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**<Case Report>**

# Primary renal fibrosarcoma with local invasion into the mesenteric membrane of a mongrel dog

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**Abstract :** A 14-year-old, 7.4 kg, neutered male mongrel dog presented with vomiting, anorexia, and hematuria starting 3 days prior to admission. Serum biochemical profiles indicated severe azotemia. Computed tomography revealed loss of normal left kidney structure. The organ was 1.5 to 2 times larger than the right kidney with mixed attenuation. Histopathologic examination was performed after nephrectomy. The renal mass and mesenteric membrane were positive for vimentin and stained blue with Masson's trichrome. In conclusion, this was a rare occurrence of primary renal fibrosarcoma, most likely originated from the renal capsule, with local invasion into the mesenteric membrane.

**Keywords :** dog, renal fibrosarcoma, renal sarcoma, renal tumor

The incidence of primary renal neoplasm is uncommon in both dogs and humans [1-3, 7, 13]. Previous reports found that the incidence of primary renal neoplasm accounts for 0.3% to 1.5% of all canine neoplasms [1, 13]. Few cases of renal sarcomas, including spindle cell sarcomas, leiomyosarcoma, osteosarcoma, hemangioma, hemangiosarcoma, fibrosarcoma, oncocytoma, and giant cell tumors, are reported [1, 2, 8, 12, 13]. In humans, primary renal sarcomas contribute to only 1% to 3% of all renal tumors [3, 7]. Canine renal neoplasms can be divided into four types according to the cellular origin: tubular or transitional cell origin, nephroblastic origin, mesenchymal origin, and nonepithelial origin [1, 2]. The most common type of renal neoplasm in dogs is carcinomas, followed by nephroblastomas, adenomas, and rarely, sarcomas [1]. The tubular-originated neoplasm is the most common form, followed by transitional cell origins, epithelial origins, and nephroblastomas [1]. No breed predispositions have been identified [1, 2]. This case report describes a rare case of primary renal fibrosarcoma that most likely originated from the renal capsule and includes local invasion to the mesenteric membrane in a dog.

A 14-year-old, 7.4 kg, neutered male mongrel dog presented with vomiting, anorexia, and hematuria for three days prior to admission. Physical examination revealed abdominal tension, severe urine dribbling, delayed skin turgor, and dry mucous membrane. The blood work profiles showed mild regenerative anemia (35%; reference, 37~55%). The results of the serum biochemical profiles revealed severe azotemia (blood urea nitrogen, BUN, 69 mg/dL; reference, 7~27 mg/dL), elevated creatinine (CRE, 5.5 mg/dL; reference, 0.5~1.8 mg/dL), mild hyperphosphatemia (6.9 mg/dL; reference, 2.5~6.8 mg/dL). The electrolyte levels showed hypernatremia (165 mmol/L; reference, 144~160 mmol/L) and hypokalemia (3.1 mmol/L; reference, 3.5~5.8 mmol/L). The coagulation profiles were within a normal range (antithrombin III, 89%, reference, 80~200%; prothrombin time, 8 sec, reference, 6.0~11.0 sec; activated partial thromboplastin time, 28.2 sec, reference, 20.0~42.0 sec). The D-dimer level was elevated to 0.67 g/mL (reference, 0.0~0.3 g/mL). The C-reactive protein concentration was also elevated to 78 mol/L (reference, 0~10 mol/L). The blood gas analysis was normal. The heartworm kit test and microfilaria test were both negative.

A thoracic radiograph showed microcardia and increased interstitial patterns. A distended urinary bladder and irregularly shaped left kidney with a radiopaque material were observed on the abdominal radiographs.

Upon abdominal ultrasonography, the left kidney showed heterogeneous echogenicity and loss of normal structure (Fig. 1A). The right kidney showed medullary rim sign and indistinct corticomedullary junction (Fig. 1B). The left adrenal gland was normal shaped. A large blood clot was detected inside the urinary bladder (UB) (Fig. 1C). Other parts of the abdominal organ were unremarkable.

