

Severe *Pseudomonas aeruginosa* Epididymo-orchitis Presenting as Pseudo-torsion After TURP: A Case Report

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Case Report

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Abstract

Acute epididymo-orchitis after transurethral prostate surgery is uncommon and may mimic testicular torsion, particularly when severe inflammation compromises testicular perfusion. We report a case of *Pseudomonas aeruginosa* acute epididymo-orchitis after transurethral plasmakinetic prostatectomy that was initially managed as testicular torsion and later complicated by septic shock. The patient underwent surgery for bladder outlet obstruction in the presence of preoperative bacteriuria and inflammatory urinary findings. Early postoperative recovery was uneventful, but inflammatory markers remained abnormal at discharge and subsequently increased. On postoperative day 16, he developed acute scrotal pain, fever, leukocytosis, and markedly elevated C-reactive protein. Doppler ultrasonography showed epididymal enlargement and absent intratesticular blood flow, prompting emergency exploration for presumed torsion. Intraoperative findings were consistent with acute epididymo-orchitis with secondary pseudo-torsion and pyocele; the testis was viable and was preserved. Wound drainage cultures repeatedly yielded *P. aeruginosa*. Despite antimicrobial therapy and surgical intervention, delayed recognition of the postoperative infectious trajectory was followed by septic shock requiring intensive care and vasopressor support. This case emphasizes that, in older men with acute scrotum after urological surgery, infection and evolving sepsis should be assessed alongside torsion, even when Doppler findings suggest absent testicular perfusion.

Introduction

Acute epididymo-orchitis is a recognized urological infection, but severe postoperative epididymo-orchitis mimicking testicular torsion after transurethral resection of the prostate (TURP) is uncommon. Testicular torsion is usually treated as a surgical emergency because delayed exploration may result in irreversible testicular ischemia; however, inflammatory or infectious conditions can occasionally produce similar clinical and Doppler ultrasonographic findings. This case is unique because the patient developed severe *Pseudomonas aeruginosa* epididymo-orchitis with pyocele and absent intratesticular blood flow on Doppler ultrasonography, leading to an initial concern for torsion, while operative findings ultimately supported infection-driven pseudo-torsion rather than primary spermatic cord torsion.

This case also highlights an unusual postoperative infectious cascade progressing from pre-existing urinary inflammatory findings and persistent postoperative inflammatory-marker abnormalities to acute scrotal presentation and septic shock. The trajectory suggests that marked pyuria, positive nitrites, elevated inflammatory markers, and persistent postoperative CRP elevation after TURP may represent early warning signs of occult infection, even before localized symptoms become apparent. By documenting this rare presentation, the case emphasizes the need for careful postoperative surveillance, timely interpretation of inflammatory markers, and prompt surgical exploration when acute scrotum cannot be distinguished from true torsion.

Case report

A 56-year-old man had been followed by urology since 2018 for progressive voiding difficulty and persistently elevated prostate-specific antigen (PSA). Over 7 years, PSA ranged from 22.5 to 40.4 ng/ml. Given the marked and sustained PSA elevation, he underwent two separate transrectal 12-core prostate biopsy sessions, both of which showed no evidence of malignancy. Despite surveillance and medical treatment, his lower urinary tract symptoms progressed, and surgical treatment for bladder outlet obstruction was planned.

On September 22, 2025, he was admitted for operative management. PSA was 40.4 ng/ml. Urinalysis showed marked pyuria, with urine leukocytes of 298.8/μl, positive leukocyte esterase (++) , and positive nitrites. Interleukin-6 was elevated at 23.2 pg/ml. These findings indicated active urinary inflammation with probable Gram-negative bacteriuria. In the absence of biopsy-proven malignancy, the combination of sustained PSA elevation, pyuria, positive nitrites, and elevated interleukin-6 suggested that chronic infection or prostatitis contributed to the PSA elevation.

