

## Recurrent hydronephrosis and spontaneous renal rupture caused by lymphoplasmacytic inflammation in a cat

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**Abstract:** A seven-year-old male cat that was previously diagnosed with – and treated for – left hydronephrosis due to suspected idiopathic proteinaceous materials in the renal pelvis, presented with a short history of anorexia and vomiting. The abdominal ultrasound revealed bilateral hydronephrosis, and the intravenous pyelography showed a moderate amount of free fluid in the retroperitoneal space at 48 hours. After the nephrectomy, the gross examination of the right kidney revealed a very thin capsule with urine leakage, and the right renal pelvis showed small, black deposits. The histopathology of the right kidney revealed hydronephrosis with compression atrophy, necrosis of the renal cortex/medulla, and a moderate lymphoplasmacytic inflammation. The crystallographic analysis revealed that the black deposits were composed of 100% protein and no minerals. The cat was diagnosed with hydronephrosis and spontaneous renal rupture caused by proteinaceous pelvic materials, secondary to the idiopathic renal lymphoplasmacytic inflammation. In addition to revealing the possibility that immune-mediated renal disease can induce spontaneous renal rupture in cats, this case report demonstrates the utility of imaging for diagnosing and monitoring hydronephrosis, detecting urine leakage, and planning surgery.

**Keywords:** computed tomography; crystallography; feline; intravenous pyelography; renal; ultrasonography

Hydronephrosis refers to the dilatation of the renal pelvis and calyces due to a renal or postrenal urinary obstruction that induces the progressive atrophy of the renal parenchyma (Rawlings et al. 2003). Hydronephrosis in cats is caused by a ureteral or urethral blockage by urinary tract calculi, inflammation, neoplasia, or retroperitoneal masses resulting in extraluminal ureteral compression (D'Ippolito et al. 2006; Ragni and Fews 2008; D'Anjou et al. 2011; Zaid et al. 2011; Cohen et al. 2012; Foster and Pinkerton 2012; Selgas et al. 2014; Evans and Fowlkes 2016). Less common causes of hydronephrosis include obstructions induced by blood clots in the renal pelvis or ureters after a renal biopsy, ectopic ureters, dried

solidified blood (DSB) calculi, and retroperitoneal fibrosis after a renal transplantation (Vanden et al. 2005; D'Ippolito et al. 2006; Ragni and Fews 2008; D'Anjou et al. 2011; Zaid et al. 2011; Cohen et al. 2012; Foster and Pinkerton 2012; Lee et al. 2014; Selgas et al. 2014). Blood clots in the urinary tract are rare and may result from urinary tract calculi, infections, inflammation, neoplasia, trauma, clotting disorders, or idiopathic causes (Rawlings et al. 2003; DiBartola 2005; Vanden et al. 2005).

A spontaneous renal rupture is defined as the rupture of the renal parenchyma due to the renal pathology (Zhang et al. 2017). Possibly reflecting the dysregulation of the immune system, a lympho-

cytic-plasmacytic inflammation is characterised by the infiltration of lymphocytes and plasma cells into certain tissues, particularly in the gastrointestinal tract and nasal or oral cavities (Willard 2003; Hall and German 2005; Simpson 2005). However, hydronephrosis and a spontaneous renal rupture due to lymphoplasmacytic inflammation in the kidney have not been reported in veterinary medicine.

We document the rare case of a seven-year-old cat with recurrent hydronephrosis and a spontaneous renal rupture with a suspected blood clot formation in the renal pelvis caused by an idiopathic lymphoplasmacytic inflammation in the kidney.

