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Journal of Veterinary Physiology and Pathology. 2026; 5(2): 35-45.

DOI: 10.58803/jvpp.v5i2.93

http://jvpp.rovedar.com/

Case Report

Bulbourethral Gland Enlargement in a Tom Cat Associated with *Proteus mirabilis* and *Escherichia coli* Infections

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ARTICLE INFO

Article History:
Received: 11/04/2026
Revised: 15/05/2026
Accepted: 31/05/2026
Published: 28/06/2026

check for updates

Keywords:
Bulbourethral gland
Cat
Escherichia coli
Proteus mirabilis
Urinary tract infection

ABSTRACT

Introduction: Bulbourethral gland disorders are rarely reported in cats, particularly in association with bacterial urinary tract infections. The present study described a case of bulbourethral gland enlargement in a domestic cat.

Case report: A 2-year-old domestic short-haired tom cat weighing 4.7 kilograms presented to the Veterinary Teaching Hospital, University of Jos, Jos, Plateau State, Nigeria, with a major complaint of dysuria, vomiting, and inappetence. The clinical evaluation revealed lymphadenopathy, bladder distension, and haematuria. Radiographic findings demonstrated bilateral renomegaly and a distended urinary bladder. The duration of study and therapy was three days. Despite supportive therapy, including 10 mg piroxicam at 0.3 mg/kg intramuscular (IM), lactated Ringer's at 4 mL/kg, 10 mg of furosemide at 2 mg/kg IM, 2 mg of dexamethasone at 0.2 mg/kg, IM, and 5 mg of metoclopramide at 0.2 mg/kg, IM, the animal died on the third day after presentation, and post-mortem examination revealed marked enlargement of the bulbourethral glands. Bacteriological cultures of the blood, urine, and kidney samples yielded isolates of both *Proteus mirabilis* and *Escherichia coli* from each sample. Histopathological examination demonstrated glandular hyperplasia with inflammatory cell infiltration in the bulbourethral gland, lungs, and heart muscle.

Conclusion: The present study highlighted the potential role of opportunistic bacterial pathogens in the pathogenesis of bulbourethral gland enlargement and underscored the importance of early diagnosis and intervention in feline lower urinary tract disease.

1. Introduction

Feline lower urinary tract disease (FLUTD) comprises a range of disorders affecting the bladder and urethra; although bacterial infections occur, they are a relatively uncommon cause in male cats compared to other species[1]. Bacterial urinary tract infections (UTIs) are relatively uncommon in cats compared with dogs but are an important subset of FLUTD, especially in adult and older animals[2-4].

FLUTD is commonly associated with conditions such as urolithiasis and urethral plugs; however, bacterial infections, whether single- or multi-organism, are increasingly recognized as contributing factors worldwide[5]. A range of bacterial species have been isolated from feline UTIs, including *Escherichia coli* (*E. coli*), *Staphylococcus* species (notably *S. felis* and *S. pseudintermedius*), *Proteus mirabilis* (*P. mirabilis*), and *Enterococcus* species[3]. Among these bacterial species, *Staphylococcus* species and *E. coli* are frequently reported in cases of obstructive FLUTD[6,7]. In particular, *E. coli* is often the predominant pathogen and accounts for a substantial proportion of feline UTIs[8]. For instance, Hřibová et al.[9] identified *E. coli* in 26 out of 177 cases showing significant bacterial growth in feline urine samples. Another study commonly isolated Gram-negative organisms, including *P. mirabilis*, while prevalent Gram-positive bacteria included *Enterococcus faecalis*, *S. felis*, and

Cite this paper as: Buba DM, Sambo IP, Dishon CH, Tizhe EV, Gurumyen GY, Tanko PN, and Bitrus AA. Bulbourethral Gland Enlargement in a Tom Cat Associated with *Proteus mirabilis* and *Escherichia coli* Infections. Journal of Veterinary Physiology and Pathology. 2026; 5(2): 35-45. DOI: 10.58803/jvpp.v5i2.93

S. pseudintermedius[10]. These infections are often associated with underlying conditions such as urolithiasis, which can predispose male cats to recurrent urethral obstruction and serious complications, including prostatic abscessation[11,12]. Although bacterial prostatitis is less common in cats than in dogs[13], largely due to the lower incidence of UTIs in felines[14], when bacterial prostatitis does occur, it is typically characterized by neutrophilic or pyogranulomatous inflammation and may occasionally involve abscess formation[15].

The prevalence of bacterial UTIs in cats differed across studies. In cats with LUTD, the prevalence of bacterial UTI has been reported to be as high as 40.8% in some populations, while a recent systematic review and meta-analysis estimated a pooled prevalence of 18.6% specifically in cats, and 29.9% in dogs and cats combined when presenting with LUTD[16]. Studies have reported a higher prevalence of bacterial cystitis in cats with FLUTD than is traditionally recognized, with significant bacteriuria detected[17]. Across previous studies, *E. coli* remained one of the most frequently isolated organisms[8,18], and in some regional studies conducted in Turkey, this prevalence may reach 89%[19].

The bulbourethral glands (Cowper's glands) are accessory sex glands located near the pelvic urethra and are responsible for mucus secretion that lubricates the urethral passage[20]. Enlargement of these glands is uncommon in cats and is poorly documented in veterinary studies. When the bulbourethral gland is enlarged, it may be associated with infection, inflammation, or obstruction of the lower urinary tract[21]. The present study aimed to describe a rare case of bulbourethral gland enlargement associated with a mixed bacterial infection in a 2-year-old domestic short-haired cat.

