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Surgical resection of a giant hepatic cyst in a cat with polycystic liver disease

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ABSTRACT: Large hepatic cysts in cats cause marked abdominal enlargement while maintaining normal baseline serum and liver enzyme levels. A 3-year-old neutered male Domestic Shorthair cat presented with progressive abdominal distension and inappetence. Radiography revealed a large, fluid-density abdominal mass causing visceral displacement, and ultrasonography confirmed the presence of multiple thin-walled, anechoic intrahepatic cysts. Exploratory laparotomy achieved complete excision of the primary 15-cm cyst, alongside partial hepatectomy. Cytological evaluation confirmed clear hypocellular fluid with hepatocyte lipid vacuolation and the absence of inflammatory cells. Integrated clinical, imaging, and macroscopic findings definitively established polycystic liver disease, demonstrating that diagnostic imaging is essential when serum biochemistry fails to reflect severe structural liver pathologies.

Keywords:
abdominal distension, biliary malformation, cytology, hepatectomy, ultrasonography

■ INTRODUCTION

Feline polycystic liver disease (PCLD) is a congenital disorder resulting from ductal plate malformation and is characterized by fluid-filled intrahepatic cysts (King *et al.* 2023). Hepatic cysts can occur in polycystic kidney disease (PKD) (Stebbins 1989); however, PCLD manifests as a hepatobiliary entity. PCLD accounts for 5.6% of liver biopsy diagnoses (Hirose *et al.* 2014). Clinical evaluation is complicated by the dissociation between expansion and liver enzyme activities; lesions may cause displacement, while panels remain within reference ranges. Because multicystic intrahepatic lesions share ultrasonographic features with vascular hamartomas, cystadenomas, and abscesses, diagnosis requires imaging and cytological or tissue evaluations (Negoescu *et al.* 2024). This report documents the imaging and surgical findings of a giant hepatic cyst in an Indonesian cat with suspected polycystic liver disease despite normal enzyme levels.

■ CASE

Signalment and history. A 3-year-old neutered male Domestic Shorthair cat weighing 5.3 kg was with progressive abdominal enlargement and inappetence. No history of trauma, vomiting, diarrhea, or altered urinary behavior.

Physical examination. The patient was alert and responsive, and the vital signs were within normal limits. The mucous membranes were moist, and capillary refill was less than 2 seconds. Palpation showed distension and a fluid wave, consistent with a fluid-filled intra-abdominal structure.

Laboratory evaluation, diagnostic imaging and differential diagnoses. CBC revealed elevated hemoglobin and MCHC, indicating mild hemoconcentration, with thrombocytopenia. ALT, AST, ALP, BUN, and creatinine levels were normal (Table 1). Right lateral radiography revealed a cranioventral soft-tissue-to-fluid-density mass displacing the gastrointestinal segments caudally (Figure 1A). Ultrasonography confirmed intrahepatic lesions with hyperechoic walls and acoustic enhancement (Figure 1B). Cystocentesis was deferred; differentials included PCLD, biliary cystadenoma/cystadenocarcinoma, vascular hamartoma, and abscessation.

Surgical intervention, cytology, and postoperative care. Laparotomy revealed multilocular intrahepatic cysts with a dominant 15 cm cyst (Figure 1C). Hepatic parenchyma without cirrhosis or fibrosis. Cyst excision and resection performed; cytology showed scant cells and no microorganisms. Management included fluids, analgesia, ceftriaxone, metronidazole, hepatoprotectants, and multivitamins. Day 5 tests showed ALT (255.03 U/L) and GGT (2.69 U/L) elevations postsurgery, recovered, and discharged stable.

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Table 1. Haematology and serum biochemistry before surgery and on postoperative day 5.