Urinalysis revealed hyposthenuria (specific gravity, 1.010;

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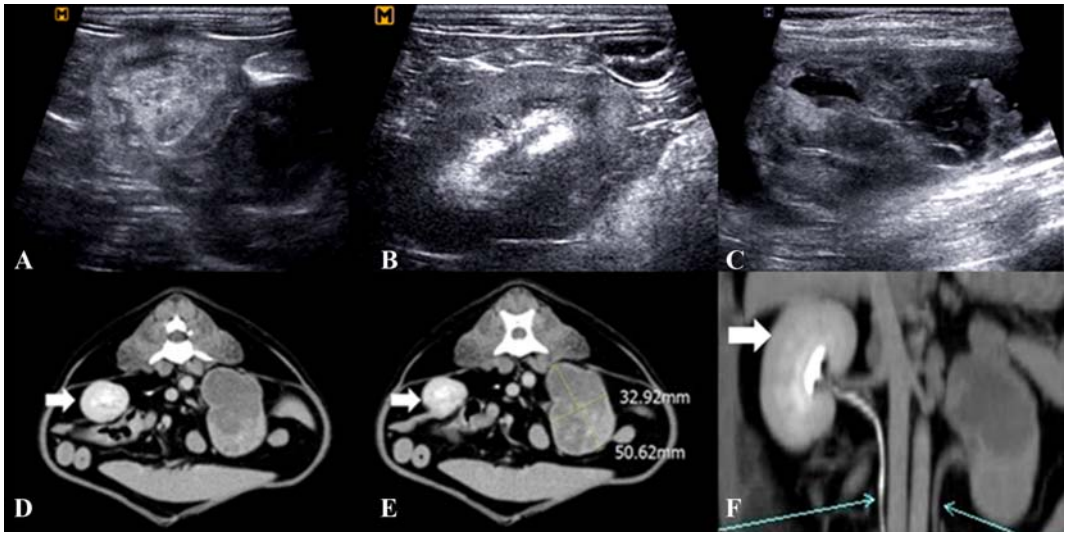


**Fig. 1.** Ultrasonographic and computed tomography (CT) findings. (A) The left kidney showed heterogeneous echogenicity and loss of normal structure, measuring 4.8 × 2.7 cm. (B) The right kidney showed an irregular margin, medullary rim sign, and indistinct corticomedullary junction. (C) A large blood clot was noticed within the urinary bladder. (D and E) Transverse images at the renal phase, enlarged left kidney without contrast enhancement, hypoattenuation at inside, multiple small mineralized lesions. The size of the left kidney measured 32.92 × 50.62 mm. The contrast enhancement was noted only within the right kidney (arrows). (F) The CT angiography at the excretory phase noted the contrast enhancement within the right kidney (thick arrow) and ureter (thin arrows).

reference, 1.015~1.045), proteinuria (170 mg/dL; reference, 0~29 mg/dL), pyuria (> 300 WBC/L; reference, 0~17 WBC/L), hematuria (> 250 RBC/L; reference, 0~7 RBC/L), and glucosuria (154 mg/dL; reference, 0~49 mg/dL). The urine protein-creatinine ratio was 0.71 (reference, 0~0.5). Cytologic examination of the urine showed non-specific amorphous casts, numerous RBCs, neutrophils, and few epithelial cells.

A tentative diagnosis was made as a primary renal tumor or abscess. To manage dehydration and azotemia, intravenous fluid was initiated with 0.9% normal saline solution with 20 mEq potassium chloride. Preventive antibiotic, cefazolin (CKD Cefazolin 30 mg/kg q8h IV; Chong Kun Dang Pharmaceutical, Korea), ranitidine (Ranitac 2 mg/kg q12 h SC; Hana Pharm, Korea), metoclopramide (MACPERAN 0.5 mg/kg q12h SC; Dong Wha Pharm, Korea), and tramadol (Tamadol 4 mg/kg q8h SC; Dongkwang Pharmaceutical, Korea) were administered. Nephrectomy was considered; anti-thrombotic agents were not selected for initial treatment. Three days after stabilization, dehydration and azotemia were improved. An additional computed tomography scan ([CT], Asteion Super 4 apparatus; Toshiba, Japan) was conducted to evaluate kidney status, including shape and function and metastasis to other organs.