2. Case report

2.1. History and clinical presentation

A 2-year-old domestic short-haired tom cat weighing 4.7 kg was presented to the Small Animal Clinic Unit of the Veterinary Teaching Hospital, University of Jos, Nigeria, on November 20, 2025, with a three-day history of dysuria, inappetence, and vomiting as the primary complaints. On presentation, the cat was slightly lethargic, with a rectal temperature of 38°C, a pulse rate of 58 beats/minute, and a respiratory rate of 22 cycles/minute. All parameters were within the normal range except the pulse rate, which was below the normal range (120-140 beats/minute). Physical examination and palpation revealed enlarged submandibular, prescapular, and popliteal lymph nodes, tenderness in the ventral pelvic region, a palpable distended urinary bladder, and straining during urination. Cystocentesis revealed haematuria. A total of 2.5 mL of urine and 2.5 mL of serum were collected to determine creatinine levels, which were 1.03 (mg/dL) and 0.731 (mg/dL), respectively. Similarly, 2.5 mL of whole blood was collected in an EDTA bottle for hematological analysis (complete blood count and differential leucocyte counts) on day one of patient presentation to the Veterinary Teaching Hospital. Hematological findings are presented in Table 1. Based on the clinical signs of dysuria, straining, and palpable distended urinary bladder, provisional diagnoses of UTI and urolithiasis were made. Therapy was started on the first day of presentation, with 10 mg of piroxicam (Jiangxi Pharma Company, Ltd, Jiangxi, China) administered at 0.3 mg/kg via intramuscular (IM) injection once[22,23] and fluid therapy with lactated Ringer's (Med-in Pharmaceutical Ltd, Ogun, Nigeria) at 4 mL/kg/hour for two consecutive days[24]. On the second day of follow-up treatment, 10 mg of furosemide (Taipei Pharm., Tech Company, Ltd, China) at 2 mg/kg, IM, once, 2 mg of dexamethasone (Hebei New Cent., Pharm., China) at 0.2 mg/kg, IM, for two consecutive days[23,25], piroxicam at 0.3 mg/kg, IM, once, and 5 mg of metoclopramide (Tianji Pharm Company, Ltd., Hubei, China) at 0.2 mg/kg, IM, was administered once[26]. An abdominopelvic radiograph was performed, revealing bilaterally enlarged kidneys and a distended urinary bladder (Figure 1A and 1B). However, the cat died shortly after the urinary bladder was emptied via cystocentesis on the third day. With the client's consent, a post-mortem examination was subsequently performed. All procedures in the present study were carried out in strict accordance with the ethical guidelines of the University of Jos, Nigeria.

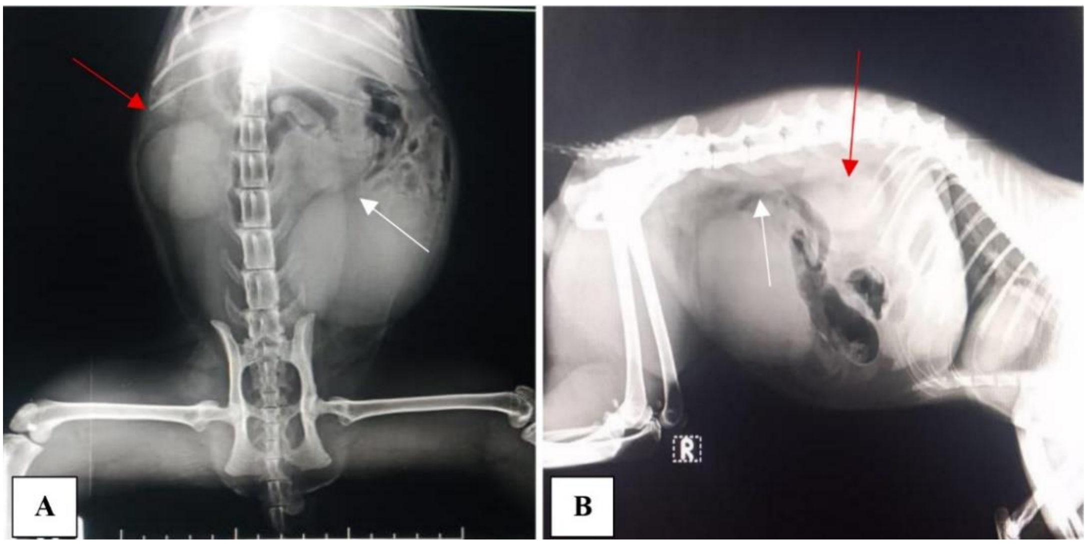

Figure 1. Abdominopelvic radiograph of a 2-year-old tom cat with a history of dysuria showing enlarged kidneys (red arrow) and a distended urinary bladder (white arrow). **A**: Dorsoventral view, **B**: Left lateral view

Table 1. Hematological findings in a 2-year-old tom cat with bulbourethral gland enlargement

Parameters	Cat values	Reference values*
Pack cell volume (%)	56	30-45
Total white blood count (cells/μL)	16,400	5,500-19,500
Segmented neutrophils (cells/μL)	14,104	2,500-12,500
Band neutrophils (cells/μL)	1640	0-300
Lymphocytes (cells/μL)	164	1500-7000
Monocytes (cells/μL)	492	0-850
Eosinophils (cells/μL)	00	0-1500
Basophils (cells/μL)	00	Rare

*Source of the reference range: Merck Veterinary Manual[27]

2.2. Sampling

2.2.1. Hematological analysis

Hematological analysis of 2.5 mL of whole blood from the tom cat was conducted at the Veterinary Teaching Hospital, University of Jos, Jos, Plateau State, Nigeria. The analysis included measurements of packed cell volume (%), total white blood cell count (× 10^3/μL), segmented neutrophils (× 10^3/μL), band neutrophils (× 10^3/μL), lymphocytes (× 10^3/μL), monocytes (× 10^3/μL), eosinophils (× 10^3/μL), and basophils (× 10^3/μL), performed using a Sysmex KX-21 automated hematology analyzer (Japan). The blood sample was collected according to the method outlined by Shabir et al.[28]. The differential leucocyte counts were performed using a cell counter and viewed under an Olympus light microscope (Shinjuku-ku, Tokyo, Japan) at 1000x magnification with oil immersion after staining the thin blood smear with Giemsa stain (Sigma-Aldrich, Germany).

