A case of dysuria following periodontal abscess

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CASE

A warehouse officer, aged 45 years, presented to his general practitioner with a three-day history of dysuria. Associated symptoms included slow urinary stream and increased frequency to 10 times daily, as well as fatigue, malaise and anorexia (patient reported lack of appetite in the preceding three days). He denied any new sexual partners, episodes of frank haematuria or dysorgasmia. He was alert and afebrile on examination.

Preceding this presentation, he was hospitalised 10 days prior for right upper front incisor periodontal abscess. This was managed through incision, drainage and intravenous antibiotic therapy. His past medical history included well-managed fatty liver.

Laboratory investigations revealed elevated prostate-specific antigen (PSA; 5.9 μg/L) and C-reactive protein (CRP; 10.4 mg/L). The initial mid-stream urine (MSU) microscopy, culture and sensitivities (MCS) revealed sterile urine with nil pyuria (leukocytes 7×10^6/L). Because of the inconsistency with the clinical picture, a repeat MSU MCS was performed five days later, which revealed mild pyuria (leukocytes 20×10^6/L) and cephalexin-resistant enterococcus faecalis (>10^8 organisms).

QUESTION 1

What is the provisional diagnosis for this presentation?

QUESTION 2

How is acute bacterial prostatitis diagnosed?

QUESTION 3

What are the risk factors for acute bacterial prostatitis?

QUESTION 4

What is the management approach to acute bacterial prostatitis?

QUESTION 5

What are the potential complications of acute bacterial prostatitis?

QUESTION 6

Is the patient's recent history of periodontal disease relevant to the development of acute bacterial prostatitis?

ANSWER 1

The symptoms are suggestive of a urinary tract infection (UTI) with prostatic involvement. In middle-aged men, this presentation is typical of acute bacterial prostatitis (ABP). Prostatitis refers to the condition of prostate gland inflammation, which is classified as per the internationally accepted National Institutes of Health Classification System detailed in Table 1.[1] ABP is a subtype of prostatitis, characterised by rapid onset of symptomatic prostate inflammation caused by bacterial invasion.[2–4]

ANSWER 2

ABP is a clinical diagnosis, based on the presence of key features, outlined in Table 2. It is imperative to recognise ABP because despite shared features with other pathologies including simple UTI and sexually transmitted infection (STI), the management approach and complication risks differ.

ANSWER 3

ABP can develop through any mechanism that allows bacteria to reach the prostate gland.[2] Known mechanisms and their respective risk factors are outlined in Table 3.

ANSWER 4

ABP can be managed safely in the community if the patient is haemodynamically stable without signs of severe infection (Table 2), with Trimethoprim 300 mg orally daily for 14 days, as first-line therapy.[7] However, the choice of antibiotic should be guided by susceptibility testing and account for patient factors including allergies. Urgent hospital referral should be made for patients who meet any of the following indications for intravenous antimicrobial therapy:[7]

Table 1. Subtypes of prostatitis

	Type 1	Type 2	Type 3	Type 4
Name[1,3,4]	**Acute bacterial prostatitis**	**Chronic bacterial prostatitis**	**Chronic prostatitis or chronic pelvic pain syndrome**	**Asymptomatic inflammatory prostatitis**
Clinical features[4]	Acute development of irritative and obstructive urinary symptoms, systemic symptoms and pain	Symptoms of ABP or remains asymptomatic. Infection persists beyond a 3-month period	Obstructive urinary symptoms without any bacterial infection	There are no urinary or systemic symptoms, despite prostate inflammation
Proportion of cases[3,4]	Up to 10%	<5%	>90%	Rare
Causes	UTI: Majority of cases are caused by Gram-negative bacteria • *Escherichia coli* (50–87%)[2] • Entereobacterales (10–30%)[2] Minority of cases are caused by Gram-positive bacteria • Enterococcus (5–15%)[2] STI: • *Chlamydia trachomatis*[3] • *Neisseria gonorrhoea*[3]	ABP treated inadequately, which progresses to chronic infection, or a new bacterial infection triggers a state of chronic inflammation[3]	Cause unknown;[4] proposed it is a neuropathic pain[3] caused by peripheral nervous system sensitisation, which causes hyperalgesia and allodynia[4]	Cause and clinical significance unknown; typically an incidental finding (eg in prostate biopsy for exploration of infertility)[4]

ABP, acute bacterial prostatitis; STI, sexually transmissible infection; UTI, urinary tract infection.