The CT scan showed the mineralized irregular shape of the right kidney and a large, homogenous mass in the UB. The left kidney showed the loss of normal structure and was 1.5 to 2 times larger in size than the right kidney with mixed attenuation, hyperattenuation at the margin, and hypoattenuation in the central area (Fig. 1D, E). On the CT angiography, the contrast agent was only detected in the right kidney; loss of the left kidney function was identified (Fig. 1F). No metastatic lesion was observed in the thoracic and other abdominal organs.

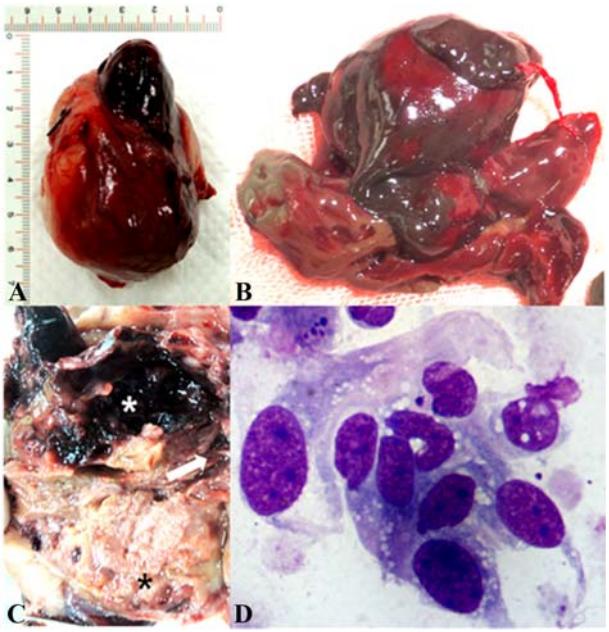


**Fig. 2.** Gross lesion and cytologic evaluations. (A) Removed left kidney size measured 7 × 5 cm. (B) A large blood clot was removed from the urinary bladder. (C) Sagittal dissection of the kidney showed multi-focal necrotic and hemorrhagic lesions and destruction of the normal structure (white asterisk, cranial pole; black asterisk, caudal pole; arrow, pelvis). (D) Impression of renal mass cytology showed mesenchymal cells with multiple prominent nucleoli, coarse chromation, and anisocytosis (diff-quick stain, ×1000).

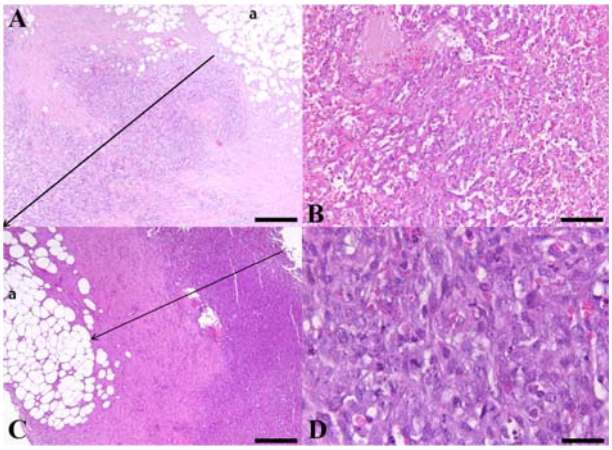


**Fig. 3.** Histopathologic examinations of left renal mass and mesenteric membrane sample. (A) A renal mass was covered by peri-renal adipose tissue, which consists of mature adipocytes (a). Invasion of proliferative connective tissues into surrounding tissue with various direction, highly packed and with high cellularity, and indistinct margins with renal capsule (arrow). (B) No visible normal renal structure was detected and changed into highly eosinophilic, acellular necrotic lesions. Proliferation of the connective tissue around the lesions with and continuity with the renal capsule was observed. (C) Mesenteric membrane sample showed similar patterns with renal mass, proliferation of eosinophilic, higly packed connective tissues, and invasion (thin arrow) into adjacent mesenteric adipose tissues (a). (D) Fibroblast of mesenteric membrane samples showed moderate to severe nuclear pleomorphism with anisokaryosis and polymorphonucleus, which is consistent with malignancy. a, aidopose tissue. H&E stain, Scale bars = 500 μm (A and C), 200 μm (B), 50 μm (D).