On September 25, transurethral plasmakinetic prostatectomy was performed for bladder outlet obstruction. Approximately 35 g of prostatic tissue was resected over 75 min. Perioperative antimicrobial prophylaxis consisted of intravenous cefuroxime 1.5 g. The immediate postoperative course appeared clinically stable. The white blood cell count was $10.4 \times 10^9/l$, and C-reactive protein (CRP) transiently increased from 14.00 to 41.73 mg/l before decreasing during the first postoperative week. The patient was discharged on October 2, postoperative day (POD) 7, with a CRP level of 14.5 mg/l, indicating incomplete inflammatory resolution.

Serial perioperative laboratory testing revealed a biphasic postoperative inflammatory course. After apparent early recovery, occult systemic inflammatory deterioration developed on POD14–15, with the white blood cell count increasing from 15.2 to $22.5 \times 10^9/l$ and CRP from 195.0 to 275.0 mg/l before overt scrotal symptoms and septic shock became evident (Table 1). The temporal relationship between biomarker trajectories, operative events, and intensive care management is summarized in Fig. 1.

Table 1
Perioperative Dynamics of Hematologic and Inflammatory Markers. *

Date(2025)	POD†	WBC(×10 ⁹ /liter)	Neutrophils(%)	Lymphocytes(%)	ALC(×10 ⁹ /liter)	Hemoglobin(g/liter)	CRP(mg/liter)	Procalcitonin
Preoperative baseline	–3	5.0	68.9	23.1	1.2	159	0.82	< 0.5
Sept. 22								
Early post-TURP course								
Sept. 25	0	10.4	79.5	13.2	1.4	123	14.00	–
Sept. 27	2	8.1	76.2	16.0	1.3	120	41.73	–
Sept. 29	4	7.0	73.6	17.3	1.2	113	16.00	–
Occult systemic deterioration								
Oct. 9	14	15.2‡	86.6‡	8.6	1.2	108	195.00‡	–
Oct. 10	15	22.5‡	92.1‡	4.0	1.0	119	275.00‡	–
Septic peak and scrotal exploration								
Oct. 11	16	34.6‡	95.0‡	1.2	0.4§	131	–	–
Oct. 12	17	25.5‡	92.8‡	2.9	0.7§	89§	297.58‡	> 10‡
Oct. 13	18	26.2‡	93.3‡	4.1	1.1	114	95.00	2–10‡
Recovery and convalescence								
Oct. 16	21	20.0‡	79.2	14.3	2.9	120	105.00	0.5–2
Oct. 19	24	14.2	82.4	9.7	1.4	122	57.00	< 0.5
Oct. 22	27	9.1	76.7	12.4	1.1	120	36.00	< 0.5
Oct. 26	31	7.5	67.3	22.4	1.8	115	11.00	–
Nov. 3	39	5.8	61.0	25.7	1.5	118	7.00	–
Nov. 4	40	5.0	61.4	24.8	–	103	–	–
* Values represent single laboratory measurements obtained during the patient's perioperative course. Reference ranges (laboratory-specific): white-cell count 4–10×10 ⁹ /liter; neutrophils 40–75%; lymphocytes 20–50%;								
absolute lymphocyte count (ALC) 1.1–3.2×10 ⁹ /liter; hemoglobin 130–175 g/liter; C-reactive protein (CRP) < 10 mg/liter; procalcitonin < 0.5 ng/ml. ED denotes emergency department, ICU intensive care unit, POD postoperative day, and TURP transurethral resection of the prostate.								
† Postoperative day is calculated from the index TURP performed on September 25, 2025. Negative values indicate days before the index operation.								
‡ Value above the upper limit of the reference range and judged to be clinically significant.								
§ Value below the lower limit of the reference range.								
To convert values for hemoglobin to millimoles per liter, multiply by 0.6206. To convert values for CRP to nanomoles per liter, multiply by 9.524.								

On POD16, the patient developed sudden severe right scrotal pain and fever. Physical examination revealed an acutely swollen and tender right hemiscrotum. Emergency colour Doppler ultrasonography demonstrated epididymal enlargement, an enlarged right testis with tunica vaginalis fluid collection, and absent intratesticular blood flow; the left testis showed preserved perfusion and normal size (Fig. 2A,B). These findings raised immediate concern for testicular torsion.