2.2.2. Urine collection

A total of 2.5 mL of urine was collected aseptically via cystocentesis (sterile method) to minimize contamination and ensure accurate laboratory analysis, which was considered the gold-standard method for obtaining sterile urine for bacteriological analysis[29]. The cat was gently restrained in dorsal recumbency, and the urinary bladder was identified by palpation. The ventral abdominal area was prepared using standard aseptic procedure. A sterile needle (22-gauge) attached to the sterile syringe was inserted percutaneously into the urinary bladder, and urine was aspirated carefully. The urine sample was transferred immediately into a sterile sample bottle and processed within 30 minutes of collection.

2.3. Post-mortem examination

The cat was presented to the post-mortem room for necropsy. Following the physical examination of the carcass, tissue samples were promptly collected, and lesions were systematically observed, starting with the trachea, lungs, heart, liver, spleen, abdominal organs, kidneys, bladder, and bulbourethral gland, which were adequately documented. The post-mortem examination was performed according to the method outlined by Cooper and Valentine[30]. Kidney tissue sample was aseptically collected from the renal cortex, placed in a sterile container, and transported immediately for bacteriological culture and analysis.

2.4. Clinical biochemistry

Blood, urine, and kidney samples were collected from the animal using sterile methods and analyzed for creatinine content using the Agape creatinine test kit according to the manufacturer's (Agappe Diagnostics Ltd, Kerala, India) instructions, and absorbance was measured using a 752N – UV-Visible spectrophotometer (Agappe Diagnostics Ltd, Kerala, India) at 492 and 505 nm, respectively. Creatinine levels were determined using the kit-provided formula, with T2 as the absorbance at 505 nm and T1 at 492 nm.

2.5. Bacteriological culture and identification

Urine and kidney tissue samples were aseptically collected from the renal parenchyma, immediately placed in a sterile container, maintained at 4°C, and submitted promptly to the microbiology laboratory of the Veterinary Teaching Hospital, University of Jos, Jos, Plateau State, Nigeria, for microbiological culture. One gram (1 g) of kidney tissue was aseptically macerated with a sterile scalpel blade and inoculated into Brain Heart Infusion (BHI) broth (TM Media, India), then incubated at 37°C for 24 hours. The broth culture was subsequently subcultured onto 5% sheep blood agar (HiMedia, India) and MacConkey agar (TM Media, India) and incubated at 37°C for an additional 24 hours.

2.6. Histopathological evaluation

The tissues of the bladder, kidney, lung, heart, spleen, liver, and bulbourethral gland were fixed in 10% neutral-buffered formalin (BHD Chemicals Ltd., Poole, England). The tissues were then dehydrated through graded ethanol levels (70%, 80%, 95%, and 100%) and cleared using xylene and molten paraffin wax. The paraffin-embedded tissues were incubated in a vacuum oven at 60°C, subsequently embedded in plastic embedding rings, and sectioned at 5 μm thickness using a microtome (Leica Biosystems, Germany). The paraffin-embedded tissues were then deparaffinized with xylene, rehydrated through graded alcohol solutions (100%, 95%, 80%, and 70%), stained with hematoxylin and eosin (H&E; BDH Chemicals LTD, Poole, England), and examined under a light microscope (Olympus Corporation, Japan) in accordance with the methodology outlined by Baker et al.[31].

3. Results

3.1. Hematological findings

The cat's hematological evaluations revealed abnormal erythrogram and leucogram. The erythrogram demonstrated mild polycythemia (relative polycythemia might be due to the chief complaint of vomiting, a possible cause of dehydration), while the leucogram revealed mild neutrophilia with a severe regenerative left shift and severe lymphopenia (Table 1).

3.2. Gross pathology

At postmortem, physical examination revealed that the carcass was not emaciated with mildly pale ocular and oral mucous membranes. There was severe congestion of the skeletal muscle tissue. The heart was mildly enlarged. The kidneys (bilateral) were severely enlarged (Figure 2) and congested (Figure 3), and the urinary bladder was markedly distended (Figure 4) with red colored urine. The ureter was completely obstructed by the markedly enlarged bulbourethral glands (Figure 5). The bladder mucosa was severely hemorrhagic, with widespread ecchymotic hemorrhages (Figure 6).

Figure 4. Severely distended urinary bladder with urine in a 2-year-old short -haired tom cat infected by *Proteus mirabilis* and *Escherichia coli* as a result of urethral obstruction by the enlarged bulbourethral gland (arrow)

Figure 2. Bilateral enlargement of the kidneys in a 2-year-old short -haired tom cat, infected by *Proteus mirabilis* and *Escherichia coli*

Figure 5. The excised severely enlarged bulbourethral gland in a 2-year-old short -haired tom cat infected by *Proteus mirabilis* and *Escherichia coli*

Figure 3. Hemorrhage and severe congestion of the corticomedullary junction (arrow) in a 2-year-old tom cat, infected by *Proteus mirabilis* and *Escherichia coli*

Figure 6. Hemorrhages in the urinary bladder mucosa of a 2-year-old short -haired tom cat infected by *Proteus mirabilis* and *Escherichia coli*

3.3. Creatinine analysis

The severely low creatinine level in the urine (1.232 mg/dL) was below the reference range (80-300 mg/dL). However, the blood creatinine level (0.731 mg/dL) remained within the reference range (0.5-1.7 mg/dL; Table 2).

