen in CPSS (Tivers & Lipscomb 2011). The
124 abdominal bruit and heart murmur were the key initial findings that made us consider an
125 APF.

126 Both of the previous cats and 15/20 dogs with AVF presented with ascites secondary to
127 portal hypertension (Chanoit *et al.* 2007). Ascites was not present in the cat reported here,
128 despite evidence of portal hypertension, instead, this cat was presented because of stunted
129 growth and gastrointestinal signs. Gastrointestinal signs were the second most frequent
130 presenting sign in dogs (Chanoit *et al.* 2007) and are the second most common presenting
131 sign in cats with CPSS (Lipscomb *et al.* 2007, Tivers & Lipscomb 2011). The shunting
132 fraction and portal hypertension are possible explanations for gastrointestinal signs.
133 Feasibly, further clinical signs could have become evident in this cat over time. Portal
134 hypertension induced ascites results from a combination of hypoproteinaemia and increased
135 portal hydrostatic pressure. The absence of ascites is partially due to absence of
136 biochemical evidence of hepatic insufficiency (e.g. normal albumin) and possibly a lower
137 shunting fraction - the presence of hepatopetal flow may support this. Furthermore it is
138 possible the portal hypertension was gradual in onset allowing for the development of
139 collateral vessels. However, as ascites secondary to portal hypertension is rare in cats (Van

140 den Ingh *et al.* 1995), it is possible, in the previous two cases, the ascites was caused by
141 right sided cardiac failure.

142

143 The heart murmur was also a key physical examination finding in this cat, also in contrast to
144 dogs; only five of 20 dogs in a previous study had a heart murmur, one of which was due to
145 pulmonary stenosis. In dogs the absence of a heart murmur in spite of a volume-overloaded
146 right heart is due to the presence of hepatic sinusoids between the AVF and heart which
147 increase resistance (Chanoit *et al.* 2007). The heart murmur detected in this cat may be due
148 to the size of shunting fraction through the MAS however; this is not supported by the
149 improvement seen in cardiac parameters following surgery. Neither of the previous cats with
150 AVF or HAVF are reported to have a heart murmur, it is possible this is a feature specific to
151 this case (Legendre *et al.* 1976, McConnell *et al.* 2006).

152 Both the cats previously reported in the veterinary literature died; one post operatively
153 following an attempt at surgical treatment (Legendre *et al.* 1976) and the other was
154 euthanised intraoperatively following abdominal exploration (McConnell *et al.* 2006),
155 whereas surgical treatment in the cat reported here was relatively straightforward with an
156 uneventful recovery. The reasons for this success are unclear although it is possible that the
157 young age of the cat reported here, compared to the age of the previously reported cat
158 (Legendre *et al.* 1976), may have played a role; specifically, the secondary cardiac and
159 hepatic changes might have been less severe. In addition, the absence of reflex bradycardia
160 (Nicoladoni or Branham's sign) following ligation of the APF in this cat, might also suggest
161 that the shunting fraction was less relative to the circulating blood volume, than in previous
162 reports. This reduced flow volume could be because of a small initial diameter of the
163 congenital APF, or because there was less time for the APF diameter to increase. An
164 alternative explanation, given that the portovenogram showed the majority of blood flow
165 through the MAS, could be that significant shunting through the MAS prevented the increase
166 in afterload required to cause the reflex bradycardia. The effect of preoperative medical

167 management is also hard to quantify; anecdotally, the authors have found animals with
168 CPSS appear to have an improved postoperative recovery if managed medically for a period
169 of time before surgery, particularly if clinical signs related to hepatic insufficiency or MAS are
170 present and this may also be applicable to animals with APF. Finally, the anatomy of the
171 abnormal vasculature in the cat reported here made it readily amenable to identification and
172 ligation without the need for extensive dissection. Clearly, there will be a great deal of
173 variation in anatomy between animals with APF and the pathoanatomy in this cat was
174 straightforward compared to previous patients on which one of us (DJB) had attempted
175 surgery.

176 Once the abnormal vessel was ligated, the long-term clinical outcome was dependent on the
177 absence of pre-existing or new collateral APF channels. Abolition of the APF (without further
178 APFs developing) would allow reverse remodelling of the heart and improved cardiac
179 function and this outcome was supported by objective improvement in cardiac size on follow
180 up echocardiography. Reduced blood flow through the MAS should result in increased portal
181 flow to the liver and improved hepatic function. As portal pressure is normally higher than
182 pressure in the CVC, there remains a strong haemodynamic reason for blood to continue to
183 flow through MAS, once they are formed, despite reversal of the inciting pathology. Residual
184 flow through MAS may explain the marginal elevation in SBA after apparently successful
185 abolition of the APF. Importantly, the cat was clinically normal six months after surgery which
186 suggested that both cardiac and hepatic systems were recovering well and according to the
187 owners, the clinical improvement has been durable.