Table 2. Features of acute bacterial prostatitis

Symptoms/signs	Features
Genitourinary	• Cloudy urine[3] • Digital rectal examination – prostate gland is tender, hot and boggy[2] • Dysorgasmia[1] • Dysuria[1] • Haematuria[1] • Increased urinary frequency[1] • Increased urinary urgency[1] • Oliguria or anuria, distended abdomen (acute urinary retention)[4] • Poorly localised pain – perineal, external genitalia, tip of penis, rectum, suprapubic region to lower back[3] • Poor urinary stream[2]
Systemic[3]	• Fever • Malaise • Myalgia • Nausea • Vomiting Severe/sepsis: • Tachycardia • Hypotension • Tachypnoea

Table continued on the next page

- severely unwell or have signs of sepsis – refer to Table 2[3,5]
- recent transrectal or transurethral prostatic manipulation[2]
- urinary retention – will additionally require catheterisation[5]
- inability to tolerate oral antibiotics[2]
- antibiotic resistance.[5]

Upon completion of the initial antibiotic course, healthcare practitioners should review the response to treatment. An additional 2–4 weeks of antimicrobial therapy should be prescribed if:[7]

- genitourinary or systemic symptoms persist
- repeat urine cultures display bacterial growth
- PSA levels remain elevated.

Supportive therapies can also be recommended to aid in symptomatic relief, including:[2]

- antipyretics
- anti-inflammatories
- alpha-blockers
- stool softeners.

Preliminary research has been conducted on direct antibiotic injection of Amikacin into the prostate gland, demonstrating

Table 2. Features of acute bacterial prostatitis (cont'd)

Investigation findings	• CRP – low specificity for ABP; however, this is useful in indicating presence of infection or inflammation[1] • First void urine specimen (PCR) – *Chlamydia trachomatis* or *Neisseria gonorrhoea*, in a ABP caused by an STI[3] • PPMT – note that this test is contraindicated in the investigation of suspected ABP because of the risk of bacteraemia; this is reserved only for investigation of suspected chronic bacterial prostatitis (refer to Table 4)[4] • PSA – not a diagnostic criterion for ABP, rather it is useful to indicate prostatic involvement, which might aid in excluding other differentials.[1] PSA is elevated (>4 µg/L) in 70% of ABP cases[1] • Urine culture – shows growth of bacterial species in ABP caused by a UTI.[1] If growth occurs, subsequent susceptibility testing should be performed to determine which antimicrobial agents the species is sensitive or resistant to[1] • Urinary dipstick, midstream urinalysis and full blood count – leukocytosis[1] • Transrectal or bladder ultrasound – indicated only if acute urinary retention is suspected to determine whether bladder emptying is incomplete[4]

ABP, acute bacterial prostatitis; CRP, C-reactive protein; PCR, polymerase chain reaction; PPMT, pre- and post-prostate massage test; PSA, prostate-specific antigen; STI, sexually transmissible infection; UTI, urinary tract infection.

Table 3. Risk factors of acute bacterial prostatitis

Mechanism	Associated risk factors
Reflux of infected urine[2]	• Malignant tumours[5] • Phimosis[6] • Prostate enlargement[6] • Urethral stricture[6] • Urinary tract stones[5]
Ascending urethral infection[2]	• Lower urinary tract intervention – indwelling catheter[2] • Transurethral surgery – infection likely to be from Pseudomonas species[4] • Urethritis[6]
Lymphatic invasion from rectum[2]	• Prostate manipulation[6] • Transrectal prostate biopsy[2]
Haematogenous infection[2]	• Cirrhosis[2] • Diabetes mellitus[2] • Immune system suppression (eg HIV/AIDS) – more likely to be infected with Cryptococcus, Salmonella or Candida species[4]

superior bacterial eradication rates (33.3% direct vs 5% IM, $P<0.05$) for ABP because of enhanced antibiotic concentration within the prostate gland.[5] As direct injection also reduces treatment duration and antibiotic resistance, it is emerging as a future treatment option for ABP.

ANSWER 5

If bacterial eradication is not achieved during ABP treatment, patients are at risk of the complications outlined in Table 4.

ANSWER 6

The literature suggests that periodontal disease and prostatitis are correlated through the theorised mechanisms outlined in Table 5. As there is no evidence to demonstrate that increased periodontal disease incidence increases the incidence of prostatitis, further research is imperative in clarifying the significance of the proposed correlation and the implications for clinical practice.[8]

CASE CONTINUED

This patient was treated for ABP with a course of Trimethoprim 300 mg orally daily for seven days after shared decision making between the patient and general practitioner. Post-treatment consultation confirmed full resolution of symptoms. Repeat tests demonstrated regression of pyuria with no subsequent bacterial growth, reduction of PSA to 0.64 g/L (within normal range) and reduction of CRP to 0.4 mg/L (within normal range). The patient experienced no further complications of ABP.

Key points

- It is important to consider ABP in male patients presenting with UTI or STI.
- Patients should be reviewed. post-treatment to confirm resolution of symptoms, no further culture growth and a normal PSA level.
- There is a possible, yet inconclusive correlation between prostatitis and periodontitis.