Table 2. Creatinine levels of the urine and blood samples in a cat with bulbourethral gland enlargement

Samples	Absorbance at 492 (T_1)	Absorbance at 505 (T_2)	Creatinine (mg/dL)	Reference value*	Inference
Urine	2.114	1.947	1.232	80-300	Severely low value
Serum	2.201	2.102	0.731	0.5-1.7	Appropriate

*Source of the reference range: Merck Veterinary Manual[29]

3.4. Bacteriological culture and identification

Isolates were characterized using colony morphology, microscopy, and biochemical properties (Table 3). On MacConkey agar, *E. coli* produced smooth, circular, moist, pink lactose-fermenting colonies, occasionally with surrounding bile precipitation (Figure 7), while *P. mirabilis* on blood agar demonstrated grayish-white colonies, smooth, raised, translucent, with entire margins measuring 0.5-1 mm in size, with a distinct swarming bulls-eye growth pattern (Figure 8). Biochemical characterization of *E. coli* and *P. mirabilis* was performed using citrate, indole, oxidase, and hydrogen sulfide (H_2S) tests (Table 3). *Proteus mirabilis* and *E. coli* were isolated from all three samples.

Figure 7. *Escherichia coli* on MacConkey agar, smooth, circular, moist, pink lactose-fermenting colonies isolated from the kidney of a 2- year-old tom cat with bulbourethral gland enlargement.

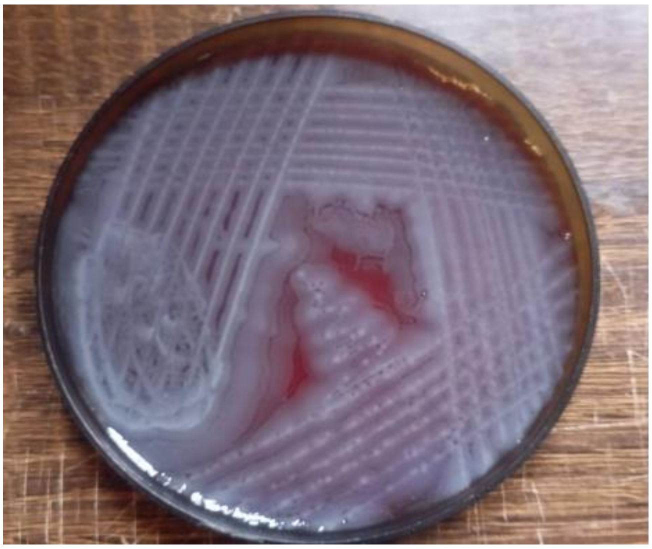

Figure 8. *Proteus* on blood agar showing grayish-white colonies, smooth, raised, and translucent, isolated from the blood of a 2-year-old short-haired tom cat with bulbourethral gland enlargement.

Table 3. Characterization of isolates using colonial morphology, microscopy, and biochemical properties

	Colonial morphology	Microscopy	Biochemicals	Sample	Inference
Isolate 1	**On blood agar:** Greyish white colonies measuring 1-2 mm in size with entire margins raised, translucent and beta-hemolytic **On MCA:** Pink colonies with slightly raised, rough margins 1-2 mm	Gram-Negative bacilli occurring singly and non-spore forming.	Citrate: Negative Indole: Positive Oxidase: Negative Catalase: Positive	Kidney Urine Blood	*Escherichia coli*
Isolate 2	**On blood agar:** Grayish-white colonies, smooth, raised, translucent, with entire margins measuring 0.5-1mm in size, with a distinct swarming bulls-eye growth pattern. **On MCA:** Colorless colonies measuring 0.5 -1 mm, transparent, raised with entire margins and moist texture.	Gram-negative bacilli occur singly and in pairs with non-spore-forming features.	Catalase: Positive Indole: Negative Citrate: Positive H_2S: Positive Motility: Positive Oxidase: Negative	Blood Kidney Urine	*Proteus mirabilis*

MCA: MacConkey agar

3.5. Histopathological findings

There was a widespread infiltration of mononuclear inflammatory cells, including epithelioid macrophages and lymphocytes, accompanied by a few polymorphonuclear cells (neutrophils; Figures 9A and 9B) in the bulbourethral gland. The glandular transitional epithelium of the bulbourethral gland was hyperplastic (Figure 9C and 9D). There was degeneration and loss of tubular epithelial cells in the kidneys, accompanied by severe interstitial hemorrhage (Figure 10). The portal areas in the liver exhibited portal venous congestion and hepatic necrosis (Figure 11A), accompanied by infiltration predominantly by neutrophils, with occasional lymphocytes (Figure 11B). Multifocal accumulations of golden-brown pigment were observed within Kupffer cells (Figure 11C). Hepatocytes exhibited vacuolar degeneration (Figure 11D). The interstitial spaces in the lungs were expanded by inflammatory cell infiltration and edema. The alveolar spaces contained oedematous fluid and hemorrhage. Pulmonary emphysema was also observed (Figure 12). The lamina propria of the urinary bladder mucosa was markedly hemorrhagic (Figure 13). There was multifocal neutrophilic infiltration within the myocardial fibers in the heart (Figure 14). The spleen revealed lymphoid follicular hyperplasia (Figure 15).

