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CASE REPORT
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Postoperative Bladder Atony in a Bitch following Surgical Resection of Urethral Leiomyosarcoma

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ABSTRACT

***Background*:** Micturition requires coordinated activity between detrusor contraction and urethral relaxation mediated by parasympathetic and somatic pathways. Disruption of these neural or myogenic connections can result in bladder atony. Itis an important postoperative complication in dogs, particularly following lower urinary tract surgery. This condition can lead to urinary retention and long-term voiding dysfunction.This case report highlights the diagnostic challenges and therapeutic considerations of postoperative bladder atony following surgical resection of a urethral leiomyosarcoma, emphasizing the importance of early recognition and integrated management for optimal recovery.

***Case*:** An 11-year-old spayed bitch mongrel underwent surgical resection and end-to-end anastomosis for a urethral mass which was histopathologically confirmed as a low-grade urethral leiomyosarcoma. On postoperative day 5, urinary retention with marked bladder distension was observed following removal of the indwelling urethral catheter. Neurological examination revealed no abnormalities, and diagnostic imaging and urinalysis excluded mechanical obstruction and urinary tract infection. The bladder was flaccid and easily expressible without voluntary voiding effort, findings consistent with bladder atony. Medical therapy including bethanechol, terazosin and metoclopramide was initiated in combinationwith manual bladder expression which was performed several times daily to prevent overdistension. Gradual improvement was noted, and spontaneous urination resumed on postoperative day 18 and persisted for 2 weeks without recurrence of retention. The patient remained clinically stable and was discharged on postoperative day 44. Pharmacologic therapy was continued and manual bladder expression performed by the owner at home. Several weeks after discharge, the patient developed lethargy and hematuria following manual expression. The dog's condition deteriorated rapidly and died. Necropsy was not performed at the owner's request.

***Discussion*:** Postoperative bladder atony is a clinically significant complication following lower urinary tract surgery. The condition is primarily attributed to transient detrusor ischemia, neural traction or excessive bladder distension during or after surgery, which may collectively impair muscle excitability and contractility. Multifactorial influences such as ischemic, neurogenic, and pharmacologic factors may contribute to bladder atony, highlighting the need for early recognition and appropriate intervention. Functional recovery depends on the severity and duration of injury, as chronic atony may lead to irreversible degeneration of smooth muscle fibers and autonomic innervation. In this case, pharmacologic and mechanical interventionssupported functional improvement of bladder contractility and partial restoration of urination. Treatment includes parasympathomimetic, α-blocking and prokinetic agents to enhance detrusor contraction and reduce outlet resistance.Manual bladder expression or intermittent catheterization is also performed to prevent overdistension. These supportive therapeutic measures can contribute to preventing long-term detrusor damage and facilitating functional recovery. This case highlights that postoperative bladder atony can develop after successful urethral surgery, emphasizing the importance of early recognition, careful postoperative monitoring, and supportive management to preserve bladder function. Delayed intervention or improper bladder handling may exacerbate detrusor injury and lead to irreversible dysfunction or rupture. Awareness of this potential complication and timely pharmacologic and mechanical support are critical to improving outcomes in similar canine cases.

Keywords: bladder atony, dog, postoperative complication, urethral neoplasm, urinary retention.

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INTRODUCTION

Lower urinary tract tumors account for less than 1% of all canine neoplasms and approximately 13% of these arise from the urethra [9]. Among urethral neoplasms, transitional cell carcinoma is the most common while leiomyosarcoma is exceptionally rare [9]. For localized and surgically accessible urethral leiomyosarcoma, surgical resection is considered the preferred treatment to achieve local tumor control and restore urinary flow [9]. Postoperative complications mainly include urinary leakage and urethral obstruction, while urinary retention may also occur [7].

Among these, bladder atony is a clinically significant cause of postoperative urinary retention that may delay recovery and lead to persistent voiding dysfunction [2]. It is defined as a loss of detrusor muscle contractility leading to incomplete bladder emptying and urine retention [8]. In human medicine, postoperative urinary retentionoccurs in up to 70% of patients, depending on the type of surgery and risk factors and some cases are attributed to bladder atony [2]. The proposed mechanism involves prolonged bladder overdistension or ischemic injury to the detrusor muscle during or after surgery, which impairs contractile function [2,12]. Contributing factors include neurogenic injury to the sacral or pelvic nerves and non-neurogenic insults such as outflow obstruction or bladder ischemia [2]. Recognition of this condition is critical because delayed management can lead to irreversible bladder dysfunction and complicate recovery.

This report describes a case of suspected bladder atony following surgical resection of a urethral leiomyosarcoma, presenting the clinical course, treatment and outcome.