Emergency scrotal exploration was performed. Approximately 15 ml of purulent fluid was found within the tunica vaginalis. The right testis was diffusely enlarged and showed approximately 120° lateral rotation. However, the spermatic cord vessels were preserved, and the intraoperative findings were not consistent with primary spermatic cord torsion. Acute epididymo-orchitis complicated by pyocele and secondary pseudo-torsion was diagnosed. The testis appeared viable and was preserved; drainage and local surgical management were performed rather than orchiectomy.

Postoperative wound drainage cultures repeatedly yielded *Pseudomonas aeruginosa*. Together with the operative findings and preceding urinary inflammatory abnormalities, these results supported a diagnosis of postoperative *P. aeruginosa* acute epididymo-orchitis with pyocele and secondary pseudo-torsion rather than true testicular torsion. Antimicrobial therapy was adjusted according to culture results and clinical evolution.

Despite emergency exploration, drainage, and antimicrobial therapy, the patient developed septic shock requiring intensive care admission, vasopressor support, and continued infection-control measures. With ongoing targeted antimicrobial therapy, supportive care, and local wound management, the infection gradually came under control. Serial postoperative Doppler examinations demonstrated progressive restoration of right intratesticular perfusion, transient hyperaemia during recovery, and eventual normalization of testicular size and blood flow (Fig. 2C–E). The testis was preserved throughout the course. The total hospital stay was 51 days.

This case illustrates a rare postoperative infectious cascade after transurethral prostate surgery. Preoperative bacteriuria, pyuria, positive nitrites, and elevated interleukin-6 suggested active infection, while persistent postoperative inflammatory-marker abnormalities preceded the acute scrotal event. Although Doppler ultrasonography suggested torsion because of absent intratesticular blood flow, operative and microbiological findings established severe *P. aeruginosa* acute epididymo-orchitis with pyocele and secondary pseudo-torsion, followed by septic shock.

Discussion

This case highlights a postoperative diagnostic cascade in which severe infection presented as acute scrotum and was initially interpreted as testicular torsion. Acute epididymo-orchitis is a recognized differential diagnosis of acute scrotal pain, but absent intratesticular blood flow on Doppler ultrasonography may create substantial diagnostic tension because this finding usually prompts urgent exploration for torsion^[1, 2]. In the present case, emergency exploration was appropriate, yet the operative findings established infection-driven pseudo-torsion with pyocele rather than primary spermatic cord torsion. A key feature of this case is that the infectious trajectory was detectable before the acute scrotal event. Preoperatively, the patient had marked pyuria, positive nitrites, elevated IL-6, and persistent PSA elevation despite two prior negative 12-core biopsies, supporting ongoing inflammatory or infectious activity rather than proven malignancy alone^[3–5]. After surgery, CRP remained abnormal at discharge and then rose further, preceding overt sepsis. This pattern suggests that postoperative inflammatory markers should not be interpreted in isolation or dismissed when the clinical course appears temporarily stable. The causative organism was *Pseudomonas aeruginosa*, a high-priority Gram-negative pathogen associated with virulence, biofilm formation, and antimicrobial resistance^[6, 7]. In urological patients, postoperative infection with *P. aeruginosa* may be particularly difficult to eradicate once tissue infection and purulent collection are established. Repeated positive drainage cultures in this patient strengthened the microbiological linkage between perioperative urinary infection and subsequent scrotal sepsis. This case also underscores the importance of source control. Once pyocele and severe local infection are present, antibiotics alone may be insufficient; timely drainage and surgical assessment are essential components of management^[8]. Nevertheless, even after source control, patients may progress to septic shock, requiring intensive care support in accordance with current sepsis principles^[9]. For resistant Gram-negative infections, early microbiology-guided escalation is also critical^[10]. Clinically, this case suggests three practical lessons. First, in older men presenting with acute scrotum after transurethral prostate surgery, infection should be considered alongside torsion even when Doppler shows absent testicular perfusion. Second, persistent postoperative CRP elevation after a procedure performed in the setting of bacteriuria should prompt closer reassessment. Third, microbiological confirmation and source control should be pursued early when the clinical course is discordant with an uncomplicated recovery. The main limitation is that this is only a single case, so the findings cannot be generalized to all TURP patients. Also, the relationship between TURP and the infection cannot be proven with certainty, and long-term follow-up information is limited.