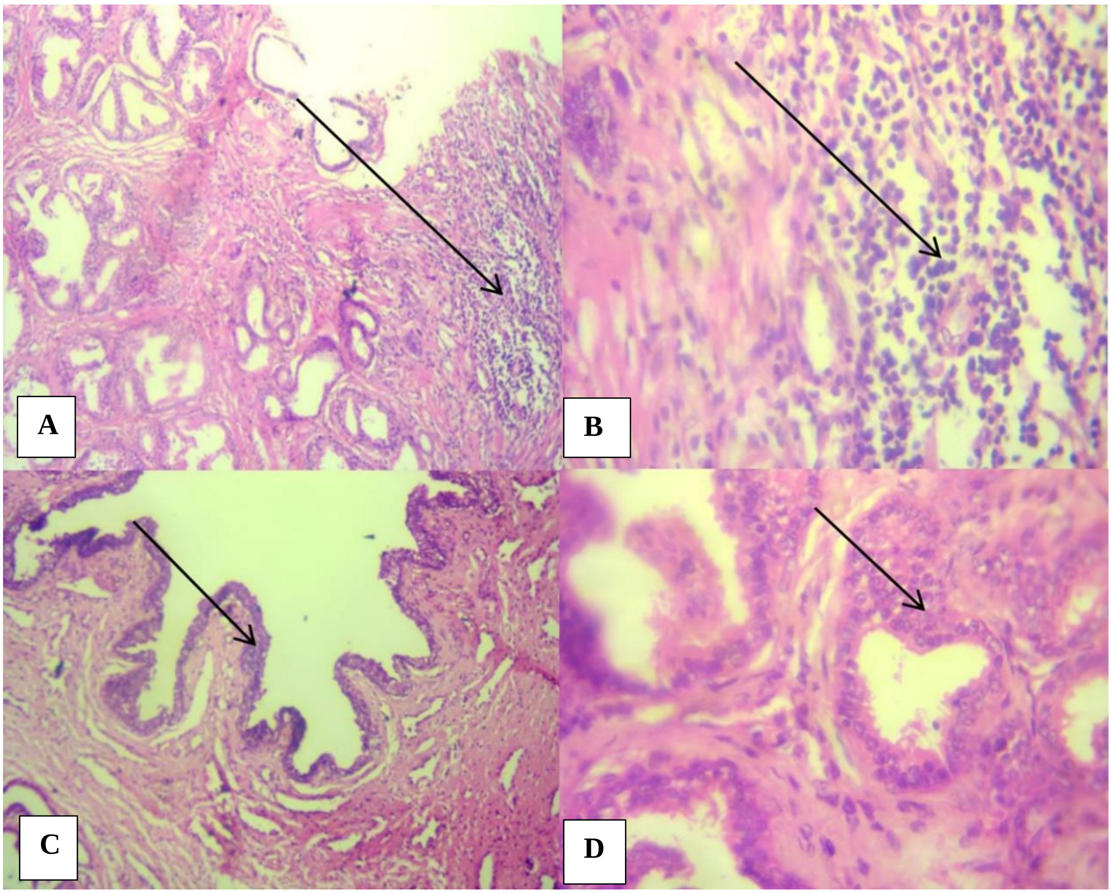

Figure 9. Bulbourethral gland in a 2-year-old short -haired Tom cat infected by *Proteus mirabilis* and *Escherichia coli*. **A**: Bulbourethral gland showing massive infiltration of inflammatory cells (arrow; 100x, H&E), **B**: Bulbourethral gland showing massive infiltration of inflammatory cells (arrow; 400x, H&E), **C**: Hyperplasia of the transitional epithelium (arrow; 100x, H&E), **D**: Hyperplasia of the transitional epithelium (arrow; 400x, H&E)

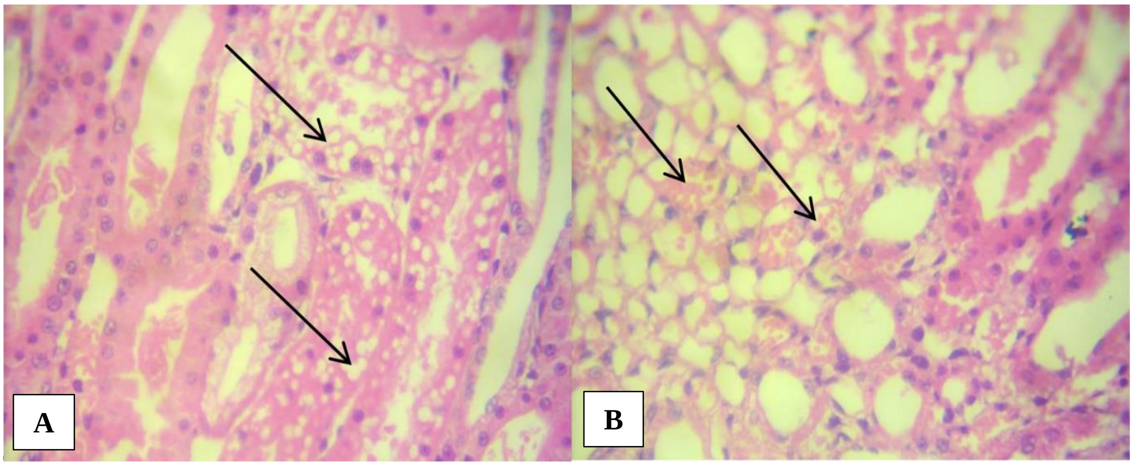

Figure 10. Kidney of a 2-year-old short -haired Tom cat infected by *Proteus mirabilis* and *Escherichia coli*. **A**: Kidney showing necrosis of the tubular epithelial cells (arrows; 400x, H&E), **B**: Kidney showing interstitial hemorrhage (arrows; 400x, H&E)