188 Diagnostic imaging was essential and CTA proved an extremely helpful form of abdominal
189 imaging in the cat reported here. Abdominal ultrasound examination identified abnormal
190 vessels adjacent to the liver and documented hepatofugal blood flow in the portal vein;
191 known to be associated with APF (Zwingenberger *et al.* 2005). The complex three-
192 dimensional nature of the abnormality is extremely difficult to appreciate in a two-
193 dimensional imaging modality, explaining the apparent discrepancies on initial inspection of

194 the imaging descriptions between ultrasound and CTA. Computed tomographic angiography
195 provided sufficient detail to allow identification of the single “feeder” vessel as a target for
196 ligation and to appreciate a single dilated termination of the abnormal vessel, allowing
197 classification of the abnormality. This is consistent with the value of CTA for abdominal
198 vascular anomalies in man (Santoro *et al.* 2009). The significant detail provided by the CTA
199 preoperatively limited the additional value of the intraoperative portovenography as the CTA
200 made identification of the APF straightforward. The portovenography did however confirm
201 preferential flow of contrast through MAS rather than a single CPSS and the cannulated
202 jejunal vessel allowed estimation of portal pressure. Echocardiographic evaluation was
203 essential for evaluating cardiac remodelling secondary to volume overload and their
204 resolution following treatment. We conclude, because of the superior anatomical information
205 gained, CTA is highly recommended in animals suspected of having such vascular
206 malformations.

207 Hepatic artery to portal venous communications are infrequently reported in the cat but they
208 are the most common AVF reported in dogs (Chanoit *et al.* 2007). Congenital HAVF are an
209 embryological abnormality, caused by a local failure of differentiation of arteries and veins
210 from a common capillary bed (Schaeffer *et al.* 2001). Dogs have been reported to have
211 different HAVF configurations (Chanoit *et al.* 2007). These include: APF - a single
212 connection between a systemic artery and the portal vein (Guzman *et al.* 2006), HAVF and
213 HAVM - direct multiple arterial and portal venous communications within the liver
214 parenchyma (Berent & Tobias 2009). It is unclear from the literature whether a true hepatic
215 artery direct to systemic vein fistula has ever been described, as most descriptions of these
216 conditions describe portal hypertension with MAS in these dogs, implying the presence of a
217 hepatic artery to portal vein anastomosis. Historically, this condition has been described as
218 HAVF or HAVM and whilst these are not inaccurate terms, it might be more accurate to
219 describe two forms of hepatic artery to portal venous fistula: namely hepatic artery to portal
220 vein fistula (HAPVF) describing a solitary vessel between the hepatic artery and the portal

221 vein and, hepatic artery to portal vein malformation (HAPVM) to describe multiple
222 anastomoses between the hepatic artery and portal vein system. In dogs, it has been
223 suggested surgical ligation is ideally suited to APF, liver lobectomy is more appropriate for
224 localized HAVM and interventional radiography (IR) guided cyanoacrylate embolism suitable
225 for those with diffuse HAVM (Chanoit *et al.* 2007).

226 The cat reported here had a direct communication between the hepatic artery and a branch
227 of the abnormally appearing portal vein, although there were a number of smaller vessels
228 between the two single vessels, importantly for both classification and treatment, the
229 abnormality originated and terminated in a single feeder vessel that was outside of the
230 hepatic parenchyma, therefore we argue, could be classified as a true APF. Regardless of
231 classification, we believe an important factor in the successful outcome of the cat described
232 here rested on the appreciation of a specific feeder vessel. This communication between
233 hepatic arterial and portal systems was only fully appreciated on CTA and we would argue in
234 such instances, occlusion of feeder vessels would represent the most appropriate initial
235 treatment option. The best treatment will depend on the pathoanatomy in each individual,
236 emphasising the importance of advanced imaging such as CTA or selective angiography.

237 In conclusion, APF are rare in cats but, a successful outcome is possible as illustrated by the
238 cat described.

239

240 No conflicts of interest have been declared.

241

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Figure legends

Figure 1

3D reconstructions of the early-phase CT angiogram. The aorta (Ao), left kidney (LK) and right kidney (RK) are identified. The tortuous anomalous tributary of the portal vein (PV) is also identified. Figure 1A is a view from the left dorsolateral aspect. The arrowheads delineate the coeliac artery and the hepatic artery. This vessel terminates in a network of small vessels in the region of the porta hepatis. Figure 1B is a view from the right lateral aspect. The arrowheads delineate a vessel which has formed from the small vessels in the region of the porta hepatis. It can be seen that this vessel terminates in a dilated portion of an aberrant portal vein tributary. Figure 1C is a ventral view. The arrowhead delineates the termination of the vessel identified in 1B. The asterisk marks the region of multiple acquired shunts, too small to be visible individually.

Figure 2

Intraoperative photograph showing the hepatic arteriovenous fistula (star), immediately caudal to the liver (Li) having been fully attenuated with an in situ Prolene ligature.