Patient Perspective

Before surgery, I had experienced progressive difficulty with urination for several years, and I hoped that the transurethral resection of the prostate would improve my symptoms and quality of life. The early postoperative period seemed acceptable at first, but I later developed sudden scrotal pain and swelling, which was frightening. When I was told that there was concern for a serious condition requiring emergency scrotal exploration, I felt anxious but understood that urgent treatment was necessary to prevent further complications.

During the subsequent deterioration and treatment for septic shock, I was very worried about the severity of the infection and the possibility of long-term consequences. I appreciated the prompt surgical management, intensive care support, antibiotic treatment, wound care, and close monitoring provided by the medical team. As my condition gradually improved and I recovered enough to be discharged, I felt grateful for the timely interventions and for the explanations given to me and my family throughout the treatment process. Although the experience was physically and emotionally difficult, I believe the rapid diagnosis and active treatment helped me recover from a potentially life-threatening complication.

Declarations

Conflicts of Interest

The authors declare that they have no conflicts of interest relevant to this manuscript.

Ethical Approval and Consent to Participate

This study was conducted in accordance with the principles of the Declaration of Helsinki. As this is a single-patient case report, formal institutional review board approval was waived by the authors' institution. Clinical trial number: not applicable.

Consent to Publish

Written informed consent was obtained from the patient for the publication of this case report, including the accompanying clinical data and images. A copy of the written consent is available for review by the journal upon request.

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Author Contribution

J.L. and J.Z. collected the clinical data, performed the literature review, and drafted the main manuscript text. X.G. interpreted the clinical data, prepared the figure and table, and critically revised the manuscript. W.X. supervised the clinical management of the patient, conceptualized the case report, critically revised the manuscript for important intellectual content, and approved the final version for submission. All authors read and approved the final manuscript.

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Data Availability Statement

All data relevant to this case report are included in the article and its supplementary materials. Additional anonymized clinical information may be made available from the corresponding author upon reasonable request.