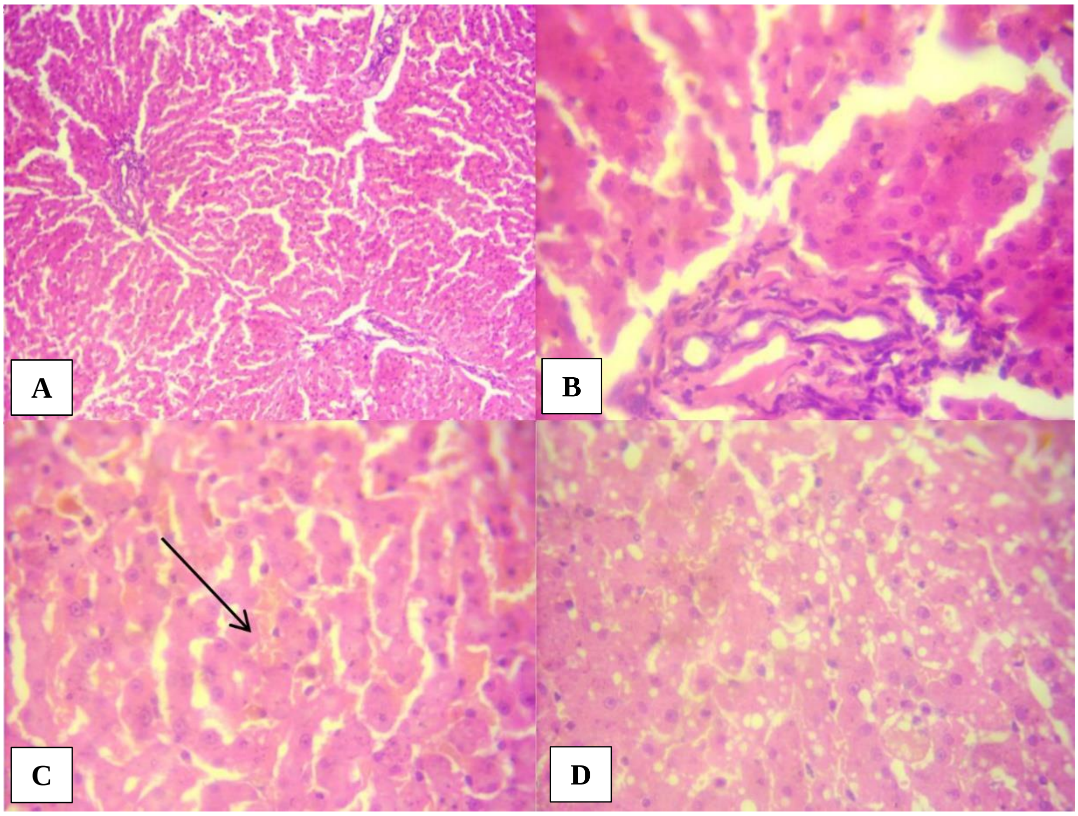

Figure 11. Liver of a 2-year-old short -haired tom cat infected by *Proteus mirabilis* and *Escherichia coli*. **A**: Liver showing hemorrhages and hepatic necrosis (100x, H&E), **B**: Liver showing coagulative necrosis of hepatocytes and infiltrating cells around the portal area (400x, H&E), **C**: Liver showing golden brown pigments (hemosiderin) in Kupffer cells, indicating hemorrhage (arrow; 400x, H&E), **D**: Liver showing fatty degeneration (400x, H&E)

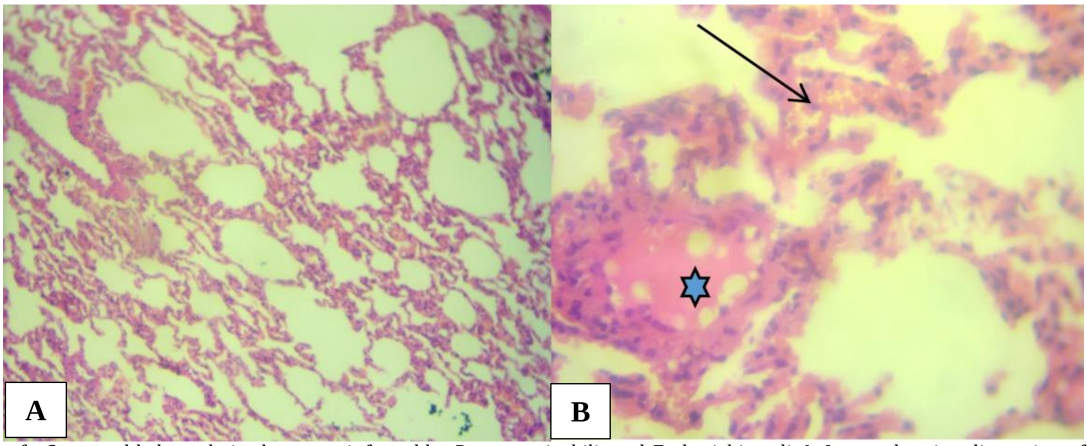

Figure 12. Lungs of a 2-year-old short -haired tom cat infected by *Proteus mirabilis* and *Escherichia coli*. **A**: Lungs showing distension of the interalveolar septae by inflammatory cells, edema, and hemorrhage (100x, H&E), **B**: Lungs showing distension of the interalveolar septae by inflammatory cells, edema (asterisk), and hemorrhage (arrow; 400x, H&E)

