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An unusual complication of bacterial prostatitis

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ABSTRACT

Acute bacterial prostatitis is a common genitourinary infection affecting males. *Escherichia coli* (*E. coli*) is a leading uropathogen implicated in cases of acute bacterial prostatitis, but is not attributed as a common cause of acute myocarditis. This article reports on an unusual case of concurrent myocarditis in a young man with *E. coli*-related prostatitis and urinary sepsis.

Introduction

Prostatitis is a common bacterial genitourinary infection. *E. coli* and *Enterococcus* family bacteria are responsible for the majority of cases, with the predominant etiology being ascending infection. Myocarditis is not considered a typical complication of *E. coli* related sepsis, and there is a scarcity of literature documenting this in clinical practice. This case demonstrates the investigation of acute chest pain in a patient with bacterial prostatitis, and aims to broaden the differential of myocarditis as a sequalae of *E. coli* related infection.

Case presentation

A 34-year-old Caucasian man presented to the Emergency Department complaining of a febrile illness and a one-month history of preceding lower urinary tract symptoms, including urinary frequency and dysuria. Prior to presentation, he had received multiple courses of antibiotics by his primary care team, which had failed to resolve his symptoms. He was otherwise a fit and well male with no medical history of note. He was sexually active with a monogamous female partner and had no previous history of urinary or sexually transmitted diseases.

On presentation, the patient's vital observations included a fever with a temperature measured at 38.6° Celsius. His blood pressure was measured at 132/84 mmHg and he was noted to be tachycardic with a heart rate of 110 beats per minute. His clinical examination was unremarkable except for a digital rectal exam which confirmed an enlarged and tender prostate. Investigations included a full blood count, with an elevated white cell count of 30.5 $\times$ 10^9/L, predominantly due to neutrophilia (neutrophil count of 28.3 $\times$ 10^9/L)). Urine samples were sent for bacterial culture and sensitivity, which were found to contain >100 $\times$ 10^6 cfu/L of non-O157:H7 *Escherichia coli*. This was globally sensitive to all antibiotics on routine sensitivity testing. There was no evidence of bacteremia on peripheral blood culture samples taken prior to the administration of intravenous antibiotics. The patient underwent computed tomography imaging as part of his evaluation, and this demonstrated prostatomegaly measuring approximately 60 cc, with external iliac nodal enlargement. The radiological findings were consistent with acute prostatitis on formal reporting.

The patient was admitted to hospital for treatment with antibiotic therapy, and ceftriaxone (cephalosporin) 2g once daily was administered intravenously as per his bacterial sensitivities and local guidelines. Forty-eight hours after admission, his presentation was complicated by the development of new-onset severe chest pain, which was central in nature. A twelve-lead electrocardiogram (ECG) performed at the time demonstrated normal sinus rhythm with no features to suggest acute ischemia. His chest pain was intermittent for the next 6 h, with serial ECGs performed revealing no new changes. Biochemistry taken at the onset of the patient's chest pain included high sensitivity troponin-T (hsTNT), which were elevated on testing at 72 ng/L (reference range 0–13 ng/L). Repeat biochemistry at three and six hours later revealed further elevation of his measured hsTNT to 190 ng/L and 303 ng/L respectively. The patient was reviewed by the general medical service, with an impression of non-ST elevation myocardial infarction (NSTEMI).

Acute transfer from our regional hospital to a tertiary hospital with

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an on-call cardiology service was arranged, and the patient underwent cardiac angiography. This demonstrated normal coronary arteries without a demonstrable ischaemic etiology for his chest pain and significantly elevated hsTNT. The diagnosis was subsequently revised to be acute myocarditis rather than NSTEMI, and the patient received supportive therapy for this including non-steroidal anti-inflammatory medications.

The patient completed five days of intravenous antibiotics and had clinical improvement with his acute prostatitis, and he had no further episodes of chest pain. He was subsequently discharged home and completed a total fourteen-day course of antibiotics.

On outpatient follow-up, the patient remained well without further evidence of urinary tract infection, or recurrence of chest pain. Cardiac magnetic resonance imaging (MRI) was performed one month after discharge to evaluate for residual disease burden. This showed normal ventricular (LVEF 59%) and valvular function, demonstrating a structurally normal heart without evidence of fibrosis.

Discussion

E. coli is a common pathogen in extra-intestinal infections, particularly of the urinary tract.[1] However, few cases of *E.coli*-related prostatitis with concurrent development of myocarditis have been reported in the literature. Documented cases of *E.coli*-related myocarditis in the context of urosepsis have been associated with poor cardiac outcomes and morbidity.[2] Myocarditis is thought to be predominantly attributable to viral, pharmacological or autoimmune causes. The prevalence of acute bacterial myocarditis is not well defined, and there are no established diagnostic criteria.[3] The definitive diagnosis of myocarditis ultimately requires invasive sampling with endomyocardial biopsy; in practice, this may be unfeasible except in postmortem.[2] Due to these factors, acute bacterial infection is not typically considered a significant differential in the clinical evaluation of chest pain.

The pathophysiology of an acute bacterial infection causing myocarditis is unclear. Viral pathophysiology has been described in animal studies, and typically involves the formation of autoantibodies following clearance of the virus by the host response. These can cause myocyte injury and may result in permanent myocardial damage.[4] It has long been established that septicemia may result in myocardial dysfunction, but the link between this process and the development of myocarditis as a result of bacterial infection remains speculative.[4] Potential mechanisms include bacterial dissemination, bacterial toxin production, altered host immune response and myocardial dysfunction in the context of sepsis.[3]

In this case report, the sequence of clinical symptoms and the radiographical absence of coronary artery disease allowed us to establish a clinical diagnosis of acute myocarditis associated with *E. coli* bacterial prostatitis. This case adds to the body of evidence implicating *E. coli* as an organism related to the development of acute bacterial myocarditis.

Conclusion

Acute myocarditis is uncommon and not typically associated with bacterial prostate infection. Misdiagnosis of acute coronary syndrome may occur in patients with elevated cardiac enzymes in the context of acute and severe systemic illness. Acute bacterial myocarditis, although unusual, should be considered in patients who have presentations suggestive of acute coronary syndrome in the context of bacterial sepsis.

Consent

The authors have received consent from the patient to perform this case report.

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Declaration of competing interest

The authors declare that they have no potential conflict of interest.

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