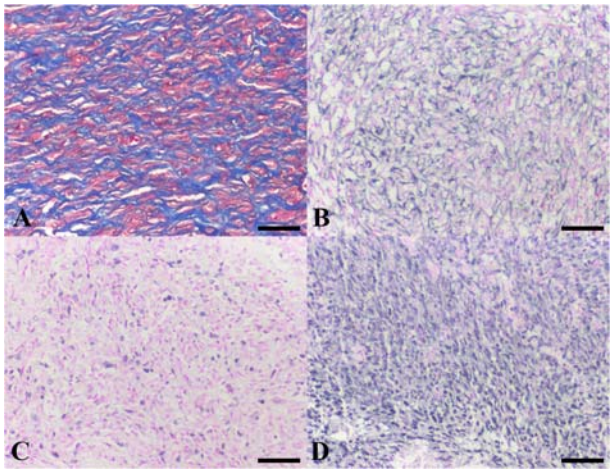


**Fig. 4.** Immunohistochemical staining. (A) Stained blue with Masson's trichrome staining. (B) positive reaction to vimentin. (C) ki-67 were showed in renal mass. (D) Positive PCNA reaction to the mesenteric membrane sample. H&E stain, Scale bars = 100 μm (A-D).

Based on the results obtained here, a primary renal-origin tumor was suspected. Unilateral nephrectomy and removal of the blood clot in the UB was performed. During the surgery, the protrusion of a hematoma-like structure of the cranial pole of the left kidney, which is attached to the adjacent mesenteric membrane, was observed. The size of the removed left kidney was 7 × 5 × 4 cm and well encapsulated (Fig. 2A). The large blood clot was removed from the UB (Fig. 2B).

Upon dissection of the kidney, necrotic hemorrhagic lesions and proliferation of abnormal tissues were identified (Fig. 2C). The cytologic evaluation of the renal mass and mesenteric membrane showed mesenchymal cells with malignancy, including multiple prominent nucleoli, anisokaryosis, coarse chromatin, and anisocytosis (Fig. 2D).

Histopathologic examinations revealed that the peri-renal region and mesentery were surrounded by eosinophilic connective tissues. The connective tissues were composed of highly packed spindle to ovoid-shaped cells with various directions of collagenofibrous fibers; the margin between the tumor and the renal capsule was indistinct (Fig. 3A). On renal parenchymas, no visible normal renal structure was detected, and eosinophilic acellular necrotic lesions replaced most of the renal cortex (Fig. 3B). The neoplastic lesion was continuous with the renal capsule that tended to extend toward the peri-renal region (externally) and the renal parenchyma (internally) (Fig. 3B). Especially in the mesentery, the tumor tissues showed severe infiltration to the mesenteric adipose tissue with mixed cellularity inflammatory cells (Fig. 3C). The fibroblast shows moderate to severe nuclear pleomorphism with anisokaryosis and polymorphonucleus, which is consistent with malignancy (Fig. 3D). Both peri-renal and mesenteric neoplastic lesions were stained blue with Masson's trichrome staining (Fig. 4A). Further immunohistochemical staining visualized with the vector SG substrate confirmed that tumor cells positively reacted with vimentin (Fig. 4B), ki-67 (Fig. 4C), and proliferating cell nuclear antigen (PCNA) (Fig. 4D) but were negative for desmin, alpha-smooth muscle actin (SMA), pancytokerain, and CD68 (data not shown). The renal mass was definitely diagnosed as a highly proliferative primary renal fibrosarcoma, which most likely originated from the renal capsule with invasion into the adjacent mesenteric membrane.