### Case presentation

A six-year-old castrated male Korean Shorthair cat weighing 6.0 kg first presented with haematuria in January 2019 and was diagnosed with cystitis and bilateral renal diverticular calculi. After

the cystitis was resolved, the cat's bilateral renal calculi were monitored with an abdominal radiography, abdominal ultrasound, and laboratory tests every month thereafter at a hospital. At the nine-month follow-up, the laboratory findings showed increased creatinine [274.3  $\mu\text{mol/l}$ ; reference range (RR): 70.8–159.3  $\mu\text{mol/l}$ ]. The total protein (8.3 g/l; RR: 5.7–7.8 g/l), albumin (3.6 g/l; RR: 2.3–3.5 g/l), and calcium (1 079.6  $\mu\text{mol/l}$ ; RR: 778.7–1 053.1  $\mu\text{mol/l}$ ) concentrations were mildly elevated. On the abdominal radiography, the location, size, and number of the renal calculi remained unchanged; however, the left kidney was slightly enlarged (2.64 times the length of the second lumbar vertebral body) relative to the right kidney (2.48 times the length of the second lumbar vertebral body). The abdominal ultrasound revealed a moderate-to-severe dilation of the left renal pelvis and a mild dilation of the left proximal ureter, with amorphous and echo-



Figure 1. Abdominal ultrasound findings in a cat with recurrent hydronephrosis and spontaneous renal rupture. Longitudinal (A) and transverse (B) images of the left hydronephrotic kidney with amorphous and echogenic materials in the left renal pelvis at the nine-month follow-up. Dorsal (C) and transverse (D) images of the right kidney at five months after the first surgery showing hydronephrosis due to the amorphous, echogenic materials in the right renal pelvis along which it was asymmetrically dilated (white arrow) and a renal calculus (D).

genic material in the pelvis (Figure 1A,B). The cat was diagnosed with left hydronephrosis with radio-lucent, amorphous, and echogenic material in the renal pelvis. The differential diagnoses included blood clots, haemorrhage, inflammatory debris, DSB calculi, or mucus plugs in the left renal pelvis. The cat was hospitalised for observation and treatment, including intravenous (i.v.) fluids (22.5 ml/h, 5% dextrose and sodium chloride; CJ Healthcare, Eumseong, Republic of Korea), amoxicillin-clavulanic acid (13.75 mg/kg, q12h, Amocla; Kuhnle Pharmaceutical Co., Cheonan, Republic of Korea), metronidazole (15 mg/kg, q12h, Metrynal; Dai Han Pharmaceutical Co., Ansan, Republic of Korea), and famotidine (0.5 mg/kg, q12h, Gaster; Dong-A ST, Seoul, Republic of Korea). After four days of treatment, the hydronephrosis was resolved and the cat was discharged. However, the cat was readmitted seven days later due to a 2-day history of anorexia, vomiting, and diarrhoea. The subsequent abdominal ultrasound revealed recurrent, moderate-to-severe left hydronephrosis with amorphous and echogenic pelvic material, a small amount of free fluid in the retroperitoneal space around the left kidney, and a mild dilation with corrugation of the intestinal loops. The laboratory tests showed leukocytosis ( $25.7 \times 10^9/l$ ; RR:  $5.5\text{--}19.5 \times 10^9/l$ ), increased creatinine ( $247.8 \mu\text{mol/l}$ ; RR  $70.8\text{--}159.3 \mu\text{mol/l}$ ), and metabolic acidosis ( $\text{pH} = 7.242$ ; RR:  $7.32\text{--}7.44$ ). The cat was hospitalised and administered fluids and an antibiotic therapy. Over next five days, the white blood cell (WBC) count and creatinine levels returned to normal, and the vomiting ceased. Despite improvement in the pelvic dilation, the hydronephrosis on the abdominal ultrasound and anorexia persisted. An intravenous pyelography (IVP) showed a normal bilateral renal excretory function. An exploratory laparotomy was performed to directly identify the cause of the left hydronephrosis and revealed a slightly dilated left renal pelvis, a normal right kidney and bilateral ureters, and no obstructive material. On the pyelocentesis, approximately 0.8 ml of a mildly hazy urine was obtained using a 26-gauge needle. The abdominal ultrasound revealed alleviation of the hydronephrosis and a reduction in the amorphous material in the renal pelvis. An ultrasound-guided cystocentesis was performed using a 23-gauge needle, and a urinalysis was performed on both samples. The specific gravities of the pyelocentesis and cystocentesis samples were 1.022 and 1.021 (RR:  $> 1.035$ ), respectively. The pyelocentesis

sample showed an increased urinary protein/creatinine ratio (UPC) (0.8; RR:  $< 0.2$ ) and predominantly red blood cells (RBCs;  $> 50/\text{high power field}$ ) on the microscopic examination of the sediment. The cystocentesis sample showed a normal UPC (0.1; RR:  $< 0.2$ ) and no mucus, bacteria, or casts. Both samples were devoid of crystals or minerals. The cat recovered well and was discharged after five days.