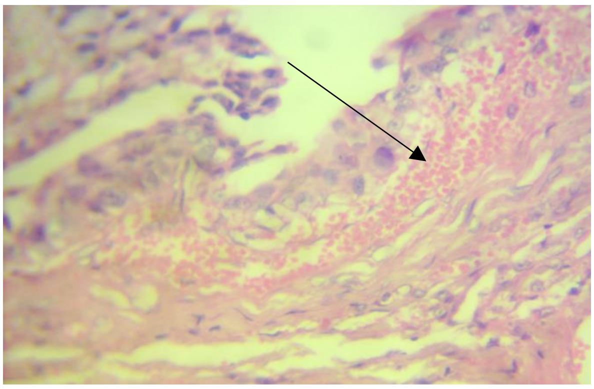

Figure 13. Urinary bladder mucosa in a 2-year-old short -haired tom cat with *Proteus mirabilis* and *Escherichia coli* infections showing severe hemorrhage of the lamina propria (400x, H&E)

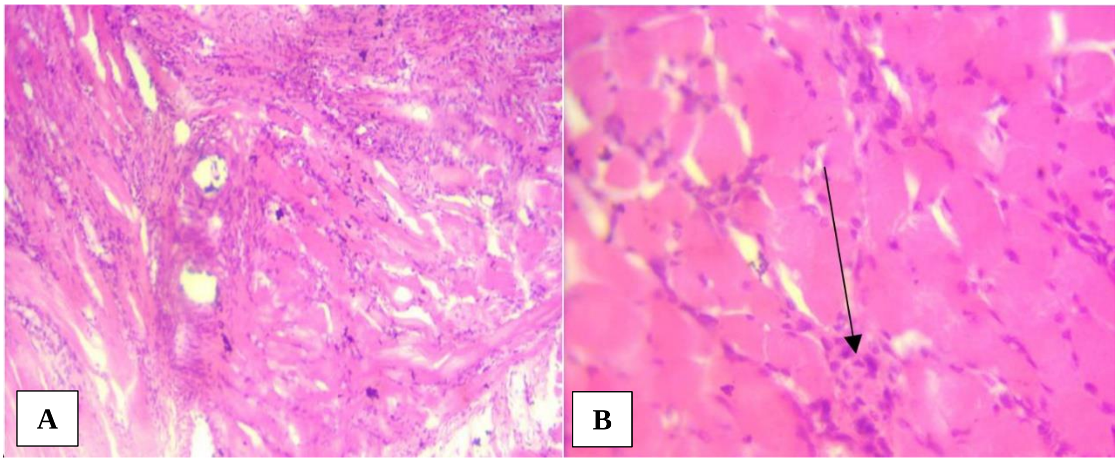

Figure 14. Cardiac muscle in a 2-year-old short-haired tom cat with *Proteus mirabilis* and *Escherichia coli* infections. **A**: Heart muscle showing severe widespread infiltration of the myofibers with inflammatory cells (100x, H&E), **B**: Heart muscle showing severe infiltration of the myofibers with inflammatory cells (arrow; 400x, H&E)

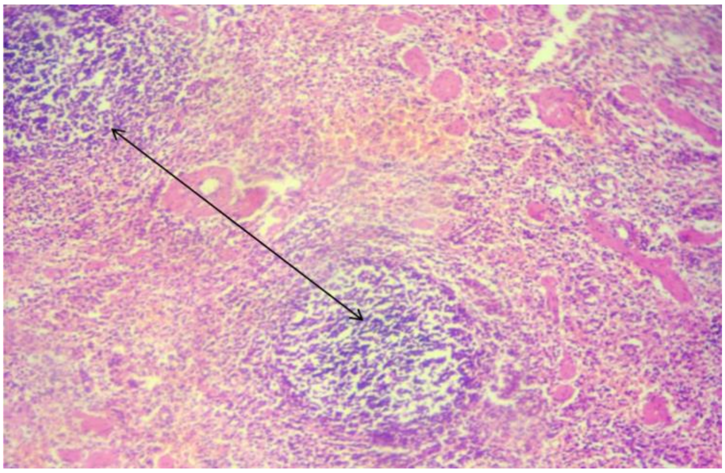

Figure 15. Spleen of a 2-year-old short-haired tom cat with *Proteus mirabilis* and *Escherichia coli* infections showing hyperplasia of the lymphoid follicles (arrow). 100x, H&E

4. Discussion

The present case highlighted a rare presentation of bulbourethral gland enlargement in a cat associated with mixed bacterial infection. *Escherichia* coli is the most commonly isolated pathogen in feline UTIs, which was similar to the findings of Puchot et al.[32], White et al.[33], Dorsch et al.[34], while *P. mirabilis* is associated with urease production and urolith formation[35,36]. The presence of *E.* coli and *P. mirabilis* in the present study suggested a potential ascending infection, possibly exacerbated by urinary stasis or obstruction, which was similar to that of Osborne et al.[37]. Enlargement of the bulbourethral glands might result from chronic inflammation, ductal obstruction, and secondary bacterial colonization, which was similar to the findings of Barsanti and Finco[38] in dogs. The observed lymphadenopathy in the present study further supported a systemic inflammatory response. Radiographic images of renal enlargement suggested a possible ascending infection involving the kidneys. Creatinine concentrations were within near-normal limits; however, acute obstruction and infection may still have resulted in rapid deterioration of the patient's condition.

In feline obstructive uropathy, sudden death after bladder decompression may result from complications such as hyperkalemia, vagal stimulation, reperfusion-related circulatory changes, and acute systemic shock. Comparable complications have been reported in cats and dogs with severe urethral obstruction[39-41]. Both organisms found in the present study were recognized uropathogens commonly implicated in complicated UTIs, particularly in males with urethral obstruction or ascending infection. These findings were consistent with the reports of Litster et al.[42] and Jacobsen et al.[43]. *Escherichia coli*, a primary uropathogen, possesses virulence factors such as adhesins, hemolysins, and endotoxins that facilitate colonization and induce mucosal injury, resulting in hematuria as observed by Litster et al.[42] and Flores-Mireles et al.[44]. The marked hemorrhagic cystitis was suggestive of bacterial-induced urothelial damage. Similarly, *P. mirabilis* likely produced urease, which hydrolyzed urea into ammonia, increasing urine pH and predisposing to mucosal irritation, inflammation, and urolith formation[43,45]. These mechanisms likely contributed to the dysuria, bladder distension, and hemorrhage recorded clinically and histologically in the present study.