Two days after surgery, hematuria was resolved; however, the dog developed drooling, restlessness, and decreased urine output. A CBC profile revealed sudden leucopenia (5,400/μL, reference, 6,000~17,000/μL) compared with the day before showing clinical signs (45,100/μL, reference, 6,000~17,000/μL). Abdominal ultrasonography noted significant ascites at the retroperitoneal space and corrugation signs in the small bowel. Ascites analysis was clear to a yellowish color, showed neutrophilic inflammation without infection, and had different characteristics compared with urine (BUN, 8 mg/dL; creatinine, 0.3 mg/dL in ascites, BUN, 33 mg/dL; creatinine 2.1 mg/dL in urine). Acute septic or aseptic peritonitis was suspected as a complication of the tumor resection; however, the owner declined further diagnostic evaluations and

treatment and requested euthanasia. Unfortunately, no further necropsy was performed and the cause of the ascites remains unclear.

Dogs with renal neoplasm commonly present with nonspecific signs such as hematuria, anorexia, weight-loss, polyuria/polydipsia, and lethargy [2]. A neutrophilic leucocytosis and polycythemia on a CBC might be detected due to paraneoplastic syndrome [2]. Azotemia, proteinuria, and hematuria were the most commonly observed with renal neoplasm [2]. Previous reports [2, 6] found polycythemia-related renal fibrosarcoma in dogs, whereas this case showed regenerative anemia due to severe hematuria.

Most of the renal neoplasms could be identified via abdominal radiography and ultrasonography [2]. However, a definitive diagnosis could only be made after nephrectomy with histopathological examination [2].

In human medicine, primary renal fibrosarcoma is very rare and malignant; it is an aggressive tumor of the kidney with a very poor prognosis [3, 7]. In dogs, fibrosarcoma usually has an oral origin (mandible) [4, 5]. However, vaccine injection, microchip implantation, and spirocercosis-related fibrosarcoma have also been reported [11, 14, 15]. One report showed the median survival time for sarcomas to be nine months; further, it found no survival differences between sarcoma types [2]. Surgical resection of the kidney prolonged survival times compared to untreated dogs or those that only received medical therapy [2]. Nephrectomy is recommended with adjunctive chemotherapy to treat renal neoplasia [2, 3].

The renal capsule consists of large amounts of fibrous and connective tissue; fibrosarcoma is usually known as originating from the renal capsule, as described herein [3, 7]. By using immunohistochemical staining, fibrosarcoma and other types of sarcomas, especially leiomyosarcoma or sarcomatoid renal cell carcinoma, need to be differentiated [3, 7]. Fibrosarcoma is positive for vimentin and negative for cytokeratin and desmin [3, 7]. However, sarcomatoid renal cell carcinomas and leiomyosarcoma are positive for cytokeratin and desmin [3, 7]. In this case, the renal mass and the mesenteric membrane sample were positive for vimentin and were stained blue with Masson's trichrome staining, confirming fibrosarcoma [2]. Additional immunohistochemical staining, including PCNA and ki-67, revealed the active proliferation of malignant tumor cells, whereas the negative results of pancytokeratin, epithelial cell marker, CD68, macrophage/histiocyte marker, desmin, and alpha-SMA ruled out other types of tumors such as leiomyosarcoma, sarcomatoid renal cell carcinoma, histicyticsarcoma, and malignant fibrous histiocytoma.

Post-operative chylous ascites are commonly detected complications from abdominal surgery due to delayed lymphatic leakage from unhealed lymphatic channels in humans [9, 10]. Urine leakage from intraoperative injuries to the urinary tract is the most common cause of postoperative ascites [9]. In this case, the characteristics of ascites and urine were different and did not follow the criteria for chylous type. The possibility of urine leakage as a cause of ascites is low in this case. It is estimated that the ascites in this case were due to mesenteric fat necrosis or inflammation at the site of the surgically resected lesion or from unintended intraoperative infection.

In conclusion, this case first described clinical presentations, diagnostic images, gross lesions, and various histopathological examinations of a rare case of primary renal fibrosarcoma that most likely originated from the renal capsule with local invasion to the adjacent mesenteric membrane in a dog. Unfortunately, undesirable complications developed and the dog had to be euthanized; no further survival time or therapeutic outcomes were evaluated after nephrectomy in this case.

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