The hydronephrosis did not recur for approximately four months. The cat presented at seven years of age with a one-day history of anorexia and vomiting.

The laboratory testing revealed a mildly increased globulin (5.3 g/l; RR:  $2.7\text{--}5.2 \text{ g/l}$ ), severe azotemia, including an increased blood urea nitrogen ( $6911.5 \mu\text{mol/l}$ ), creatinine ( $929.2 \mu\text{mol/l}$ ) and phosphorus ( $9.4 \text{ mg/l}$ ; RR:  $2.6\text{--}6.0 \text{ mg/l}$ ), and metabolic acidosis ( $\text{pH} = 7.248$ ). However, the right and left kidneys were enlarged to 2.74 and 2.65 times the length of the second lumbar vertebral body, respectively. The abdominal ultrasound showed moderate right hydronephrosis and recurrent left hydronephrosis secondary to the amorphous and echogenic pelvic material in the renal pelvis (Figure 1C). The right hydronephrosis was accompanied by a fluid-dilated, tubular structure connected to the renal pelvis toward the periphery of the right renal parenchyma (Figure 1D). Moreover, a small amount of free fluid with increased fat echogenicity was identified around both kidneys, suggesting pyelonephritis. The cat was hospitalised for a fluid and antibiotic therapy. Within six days, all the laboratory findings had returned to normal and the cat had stopped vomiting and regained its appetite. However, despite the ongoing treatment, the cat became anorexic again and developed an increased creatinine concentration ( $247.8 \mu\text{g/l}$ ) on day seven. The abdominal ultrasound revealed persistent moderate bilateral hydronephrosis. Computed tomography (CT) imaging with pre- and post-contrast was performed to investigate the hydronephrosis and the dilated tubular structure connected to the renal pelvis of the right kidney (Figure 2A). The post-contrast images revealed a very thin capsule on the right kidney, and the dilated tubular structure was identified as an asymmetric, severely dilated renal pelvis (Figure 2B). No other obstructions were noted. An IVP was then performed for 48 h showing good initial nephrographic opacification of both kidneys. However, the right kidney showed no evidence of pyelographic opacification during the procedure, and the left kidney showed a delayed pyelogram with hy-

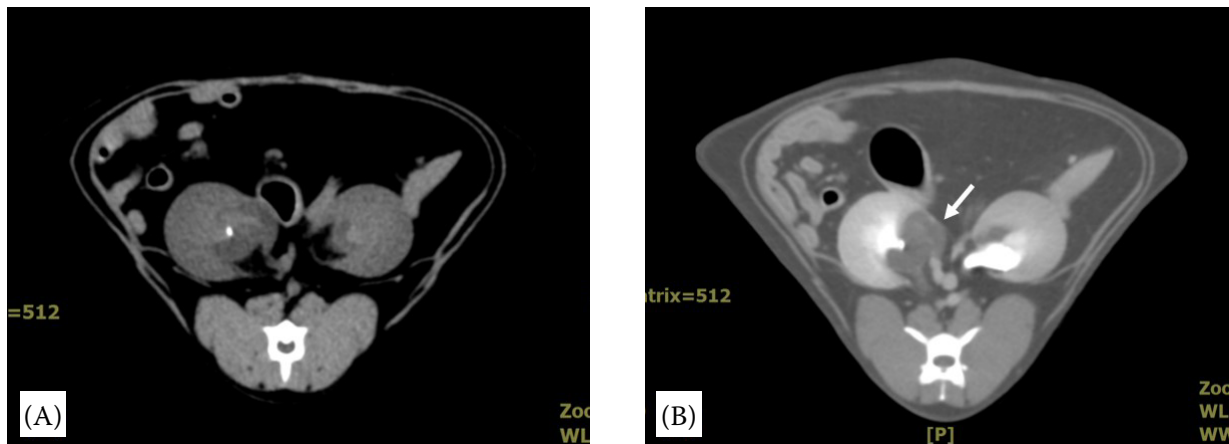


Figure 2. Pre-contrast (A) and post-contrast (B) transverse computed tomography (CT) images of the right renal pelvis in a cat with recurrent hydronephrosis and spontaneous renal rupture