Parameter	Baseline Value	Day 5 Post-Op	Reference Range
WBC (10^3/uL)	7.9	n.a.	5.5 - 19.5
RBC (10^6/uL)	6.77	n.a.	5 - 10
HGB (g/dL)	16.5	n.a.	8 - 15
MCHC (g/dL)	44.1	n.a.	30 - 35
MCH (pg)	24.2	n.a.	12.5 - 17.5
MCV (fL)	55	n.a.	39 - 55
HCT (%)	37.2	n.a.	24 - 45
PLT (10^3/uL)	227	n.a.	300 - 800
BUN (mg/dL)	27	n.a.	16 - 36
CREA (mg/dL)	0.9	n.a.	0.8 - 2.4
ALT (U/L)	63	255.03	12 – 130
AST (U/L)	28	n.a.	0 – 48
ALP (U/L)	28	n.a.	14 – 111
GGT (U/L)	n.a.	2.69	0 – 2.0

Note: White Blood Cells (WBC), Red Blood Cells (RBC), Hemoglobin (HGB), Hematocrit (HCT), Platelets (PLT), Blood Urea Nitrogen (BUN), Creatinine (CREA), Alanine Aminotransferase (ALT), Aspartate Aminotransferase (AST), Alkaline Phosphatase (ALP), Gamma-Glutamyl Transferase (GGT), n.n.= data not available.

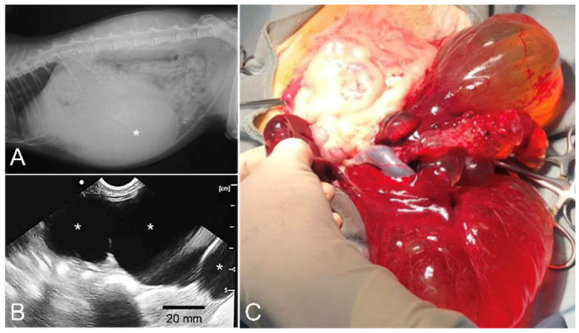


Figure 1. Radiological findings. (A) Lateral radiogram shows radiopaqueness (*) in the abdomen, and (B) ultrasonography shows a cyst in the liver with a hyperechoic rim and anechoic contents (*), and (C) multiple cysts in the liver, the cyst fluid appears clear and the liver parenchyma is normal.

■ RESULTS AND DISCUSSION

The hallmark feature of this case was the profound discrepancy between the massive 15-cm intrahepatic cyst and the completely normal baseline serum hepatic enzyme activities. In feline PCLD, clinical severity varies significantly; for instance, King *et al.* (2023) documented severe PCLD characterized by icterus, marked liver enzyme elevations, and life-threatening cyst rupture leading to hemoperitoneum. In contrast, the present cat demonstrated that substantial anatomical distortion can occur without inducing overt hepatocellular necrosis or biliary stasis. Consequently, normal liver enzyme profiles should not be used to rule out severe structural hepatobiliary disease, underscoring the limitations of serum biochemistry alone in quantifying cystic burden.

Multimodal diagnostic imaging was pivotal in achieving an accurate pre-surgical localization and characterization. While abdominal radiography confirmed a distinct soft-tissue mass effect with visceral displacement, ultrasonography definitively established the intrahepatic origin and fluid-filled nature of the cysts. Differentiating PCLD from other space-occupying hepatic lesions—such as biliary cystadenomas or hepatic vascular hamartomas, which often exhibit overlapping ultrasonographic features (Negoescu *et al.* 2024)—requires comprehensive diagnostic synthesis. Here, cyst-fluid cytology provided crucial confirmation by demonstrating low cellularity, clear fluid, and an absence of inflammatory cells or infectious agents. These cytological characteristics, combined with hepatocyte lipid vacuolation, confirmed a non-suppurative, benign process and effectively ruled out septic peritonitis, hepatic abscessation, or active inflammatory hepatopathy.

Surgical extirpation combined with partial hepatic resection successfully resolved the primary space-occupying lesion and alleviated visceral compression. The transient postoperative elevations in serum ALT (255.03 U/L) and GGT (2.69 U/L) observed on Day 5 are well-recognized clinicopathological consequences of direct surgical trauma, parenchymal transection, and subsequent hepatocellular regeneration following hepatectomy. The patient's rapid clinical recovery, restored appetite, and stable discharge confirm the efficacy of surgical intervention in managing symptomatic feline PCLD.

■ CONCLUSION

Feline polycystic liver disease can cause intra-abdominal organ displacement without altering liver enzyme activity. Preoperative imaging is crucial for characterizing cyst architecture and guiding surgery. Complete cyst excision with partial hepatectomy resolves lesions and compression, underscoring the importance of imaging despite the underestimated hepatobiliary pathology.

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■ ETHICS AND DECLARATIONS

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