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Table 4. Complications of acute bacterial prostatitis

Complication	Clinical features	Further investigations	Treatment	Other notes
Chronic bacterial prostatitis[1]	Symptoms of ABP persist, or patient is asymptomatic	PPMT – leukocytosis in post-prostatic massage urine specimen, positive culture	Antibiotics for 6 weeks	There is lower tissue penetration of antibiotic therapy because of less inflammation compared to ABP
Chronic pelvic pain syndrome[1]	Penile pain, inguinal pain, pudendal discomfort, prostate hyperplasia, bladder outlet obstruction	PPMT – no bacteraemia detected	Alpha-1 blockers, phosphodiesterase type 5 inhibitors	Can impact mental health and functioning causing insomnia and depression
Epididymitis[1]	Urethral damage, genital swelling, tenderness	Ultrasound – to rule out testicular torsion	Oral quinolone for 14–21 days	If untreated it can narrow the passage sperm must pass and contribute to infertility
Prostate abscess	Elevated inflammation markers despite antibiotic treatment[3]	Transrectal ultrasound[6]	If >1 cm, perform surgical drain (TURP)[6]	Typically caused by *Escherichia coli* and MSSA.[1] Has an incidence of 2–18%.[2] Risk of prostate abscess increases in immune-suppressed patients[2]
Sepsis[3,5]	Tachycardia, hypotension, pyrexia, tachypnoea, neutrophilia or neutropenia	Blood cultures, platelets, bilirubin, creatinine	Intravenous antibiotics, vasopressors, fluids, oxygen	Prostatic massage contraindicated when performing DRE for ABP patients

ABP, acute bacterial prostatitis; DRE, digital rectal examination; MSSA, methicillin-sensitive *Staphylococcus aureus*; PPMT, pre- and post-prostate massage test; TURP, transurethral, percutaneous or open.

Table 5. Proposed prostatitis and periodontal disease correlation

Proposed mechanism	Evidence
Hematogenous spread of pathogens from the periodontium to the prostate gland[8]	There are common pathogens in periodontal disease and prostatitis, including: *Escherichia coli*, *Porphyromona gingivalis*, *Fusobacterium nucleatum* and *Actinomyces actinomycetemcomitans*[8]
Systemic inflammatory cytokines (IL-6, TNF-α, IL-1) released during periodontitis predispose patients to prostatitis because of chronic inflammation and endothelial dysfunction[8,9]	Inflammatory cytokines are found in the serum of prostatitis patients[10]
The periodontium as a site of PSA generation elevated distant PSA levels[8,10]	The prostate gland has inflammatory reactions to elevated PSA levels[8]
All of the above	Clinical evidence supports the proposed correlation, as a cohort of periodontitis patients followed over a 15-year study period were found to have an 11% rate of prostate disorder and a 4.6-fold increase in prostatitis risk (P=0.001)[9]

IL, interleukin; PSA, prostate-specific antigen; TNF, tumour necrosis factor.

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References

1. Yang Y, Shigemura K, Maeda K, et al. The harmful effects of overlooking acute bacterial prostatitis. Int J Urol 2024;31(5):459–63. doi: 10.1111/iju.15390.
2. Matsumoto M, Yamamoto S. AAUS guideline for acute bacterial prostatitis 2021. J Infect Chemother 2021;27(9):1277–83. doi: 10.1016/j.jiac.2021.06.001.
3. Kanani S, Mujtaba N, Sadler P. Acute and chronic prostatitis. InnovAiT 2021;14(1):33–37. doi: 10.1177/1755738020966359.
4. Khan FU, Ihsan AU, Khan HU, et al. Comprehensive overview of prostatitis. Biomed Pharmacother 2017;94:1064–76. doi: 10.1016/j.biopha.2017.08.016.
5. Marquez-Algaba E, Burgos J, Almirante B. Pharmacotherapeutic interventions for the treatment of bacterial prostatitis. Expert Opin Pharmacother 2022;23(9):1091–101. doi: 10.1080/14656566.2022.2077101.
6. Shakur A, Hames K, O'Shea A, Harisinghani MG. Prostatitis: Imaging appearances and diagnostic considerations. Clin Radiol 2021;76(6):416–26. doi: 10.1016/j.crad.2021.01.007.
7. Therapeutic Guidelines. Antibiotic – Prostatitis. Therapeutic Guidelines Limited, 2019. Available at https://tgldcdp.tg.org.au/viewTopic?etgAccess=true&guidelinePage=Antibiotic&topicfile=prostatitis&guidelinename=Antibiotic&sectionId=toc_d1e47#toc_d1e47 [Accessed 17 March 2024].
8. Ortíz de Urbina Comerón P, Zubizarreta-Macho Á, Lobo Galindo AB, Montiel-Company JM, Lorenzo-Gómez MF, Flores Fraile J. Relationship between prostate inflammation and periodontal disease – A systematic review and meta-analysis. J Clin Med 2023;12(18):6070. doi: 10.3390/jcm12186070.
9. Fu E, Cheng CM, Chung CH, et al. Association of chronic periodontitis with prostatic hyperplasia and prostatitis: A population-based cohort study in Taiwan. J Periodontol 2021;92(1):72–86. doi: 10.1002/JPER.19-0706.
10. Boyapati R, Swarna C, Devulapalli N, Sanivarapu S, Katuri KK, Kolaparthy L. Unveiling the link between prostatitis and periodontitis. Contemp Clin Dent 2018;9(4):524–29. doi: 10.4103/ccd.ccd_746_18.

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