Case Report

Post colonoscopic acute bacterial prostatitis: a case report

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ABSTRACT

Colonoscopy is a common procedure for diagnosing a wide range of conditions and symptoms affecting the large bowel. Research has shown that the examination itself may induce transient bacterial infections. Specifically acute bacterial prostatitis (ABP) has little mention in medical literature as a recognized complication of this procedure. Here we discuss a 37 year old male presenting with symptoms suggesting lower urinary tract infection after having undergone colonoscopy followed by recurrent episodic haematuria and lower urinary tract symptoms (LUTS). Physicians and endoscopists should be aware of the risk of acute bacterial prostatitis as a potential complication of colonoscopy in order to minimize misdiagnosis as well as the complications associated with the delayed treatment of it. In addition male patients and the immunocompromised should be fully counselled regarding this risk prior to undertaking this procedure.

Keywords: Colonoscopy, Acute bacterial prostatitis, Episodic haematuria

INTRODUCTION

Colonoscopy is a diagnostic and therapeutic procedure that allows visualization and treatment of the large bowel and terminal portion of the ileum.

The risk of serious complications associated with this procedure is low. Complications that are commonly cited include those related to sedation and bowel preparation, bleeding, perforation and infection. Most commonly reported infections following colonoscopy include respiratory (aspiration pneumonia) and gastrointestinal.

The rate of genitourinary infection post colonoscopy has been reported to be between 0.045 and 0.048 per 1000 procedures. However, it is unclear whether this figure includes acute bacterial prostatitis.

What is well known is that prostate manipulation from procedures such as transrectal or transurethral prostate biopsy, cystoscopy, and catheterization can increase the risk of developing acute bacterial prostatitis. Although there are no reports of bacterial prostatitis occurring acutely after colonoscopy Tsai et al have reported an association between antecedent colonoscopy and chronic prostatitis/chronic pelvic pain syndrome. The standard for diagnosing acute bacterial prostatitis (ABP) relies on the finding of bacteria within prostatic fluid. However, techniques to express prostatic fluid can be difficult to carry out and are hardly performed in clinical practice.

Rather, chronic bacterial prostatitis is often diagnosed clinically and empirically treated with antibiotics when men present with chronic or recurring lower urinary tract symptoms, especially in the presence of bacteriuria.

In this paper we present a case of acute bacterial prostatitis occurring acutely after the patient had undergone colonoscopy and its successful treatment and follow up.
CASE REPORT

A 37 years old male patient with a 10 year history of ulcerative colitis underwent colonoscopy in the ambulatory unit for evaluation of disease extent and severity. The procedure was carried out under sedation and was uneventful intraoperatively. Colonic appearance was in keeping with a mayo 2 severity score for left sided ulcerative colitis. Multiple tissue biopsies were taken for which the histological examination was in keeping with UC. The patient was discharged home with treatment in the form of budesonide MMX 9 mg PO OD, mesalazine granules 4g PO OD, mesalazine enema 1g/2 weekly.

24 hours later the patient presented to the health centre with symptoms of fever, rigors, myalgia, urinary hesitancy, dysuria, strangury and haematuria. He was afebrile at the time of presentation. Clinical examination was unremarkable. A digital rectal examination was not performed.

Urinalysis revealed turbid urine and confirmed the presence of haematuria with >50 red blood cells (RBC)/hpf, with no presence of urinary nitrite or leucocytes. Complete blood count (CBC) and renal function was within normal limits. Serum prostate-specific antigen (PSA) and C-reactive protein (CRP) was not analysed. He was diagnosed as having a suspected urinary tract infection and discharged home with co-amoxiclav 625 mg TID for 7 days. His symptoms had cleared over the next 24 hours.

He represented with symptoms of haematuria, dysuria, strangury and urinary hesitancy after 2 months and was treated empirically with ciprofloxacin 500 mg PO BID for 7 days. Ultrasound examination of the renal tract at this stage revealed a solitary right kidney and a normal sized prostate with no significant post void bladder urine.

