Successful Management of Spontaneous Haemoperitoneum due to Peliosis Hepatis in a Cat Using Surgical Intervention, Autologous, Xeno- and Allogenic Blood Transfusion

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Introduction

Peliosis hepatis can cause spontaneous haemoperitoneum in domestic cats, and is characterised by blood-containing cystic structures within liver parenchyma[1,2,3]. The condition has been suggested to have an infectious cause in dogs, cats and humans[4,5] and has been linked to *Bartonella henselae* in both humans[6] and dogs[5] , but, retrospective research has failed to identify *B. henselae* in cats. The aetiology of peliosis hepatis in cats remains unknown. We report the first case of successful management of spontaneous haemoperitoneum secondary to peliosis hepatis in a cat (without evidence of *B. henselae* infection) using surgical intervention, autologous, xeno- and allogenic blood transfusion.

Image 1: Alfie, recovering post op.

Case Presentation

A 5 yr castrated male DSH cat weighing 4.5 kg presented for acute collapse. The cat had pale mucous membranes, a CRT of 2 seconds, bradycardia and weak femoral pulses. The patient was open-mouth breathing and hypothermic. Figure 1 shows further examination findings and diagnostic findings. Figure 2 summarises the case progression. Figure 3 summarises the transfusions administered. The cat had received no transfusions prior to admission.

Figure 1: Examination & significant diagnostic findings

Parameter	Result
Heart rate	120 bpm
Respiratory rate	80 bpm
Temperature	35.1 degrees C
Peripheral blood pressure	Unobtainable via doppler
Haematology	Non-regenerative anaemia (Hct 19%; 24–45%) Thrombocytopaenia (manual count 6-8 platelets/HPF)
Biochemistry	Azotaemia (creatinine 220 μmol/L; 27–180, urea 11 mmol/L; 3.6–10.7) Increased serum ALT (232 U/L; 20-100) Hyperglycaemia (21.6 mmol/L; 3.9 – 8.3) Hypoproteinaemia (52 g/L; 54 – 82)
Venous blood gas, acid-base	Normal anion gap metabolic acidosis Hyperlactataemia Respiratory alkalosis (pH 7.309, $PvCO_2$ 3.81 kPa, BE(ecf) -12.2 mmol/L, lactate 6.43 mmol/L)
Thoracic ultrasound (brief)	No pericardial or pleural effusion
Abdominal ultrasound	Moderate volume peritoneal effusion; mildly large, heterogenous hyperechoic liver, small spleen and steatitis
Abdominocentesis	Haemoperitoneum (packed cell volume 26%, total solids 63g/L)
PT and APTT	Within normal limits (PT 8.2s; 13-20, APTT 142.8s; 96-122)

Figure 2: Case progression

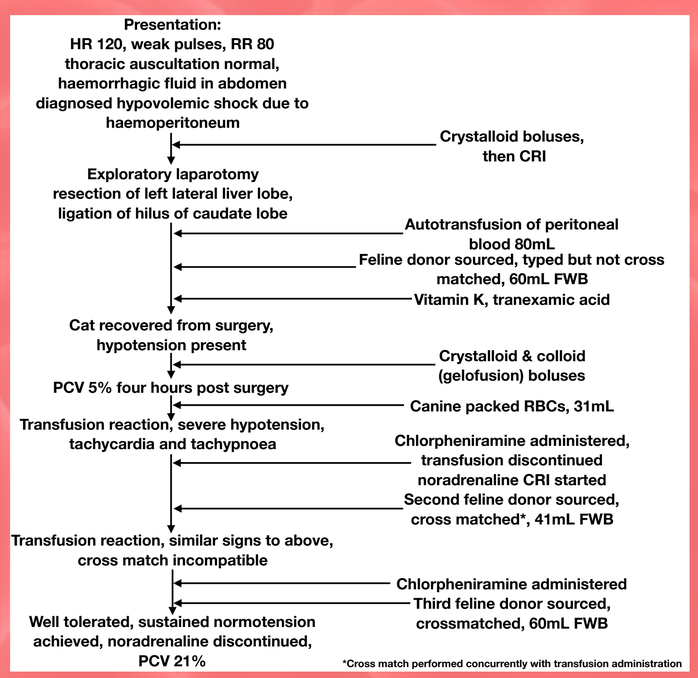

Figure 3: Transfusions administered

Transfusion	Volume Administered	Comments
1. B → B (Autotransfusion)	80 mL	Blood collected from abdomen & administered during surgery
2. B → B	60 mL	Blood typed but no cross match performed
3. B → B (Xenotransfusion)	31 mL	Discontinued administration due to transfusion reaction
4. B → B	41 mL	Discontinued administration due to transfusion reaction & cross match incompatibility
5. B → B	60 mL	Cross match performed - compatible