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Figures

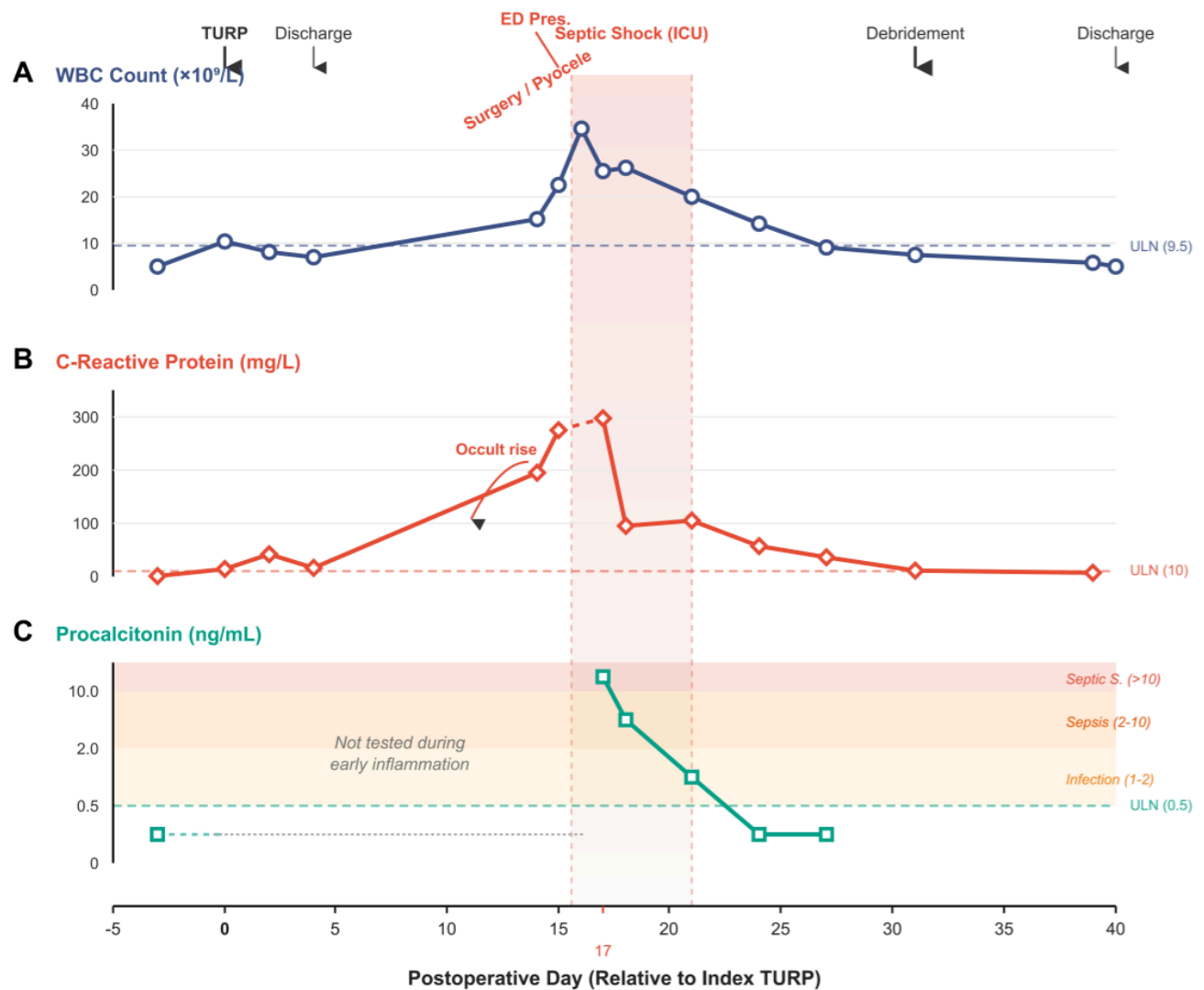


Figure 1

Multimodal inflammatory marker trajectories and clinical timeline of postoperative septic shock.

(A) White blood cell (WBC) count dynamics. (B) C-reactive protein (CRP) surges. (C) Procalcitonin (PCT) variations mapped against clinical risk strata. The temporal alignment of these three panels illustrates a critical postoperative diagnostic gap and highlights the clinical warning message of this case. While classical systemic markers (WBC and CRP) demonstrated a prominent, yet clinically silent "occult surge" starting as early as postoperative day (POD) 14 in an outpatient setting, PCT—a highly specific biomarker for bacterial sepsis—was not assessed until the acute onset of overt septic shock and localized scrotal symptoms on POD 17. Upon admission to the intensive care unit (ICU), PCT spiked dramatically into the severe septic shock risk zone (>10 ng/mL, highlighted in red shade in Panel C). The vertical red-shaded column across all subplots represents the critical deterioration period (POD 16–21) characterized by emergency scrotal exploration (pyocoele and pseudo-torsion discovery), overt septic shock, and ICU management. Horizontal dashed lines denote the upper limits of normal (ULN) for WBC ($9.5 \times 10^9/L$), CRP (10.0 mg/L), and PCT (0.5 ng/mL). *Clinical caution:* Sole reliance on the sudden presentation of acute local symptoms or late-stage testing of severe septic markers (such as PCT) obscures earlier systemic deterioration. The early dissociation between the apparently stable clinical phenotype and dramatically rising WBC/CRP on POD 14–15 represented a missed early window for source control prior to the life-threatening epididymo-orchitis cascade. (Note: Dashed connecting lines denote interpolated periods lacking specific daily testing data).