The pre-contrast image shows a right renal diverticular calculus (A). The post-contrast CT image shows hydronephrosis of the right kidney with a thin capsule (white arrow), accompanied by an asymmetric and severe dilation of the pelvis (B)

hydronephrosis and hydroureter (Figure 3A,B). A moderate amount of free fluid in the retroperitoneal

space around the right kidney was newly identified (Figure 3C,D). This fluid was also seen on the abdom-

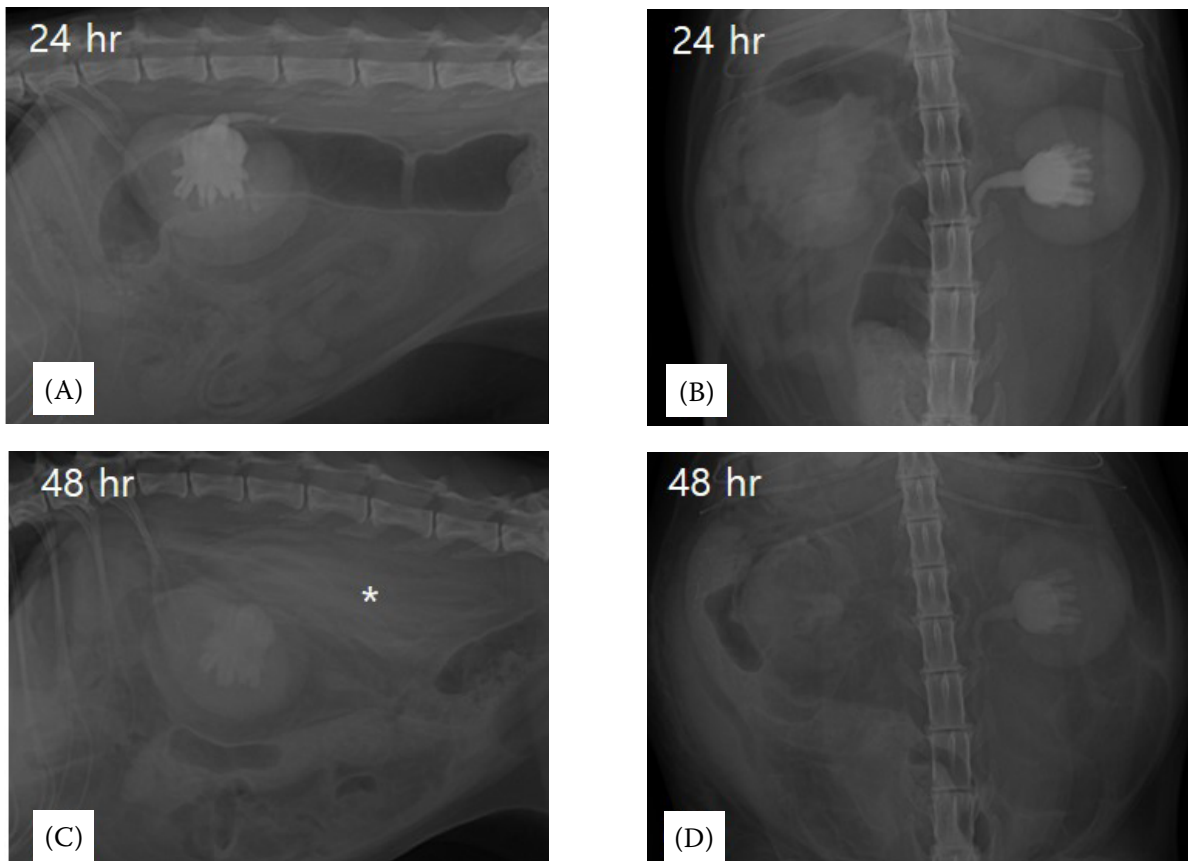


Figure 3. Intravenous pyelography at 24 h (A, B) and 48 h (C, D) in a cat with recurrent hydronephrosis and spontaneous renal rupture