The patient remained in ICU for 6 days. He developed anorexia which didn't improve with mirtazapine and an oesophageal feeding tube was placed. CBC and biochemistry analysis 8 days after presentation revealed the anaemia and azotaemia had resolved. Other abnormalities in liver enzymes were observed, but no further workup was performed. He subsequently resumed eating and the feeding tube was removed 16 days after his initial presentation.

Histopathology of the affected liver lobe diagnosed peliosis hepatis with multifocal mild neutrophilic infiltration. See images 2, 3. *Bartonella* serology and FISH of the hepatic tissue were negative.

Image 2: Liver (formalin fixed), cat, peliosis hepatis; multifocal mainly subcapsular irregular well circumscribed dark red areas in the hepatic parenchyma.

Image 3: Liver, cat, peliosis hepatis; the photomicrograph shows multifocal marked dilation of sinusoids that contain large numbers of erythrocytes, few neutrophilis and fibrin. There is mild compression of surrounding hepatic parenchyma. Haematoxylin & Eosin, bar 100um.

Discusssion

Peliosis hepatis is a vasculoproliferative disorder characterised by multiple blood-filled cavities in the liver[7] which can lead to acute onset haemoperitoneum and death. Culp et al.[8] reported 65 cats with spontaneous haemoperitoneum; none had peliosis hepatis reported as the diagnosis, suggesting it is a rare cause of haemoperitoneum in the cat. Of the nine cats reported in the study by Brown et al[1] with hepatis peliosis, eight had evidence of intra-abdominal haemorrhage. A review by Buchmann et al[2] of 26 cats with hepatis peliosis found massive intraabdominal haemorrhage with hypovolaemic shock was present in three cats and there was focal rupture of peliosis hepatis in two of these. A single report[3] describes the condition in a 10 year old cat, however, the diagnosis was made post mortem.

The pathogenesis of peliosis hepatis is unknown[7]. Many underlying diseases and administration of certain drugs have been reported to cause peliosis hepatis in humans, and there are links to *B. henselae* in other species[5,6], but not cats.[6] In dogs, there are two reports of successful management of haemoperitoneum secondary to peliosis hepatis[5,9]. These reports and the case presented here highlight the need to consider peliosis hepatis as a diagnostic differential in cases presenting with haemoperitoneum.

Blood transfusions in cats are not benign procedures, and while cross-matching is not an absolute pre-requisite for dogs and cats receiving a first transfusion, it is the 'gold standard'; this case highlights its importance. Clinical feline autologous blood transfusion has only been described twice previously[10,11]. In our case, it was performed as a salvage procedure while a donor cat was sourced. After the donor cat arrived at the hospital, the need for the blood was so urgent that there was not time to perform a cross match. Xenotransfusion (canine pRBCs) was utilised due to immediate need for blood with no available cat blood nor HBOC solution. It had to be discontinued due to signs of a transfusion reaction. There have been several reports of dog blood being administered to cats[12]. It is not known if the reaction in our case was due to the dog blood, a delayed hypersensitivity reaction to the allogenic blood transfusion, or to the autologous transfusion (less likely). Reports of cats receiving canine donor blood have described that antibodies to dog blood do not develop for 4-7 days[13]. It is generally accepted that antibodies do not form until 3-5 days after allogenic transfusions. However, this cat did demonstrate transfusion reaction to subsequent donor blood administration within 24 hours of the first. It is not known whether *Mik* antibodies were responsible for the reaction. This serves to highlight the importance of cross matching for blood transfusions in all cats, if time permits.

At the time of operating the surgeon commented that there was ongoing 'ooze' of blood from the affected liver. We suspect that this was the cause of ongoing haemorrhage and need for further transfusions.

In conclusion, this is the first case report of successful treatment of haemabdomen secondary to peliosis hepatis is a cat. Surgical intervention as well as transfusions from multiple sources was required. Hepatis peliosis should be considered as a differential for cases presenting with haemabdomen.

Image 4: Alfie (front), recuperating at home

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