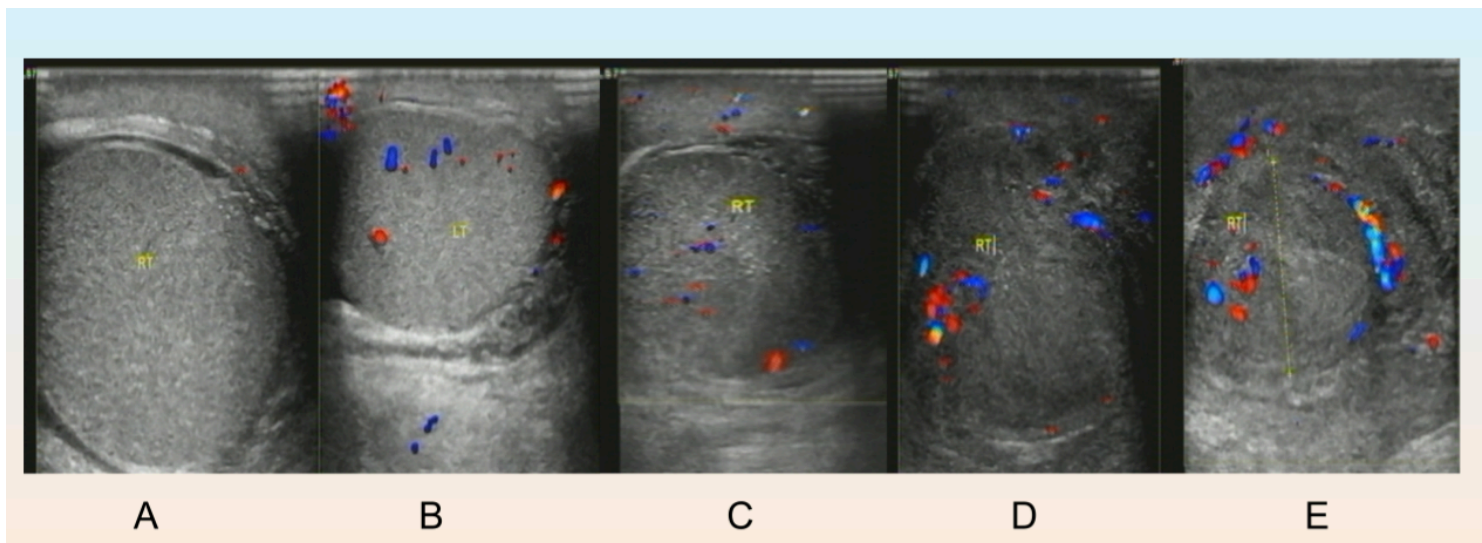


Figure 2

Serial Scrotal Doppler Ultrasonography Demonstrating the Dynamic Evolution of Acute Epididymo-Orchitis with Secondary Pseudo-Torsion.

Panel A. Right hemiscrotum on October 11 (postoperative day 16, pre-exploration): Absent intratesticular perfusion, marked testicular enlargement (5.0×4.0 cm), and turbid fluid within the tunica vaginalis — imaging findings mimicking testicular torsion but occurring in the context of systemic sepsis (WBC $34.6 \times 10^9/L$, CRP 297.6 mg/L).

Panel B. Left hemiscrotum on October 11 (pre-exploration): Normal intratesticular blood flow and testicular size, confirming the unilateral nature of the inflammatory process.

Panel C. Right hemiscrotum on October 13 (postoperative day 2): Restoration of intratesticular perfusion following emergency exploration, debridement, and tunica vaginalis flap inversion; however, testicular enlargement persisted due to residual inflammatory edema.

Panel D. Right hemiscrotum on October 29 (postoperative day 18): Hyperemic intratesticular flow indicative of active reperfusion and healing, with persistent but reduced enlargement — consistent with post-inflammatory tissue remodeling.

Panel E. Right hemiscrotum on November 12 (postoperative day 32): Normal intratesticular perfusion and complete resolution of testicular enlargement, confirming successful testicular salvage and resolution of acute inflammation.

Note: The initial Doppler findings (Panel A) underscore a critical diagnostic pitfall: severe acute epididymo-orchitis (AEO) can produce false-negative vascular signals due to inflammatory edema and vascular compression, thereby mimicking true torsion. Intraoperative findings of preserved spermatic cord vascularity and reversible discoloration with warm saline confirmed pseudo-torsion, not mechanical torsion — highlighting the necessity of integrating imaging, inflammatory markers, and surgical inspection in post-procedural acute scrotum.