Bulbourethral gland lesions, characterized by epithelial hyperplasia and mixed inflammatory cell infiltration in the present study, suggested chronic irritation and ascending infection. The presence of epithelioid macrophages and lymphocytes indicated a sustained immune response, whereas neutrophils reflected ongoing acute inflammation. These lesions were suggestive of bacterial proliferation along the urethra, as previously reported in complicated UTIs caused by urease-producing organisms such as *P. mirabilis*[45,17]. Enlargement of accessory sex glands in male animals may occur secondary to obstruction, infection, or inflammation of the lower urinary tract.

Renal lesions, including tubular epithelial necrosis and interstitial hemorrhage, suggested ascending pyelonephritis or systemic bacterial dissemination. Both *E. coli* and *P. mirabilis* can ascend from the lower urinary tract to the kidneys, leading to tubular damage and compromised renal function[42,46]. The mild elevation in serum creatinine before death might reflect early renal impairment, although post-mortem findings indicated more severe renal involvement.

The pulmonary lesions from the present study, including interstitial inflammation, edema, hemorrhage, and emphysema, were suggestive of systemic inflammatory response syndrome or septicemia. Gram-negative bacteria such as *E. coli* release endotoxins (lipopolysaccharides), which can trigger widespread vascular damage, increased permeability, and inflammatory cell infiltration in multiple organs, including the lungs[44,47]. These changes of pulmonary edema, hemorrhages, and inflammation were consistent with acute lung injury secondary to septicemia in the present study.

Hepatic findings, including portal congestion, neutrophilic infiltration, and hepatocellular vacuolar degeneration, further supported the diagnosis of systemic infection. Kupffer cell activation and accumulation of golden-brown pigments likely reflected increased phagocytic activity and the breakdown of erythrocytes due to hemolysis or systemic inflammation[47,48]. Similarly, splenic lymphoid hyperplasia indicated antigenic stimulation in response to infection, while neutrophilic infiltration of cardiac muscle suggested bacterial toxemia or early septic myocarditis.

Overall, the kind of lesions observed in the present case supported a diagnosis of severe ascending UTIs with secondary systemic dissemination. The synergistic pathogenic effects of *P. mirabilis* and *E. coli* likely exacerbated disease progression, leading to urinary obstruction, widespread inflammation, and eventual death. The present case highlights the importance of early diagnosis and aggressive management of UTIs in male cats to prevent complications such as glandular involvement, renal damage, and septicemia.

5. Conclusion

The present study reported a rare, fatal, complicated UTI in a young tom cat, caused by a mixed *E. coli* and *P. mirabilis* infection, with unusual involvement of the bulbourethral glands. The current findings emphasized the importance of early diagnosis of urinary obstruction, prompt initiation of antimicrobial therapy, and comprehensive evaluation of the accessory glands in complicated UTIs. Identification of the bacterial isolate was based on morphology and biochemical tests, and molecular identification, antimicrobial susceptibility testing, and advanced diagnostic imaging were not performed due to limitations. The present study was limited to a single case, thereby restricting wider extrapolation of the findings. The bulbourethral gland was not cultured due to severely hardened, enlarged tissue that could not be macerated. Furthermore, the histopathological lesions observed were not pathognomonic for *E. coli* and *P. mirabilis*, as similar lesions may occur in systemic infections caused by other Gram-negative organisms. Future studies should investigate the prevalence and pathological significance of bulbourethral gland involvement in FUTIs using larger case populations. Molecular characterization and antimicrobial susceptibility profiling of *E. coli* and *P. mirabilis* isolates associated with complicated UTIs in felines are also recommended to improve diagnostic accuracy and therapeutic management.

Declarations

Acknowledgments

The authors sincerely acknowledged the Veterinary Teaching Hospital at the University of Jos, Plateau State, Nigeria, for granting us access to the Veterinary Clinical Pathology and Microbiology Laboratories for sample analysis. Our appreciation is also extended to Mr. Inusa Sunday Arangu and Mr. Dechi Lokrit Peter for their assistance with the hematological and microbiological components of the research, respectively. Additionally, the authors express their gratitude to Mr. Alhassan Cyprian of the Histology Laboratory unit at Bingham University, Jos, Plateau State, Nigeria, for their support in tissue processing for histopathological examination.

Availability of data and materials

The manuscript contains all datasets generated and/or analyzed in this study, and are available upon reasonable requests from the corresponding author.

Authors' contributions

Deborah Maigawu Buba did the postmortem, drafted the main manuscript, and presented the results and findings. Iliya Paul Sambo carried out the clinical assessment and monitored the case. Charibu Hurdison Dishon was responsible for the microbiological analysis, and Emmanuel Vandi Tizhe reviewed the manuscript. George Yilzem Gurumyen assisted with the postmortem and reviewed the manuscript. Polycarp Nwunuji Tanko and Asinamai Athliamai Bitrus reviewed the manuscript. All authors have read and approved the last edition of the manuscript.

Ethical considerations

Prior to publishing this case, the authors reviewed all ethical considerations, including plagiarism, consent to publish, and data fabrication or falsification, and confirmed that no artificial intelligence was used in conducting and writing this study.

Competing interests

The authors declared no conflict of interest.

Funding

This study received no specific grant from any funding agency or organization.

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