Bilaterally, good initial nephrographic opacification is shown; the right kidney shows no pyelographic opacification until 48 hours. The left kidney shows a delayed pyelogram with hydronephrosis and hydroureter during the procedure. A moderate amount of free fluid in the retroperitoneal and peritoneal spaces is newly identified at 48 hours (C, asterisk)



inal ultrasound. The ultrasound-guided abdominal fluid analysis revealed that the creatinine concentration in the abdominal fluid was higher (1 681.4 µg/l) than that in the serum (16 mg/l) suggesting the presence of a uroabdomen. An exploratory laparotomy was performed to detect the urine leakage from the thin right renal capsule. During surgery, a moderate-to-severe amount of abdominal fluid was suctioned; a thin capsule with vascularisation, congestion, and an irregularly coarse parenchymal surface adjacent to the right renal pelvis was identified. Urine leakage was present (Figure 4A), and the proximal lumen of the right ureter showed a mild dilation with palpable, small, and soft deposits. A right nephrectomy was performed. The gross examination of the right kidney revealed several small, black deposits in the right renal pelvis and proximal ureteral lumen (Figure 4B). The histopathology of the right kidney revealed hydronephrosis with compression atrophy, necrosis of the renal cortex/medulla, and a moderate lymphoplasmacytic inflammation. The crystallographic analysis showed that the deposits were composed of 100% protein and no minerals. The cat was hospitalised after the surgery, and abdominal fluid was again detected on the abdominal ultrasound on the third day, with no definitive cause identified. The owner decided to discontinue treatment and requested to take the cat home, and it succumbed to the disease after 10 days.

## DISCUSSION

Our case involved a seven-year-old cat with persistent and marked bilateral hydronephrosis associated with amorphous, echogenic pelvic materials with no shadowing or mineralisation identified on the diagnostic imaging. In such cases, an exploratory laparotomy can help directly identify the pelvic material. Although there was a 5-month interval between the first and second surgeries for the left and right kidneys, respectively, the hydronephrosis on both sides was attributed to the same cause because of their similar imaging characteristics. The predominance of RBCs on the cytology at the time of the first surgery and the protein composition of the deposits in the renal pelvis affirmed our presumption that the deposits were renal blood clots with necrosis. The lack of haematuria or cloudy urine on the gross examination prompted our consideration of other aetiologies. Most mucus plugs observed in cats are composed of large quantities of matrices with varying amounts of minerals, and fewer than 10% of plugs lack crystals (Houston et al. 2003). Hence, we ruled out mucus plugs. DSB calculi are radiolucent and do not appear as discrete calculi on an abdominal ultrasound (Westropp et al. 2006; Novak and Craig 2011). However, unlike gelatinous blood clots, these calculi are very firm and stone-