He presented again to the emergency department 8 months later complaining of abdominal pain and haematuria, dysuria and strangury. Urinalysis confirmed the presence of haematuria and was negative for leucocytes and nitrites. A computed tomography of kidneys, ureters and bladder (CT KUB) confirmed a solitary right kidney and revealed a 2.5 mm calcific density along the posterior aspect of the prostate gland. He was discharged from hospital with a presumptive diagnosis of having passed a renal stone. Outpatient investigation with flexible cystoscopy later revealed dilated prostatic veins over the prostatic urethra as the source of bleeding. The urinary bladder was normal in appearance and cytology was negative for malignancy.

He was subsequently treated with levofloxacin 500 mg OD for 4 weeks with complete resolution of symptoms.

DISCUSSION

ABP refers to swelling and inflammation of the prostate gland and can be caused by bacterial colonization of the prostate gland from the urinary tract and from direct extension or lymphatic spread from the rectum.

It is most commonly caused by infection from members of the Enterobacteriaceae family, but organisms from other families can be responsible and are more likely in certain high-risk populations. Escherichia coli is the most common isolate from urine cultures and is the causative agent in the majority (approximately 50% to 90%) of cases.

Other commonly isolated organisms include Proteus, Klebsiella, Enterobacter, Serratia, and Pseudomonas. ABP may also result from various sexually transmitted infections such as Neisseria Gonorrhoeae and Chlamydia trachomatis.

Risk factors thought to predispose to ABP include phimosis, intraprostatic ductal reflux, unprotected vaginal/anal intercourse, development of a urinary tract infection, congenital abnormality of the ureter and sexual abuse.

Patients with acute bacterial prostatitis typically present with symptoms of fever, chills, malaise, myalgia, dysuria, irritative urinary symptoms (frequency, urgency, and urge incontinence), pelvic or perineal pain, and cloudy urine. There may also be pain at the tip of the penis. Swelling of the acutely inflamed prostate can also cause voiding symptoms, ranging from dribbling and hesitancy to acute urinary retention.

Laboratory findings of leukocytosis, pyuria, bacteriuria, or an elevated serum PSA level can support the diagnosis and should prompt consideration of a digital rectal exam. Urine gram stain and culture should be obtained in all men suspected of having ABP in order to definitively establish etiology.

In our case the patient’s clinical features and the fact that he was successfully treated with antibiotics points strongly to a diagnosis of ABP. The appearance of the prostate gland with calcification and dilated veins lends further support to this diagnosis.

Limitations in the initial assessment of the patient’s condition include the fact that the serum PSA was not checked and a digital rectal examination was not performed. In addition, evidence of microbiology etiology was not obtained on specimen culture.

There was prompt resolution of symptoms with initial antibiotic treatment, however there was a recurrence of symptoms soon after cessation of antibiotic treatment. The first 2 courses of treatment given were both for 1 week each. Shorter antibiotic regimens have been associated with progression to chronic symptoms. Not all antibiotics can penetrate into prostatic tissue and the presence of acute inflammation generally allows entry of drugs that would not otherwise achieve therapeutic levels.
A more prolonged course of antimicrobial therapy should be favoured because of limited antimicrobial penetration into the prostate and the development of protected microcolonies deep within the inflamed gland that can be difficult to reach with antimicrobials. In our case there was complete resolution of symptoms without recurrence following a 4 week treatment regimen of levofloxacin 500 mg OD.

Fluorquinolones achieve high levels in prostatic tissue. Although this may not be an issue in the acute setting, where prostatic inflammation allows penetration of a broader range of antibiotics. The ability of an antibiotic to penetrate prostate tissue is thought to be important during prolonged therapy, while inflammation is resolving.

**CONCLUSION**

Prostatitis syndromes are common and can significantly impact quality of life. In addition delayed diagnosis and treatment of ABP can result in serious and difficult to treat complications including chronic prostatitis, metastatic infection (spinal or sacroiliac), abscess and sepsis. This case report highlights the importance of maintaining a high index of suspicion for ABP in males presenting after colonoscopy as well as fully counselling patients prior to undergoing colonoscopy. Further studies are needed to fully appreciate the mechanism of injury and fully determine the risk posed.

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**REFERENCES**