CASE

An 11-year-old bitch mongrel was presented with a urethral tumor. Surgical resection of the urethral tumor with end-to-end anastomosis was performed. Histopathology[1] confirmed a low-grade urethral leiomyosarcoma (Figure 1). General anesthesia was induced with intravenous propofol[2], midazolam[3]andmaintained with isoflurane inhalation[4]. Intraoperative analgesia was achieved using a continuous rate infusion (CRI) of remifentanil[5]. Postoperatively, remifentanil CRI was continued until postoperative day (POD) 2, followed by transdermal fentanyl patch[6]. To maintain urethral patency, an indwelling urinary catheter was retained until POD 5. However, no voluntary urination was observed approximately 32 h after catheter removal.

On POD 6, an indwelling catheter was reinserted to relieve the retention and prevent overdistension. Neurological examination revealed a distended urinary bladder with intact genito-anal reflexes and normal anal tone. No additional neurological abnormalities were identified. Serum biochemical analysis (Catalyst One)[1] showed mild elevation of C-reaction protein (35 mg/L; reference range: 1-10), with no other remarkable finding. Urinalysis of manually expressed urine showed pyuria (White blood cell> 5/high-power field) with non-degenerate neutrophils and no bacteria, consistent with sterile inflammation. Also, ultrasonography revealed no evidence of urethral rupture, urolithiasis, urethral stricture, or proximal urinary tract dilation suggestive of mechanical obstruction. While a subtle urethral stricture could not be completely ruled out by ultrasonography, the smooth passage of the catheter without resistance markedly decreased the likelihood of clinically significant mechanical obstruction. Moreover, no evidence of any voluntary effort to void was observed and the bladder was soft and flaccid, readily expressible with minimal manual pressure. Considering these findings with the absence of preoperative dysuria or urinary retention, a provisional diagnosis of bladder atony secondary to urethral surgery was made.

Pharmacologic management was initiated on POD 11. Treatment included bethanechol[7] [2.5 mg/kg - orally every 12 h] and terazosin[8] [1 mg/kg - orally every 12 h]. As spontaneous urination remained absent, metoclopramide[9] [0.2 mg/kg - orally every 8 h] was subsequently added. The dose of bethanechol increased over several days to enhance bladder contractility. However, vomiting, diarrhea, and hypersalivation occurred, which resolved after dose reduction. On POD 18, 6 days after starting metoclopramide, spontaneous voiding of a small volume was noted following removal of the urinary catheter (Figure 2). The patient was subsequently managed with intermittent manual expression every 8 h to prevent over-distension. The volume and frequency of spontaneous voiding began to gradually increase around POD 18. The dog was discharged on POD 44 with ongoing prescriptions for terazosin and metoclopramide, and manual bladder expression was continued at home by the owner.

Twenty-four days after discharge, the dog was re-presented with acute-onset hematuria and abdominal distension. Point-of-care ultrasonography revealed a large volume of free anechoic fluid in the peritoneal cavity, most prominent around the urinary bladder, with swirling hyperechoic debris. Abdominocentesis revealed that the potassium concentration in the peritoneal fluid was 7.7 mmol/L, consistent with a diagnosis of uroperitoneum. These findings indicated urethral rupture as the cause of uroperitoneum. The rupture was presumed to have resulted from structural fragility at the surgical anastomosis site with forceful manual bladder expression by the owner. Despite intensive stabilization efforts, the bitch exhibited rapid clinical deterioration and died.

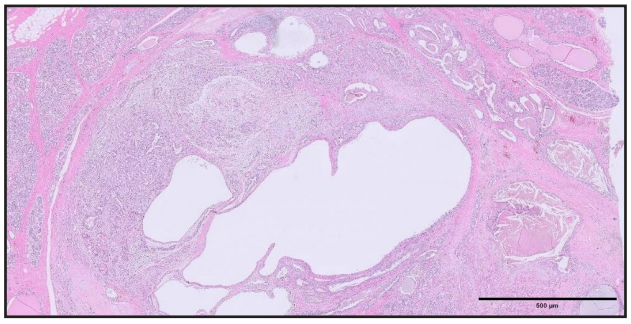

Figure 1. Histopathologic image of the urethral mass. It shows a spindle cell neoplasm effacing the urethral wall, composed of coalescing lobules arranged in poorly defined, intersecting fascicles. The tumor shows mild nuclear pleomorphism and pale eosinophilic cytoplasm, consistent with a mural sarcoma (suspected leiomyosarcoma). [HE stain ×20; Scale bar = 500 μm].

Figure 2. The patient exhibiting spontaneous urination outdoors on postoperative day (POD) 18. A small volume of urine is observed on the ground after removal of the indwelling urinary catheter, indicating partial recovery of bladder contractility.