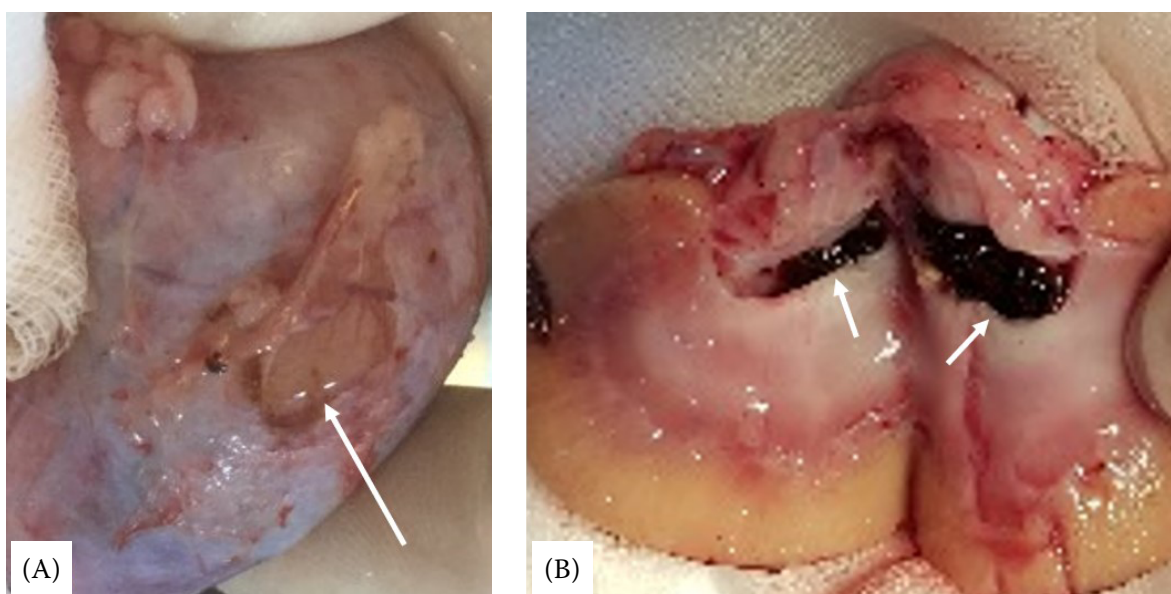


Figure 4. The right kidney and renal pelvis of a cat with recurrent hydronephrosis and spontaneous renal rupture after the right nephrectomy

This was accompanied by urine leakage (A, white arrow). A substantial amount of small, black deposits (100% protein composition) could be seen in the right renal pelvis (B, white arrows)

like (Westropp et al. 2006). Therefore, DSB calculi were less likely to be present in our case, because the pelvic material was soft when palpated during surgery. In addition, the hydronephrosis showed a spontaneous resolution with fluid and antibiotic therapy during hospitalisation, and the left hydronephrosis improved after the pyelocentesis during the first exploratory laparotomy. We presumed the material to be renal blood clots caused by an idiopathic lymphoplasmacytic inflammation because there was no history of abdominal surgery or trauma; moreover, chronic renal disease, blood clotting disorders, and other inflammations or infections in the urinary tract were excluded by the imaging and laboratory examinations, including the symmetric dimethylarginine test and urinalysis.

Our case shows that progressive hydronephrosis can result in spontaneous renal rupture and identifies the unique characteristics of hydronephrosis with radiolucent, amorphous, non-shadowing, and echogenic materials on diagnostic tests. We further demonstrated that the right renal rupture resulted from a lymphoplasmacytic inflammation, atrophy, and necrosis of the renal parenchyma due to progressive hydronephrosis. In addition, while the moderate ascites that recurred 3 days after the right nephrectomy could be attributed to the spontaneous rupture of the left kidney; this could not be confirmed because the owner refused further examinations of the cat. If the presence of a lymphoplasmacytic inflammation in the kidney had been confirmed, we could have improved the cat's condition or prolonged its life with dietary modification (e.g., hypoallergenic diet) and the administration of high-dose corticosteroids or immunosuppressive drugs (Willard 2003; Zaid et al. 2011; Zhang et al. 2017).

Although the diagnostic imaging modalities could not definitively confirm the causative material for the hydronephrosis, they helped to diagnose and monitor the hydronephrosis, detect the urine leakage, and plan the surgery. Our findings suggest that blood clots or a proteinaceous material should be considered as differential diagnoses in cats presenting with an upper urinary tract obstruction due to radiolucent, amorphous, and echogenic material with no shadowing. The continuous monitoring with an ultrasound and CT is important in such cases. The rapid surgical removal of clots or the injection of thrombolytic agents such as streptokinase into the clots may prevent progressive hy-

dronephrosis and renal rupture. If a specific cause cannot be identified from test results, an immune-mediated disease should be tested by biopsy, when possible. If a biopsy is infeasible, treatment with corticosteroids and a dietary change should be instituted, and the cat's response should be closely monitored.

### Conflict of interest

The authors declare no conflict of interest.

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