DISCUSSION

Bladder atony is defined as a loss of normal detrusor contractility that leads to ineffective bladder emptying and marked urinary retention[6]. Bladder atony is most frequently associated with neurogenic injury, prolonged bladder overdistension due to obstruction and ischemic or reperfusion-mediated damage [6]. In the present case, bladder atony was regarded as the most probable cause of urinary retention. Several pathophysiologic mechanisms may have contributed to the development of bladder atony. Possible etiologies include urethral stricture secondary to the urethral mass or postoperative complications, primary detrusor atony, detrusor-urethral dyssynergy(DUD), upper motor neuron bladder dysfunction, and intraoperative neurogenic or ischemic injury to the detrusor muscle.

Bladder atony can develop when the detrusor muscle experiences intraoperative nerve injury or ischemic damage [6]. Repeated episodes of bladder ischemia and reperfusion can induce oxidative stress, promote the release of tissue-damaging molecules [12]. These changes can cause denervation of the detrusor and lead to temporary overactivity, which may progress to underactivity or complete atony if the damage continues [12]. In the present case, urethral mass excision with end-to-end anastomosis required traction and mobilization of the bladder and urethra. Such maneuvers can reduce blood flow to the detrusor or injure the pelvic and hypogastric nerves. Prolonged surgical time and the use of electrocautery near neurovascular bundles, along with extensive dissection of the bladder neck and urethra required to obtain sufficient length for tension-free anastomosis, may compromise neural integrity or cause localized ischemia. After surgical or ischemic injury, the bladder wall generally regains its normal tissue strength within 2-3 weeks and complete re-epithelialization occurs within 30 days [5,10]. Transient intraoperative insults during this period likely suppressed detrusor contractility in the early postoperative stage and explain the patient's delayed onset of spontaneous urination and gradual functional recovery observed during follow-up.

Perioperative pharmacologic agents may also have contributed to the development of transient postoperative bladder atony in this case. Drugs administered during anesthesia, including remifentanil, fentanyl, and midazolam, exert central nervous system depressant effects and are known to impair the micturition reflex through modulation of μ- and κ-opioid receptor path-

ways [2]. These agents can suppress afferent sensory input to the pontine micturition center and reduce detrusor tone, thereby predisposing patients to early postoperative urinary retention and detrusor hypocontractility consistent with bladder atony [2]. The effects of these drugs are generally reversible, but their use intraoperatively and in the immediate postoperative period may have intensified the transient detrusor dysfunction observed in the early postoperative period of this case.

Another possible contributor to the development of bladder atony in this case was DUD. DUD is a functional form of outflow obstruction in which the urethral sphincter fails to relax appropriately during detrusor contraction [11]. The underlying cause in veterinary patients often remains unclear and is typically suspected when clinical findings include a large residual urine volume, absence of mechanical obstruction, and a normal neurological examination [11]. This patient met all of these criteria. Based on these findings, functional outflow resistance related to DUD was regarded as a potential factor contributing to transient detrusor fatigue and the development of postoperative bladder atony [6,11].

In both human and veterinary medicine, conservative treatment including intermittent catheterization is mainly used [3]. Management of bladder atony does not aim for complete normalization of detrusor contractility, instead, it focuses on maintaining efficient bladder emptying, minimizing post-void residual urine, and preserving the patient's quality of life [3]. Other treatment options have been proposed for bladder atony in human medicine, advanced modalities such as sacral neuromodulation, stem cell therapy, or botulinum toxin injection have shown some efficacy, yet these interventions are rarely applicable in veterinary practice due to technical and financial constraints[3]. Veterinary management relies on conservative therapy using manual expression, intermittent catheterization, and pharmacologic agents such as muscarinic agonists, α-blockers, and prokinetics.

The clinical progression of detrusor activity has been rarely documented in veterinary medicine and only a few reports have been described in human medicine. Response to therapy differs according to the underlying etiology [4]. In neurogenic bladder, pharmacologic stimulation with bethanechol has been reported to facilitate voluntary voiding within several days to a few weeks [4]. Functional improvement may extend for several months with ongoing neural regeneration [4]. Ischemic bladder atony caused by detrusor hypoperfusion or reperfusion injury demonstrates a much slower regenerative process [1]. Experimental models have shown that moderate ischemia allows partial restoration of bladder function within 2-6 weeks, whereas severe ischemia leads to irreversible fibrosis and denervation [1]. In pharmacologic or functional underactivity, combination therapy with bethanechol and α-blockers improves post-void residual volume and symptom scores within approximately four weeks [3]. In DUD cases, the time to resolution or improvement of urinary signs ranged from approximately 2 to 5 weeks, with a few cases showing delayed recovery up to several months [11].

In this case, spontaneous urination resumed on POD 18 and gradually improved until discharge on POD 44. Although the dog regained the ability to void voluntarily, intermittent manual expression was still required to achieve complete bladder emptying, indicating partial functional recovery of detrusor activity. The recovery period was longer than that typically reported in neurogenic or pharmacologic underactivity. The delayed improvement likely reflected a multifactorial pathogenesis involving transient intraoperative ischemia, partial neural traction, and temporary pharmacologic suppression of detrusor contractility. Despite partial recovery of detrusor function, this case maintained acceptable quality of life and voluntary urination with supportive management including pharmacologic therapy and manual expression.

In conclusion, this case underscores the importance of recognizing bladder atony as a clinically significant and potentially manageable postoperative complication following urethral surgery in dogs, particularly those undergoing resection of urethral tumors. Multiple contributing mechanisms including ischemic injury, pharmacologic suppression, and functional outflow obstruction should be considered. Prompt recognition and a multimodal treatment approach combining pharmacologic therapy with bladder decompression are essential to achieving favorable clinical outcomes.

MANUFACTURERS

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[6]Janssen Korea. Seoul, Republic of South Korea.

[7]Boehringer Ingelheim. Ingelheim am Rhein, Germany.

[8]Pfizer. New York, NY, USA.

[9]Sanofi. Paris, France.

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***Declaration of interest*.** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

REFERENCES

1 **Andersson K.E, Nomiya M., Sawada N. & Yamaguchi O. 2014.** Pharmacological treatment of chronic pelvic ischemia. *Therapeutic Advances in Urology*. 6(3): 105-114. DOI: 10.1177/1756287214526768

2 **Baldini G., Bagry H., Aprikian A., Carli F., Warner D.S. & Warner M.A. 2009.** Postoperative urinary retention: anesthetic and perioperative considerations. *Anesthesiology*. 110(5): 1139-1157. DOI: 10.1097/ALN.0b013e31819f7aea

3 **Bayrak Ö. & Dmochowski R.R. 2019.** Underactive bladder: A review of the current treatment concepts. *Turkish Journal of Urology*. 45(6): 401. DOI: 10.5152/tud.2019.37659

4 **Diokno A.C. & Koppenhoefer R. 1976.** Bethanechol chloride in neurogenic bladder dysfunction. *Urology*. 8(5): 455-458. DOI: 10.1016/0090-4295(76)90274-0

5 **Han S., Dong K., Shen M., Wang X., Jiang W., Zhao W. & Xu D. 2019.** Observation and mechanism study of bladder wound healing after transurethral holmium laser resection of bladder tumor. *Lasers in Medical Science*. 34(6): 1217-1227. DOI: 10.1007/s10103-018-02713-0

6 **Kendall A., Byron J.K., Westropp J.L., Coates J.R., Vaden S., Adin C., Oetelaar G., Bartges J.W., Foster J.D., Adams L.G., Olby N. & Berent A. 2024.** ACVIM consensus statement on diagnosis and management of urinary incontinence in dogs. *Journal of Veterinary Internal Medicine*. 38(2): 878-903. DOI: 10.1111/jvim.16975

7 **Kim J.H., Lee J.A., Kim I.H., Jang D.W. & Kang H.G. 2014.** Prognosis and surgical treatment of the urethra embedding leiomyosarcoma in a dog. *Journal of Veterinary Clinics*. 31(4): 307-312. DOI: 10.17555/ksvc.2014.08.31.4.307

8 **Moore T.D. 1937.** Atony of Bladder: Report or Case Treated BY Presacral Neurectomy. *The Journal of Urology*. 37(6): 790-795. DOI: 10.1016/S0022-5347(17)72083-6

9 **Norris A.M., Laing E.J., Valli V.E., Withrow S.J., Macy D.W., Ogilvie G.K., Tomlinson J., McCaw D., Pidgeon G. & Jacobs R.M. 1992.** Canine bladder and urethral tumors: a retrospective study of 115 cases (1980–1985). *Journal of Veterinary Internal Medicine*. 6(3): 145-153. DOI: 10.1111/j.1939-1676.1992.tb00330.x

10 **Shin P.T. & Aoki K. 2022.** Successful conservative management in a dog with substantial urinary bladder ischemia. *The Canadian Veterinary Journal*. 63(12):1193.

11 **Stilwell C., Bazelle J., Walker D., Stanzani G. & Florey J. 2021.** Detrusor urethral dyssynergy in dogs: 35 cases (2007-2019). *Journal of Small Animal Practice*. 62(6): 468-477. DOI: 10.1111/jsap.13286.

12 **Yamaguchi O., Nomiya M. & Andersson K.E. 2014.** Functional consequences of chronic bladder ischemia. *Neurourology and Urodynamics*. 33(1): 54-58. DOI: 10.1002/nau.22517.