

Infective Endocarditis Following TURP Procedures: A Case Report and Review of Literature

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Abstract A common treatment and management of BPH is transurethral resection of the prostate (TURP) with at least 150,000 TURPs performed per year in the United States. Rates of bacteremia following TURP can be as low as 1% when antimicrobial prophylaxis is given. Patients can develop many common sequelae as a result of bacteremia secondary to TURP including bacteriuria and urinary tract infection; however, more serious complications such as endocarditis are quite rare (~1 in 4200 cases). Here we present a case of a 67 year-old-male with BPH who underwent two TURP procedures; both with appropriate antibiotic prophylaxis who was subsequently diagnosed with endocarditis

Keywords: Endocarditis, TURP, BPH, infective endocarditis

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1. Introduction

The prevalence of benign prostatic hyperplasia (BPH) increases after the age of 40, with BPH affecting 70% of US men 60–69 years of age and 80% of those 70 years of age or older [1]. A common treatment and management of BPH is transurethral resection of the prostate (TURP) with at least 150,000 TURPs performed per year in the United States [2]. TURPs are associated with significant bacteremia if antimicrobial prophylaxis is not perioperatively [3]. Rates of bacteremia following TURP range from 0-31%, but is as low at 1% when antimicrobial prophylaxis is given [3,4]. Patients can develop many common sequelae as a result of bacteremia secondary to TURP including bacteriuria and urinary tract infection; however, more serious complications such as endocarditis are quite rare (~1 in 4200 cases) [5]. Here we present a case of a 67 year-old-male with BPH who underwent two TURP procedures; his first in April 2019 and second in September of 2019, with adequate antibiotic prophylaxis, who was subsequently diagnosed with endocarditis in November 2019.

2. Case Report

Patient is a 67-year-old male with a past medical history of diabetes, hypertension, BPH s/p two TURP procedures in April 2019 and September 2019 who presented to the hospital complaining of chest and abdominal pain for two days. Patient explained that he began experiencing chest pain after stretching over a bar

for prolonged periods of time while cleaning the trunk of his car. His pain was initially 9/10 in intensity and improved significantly with tylenol. He described his abdominal pain as dull and associated with one episode of diarrhea. Patient also complained of general malaise, weakness and a 20 pound unintentional weight loss over three months. Blood pressure on admission was 149/66 mmHg, heart rate was 92 beats per minute, temperature was 99.6 degrees F, respiratory rate was 19 breaths per minute, and pulse oximetry was 100% on room air. Patients' physical exam was significant for mild tenderness over his sternum and his left chest wall; his cardiac and lung exam was unremarkable.

Laboratory work on admission was significant for elevated lipase of 218 (13-60U/L), ESR of 127 (0-20mm/h), CRP 119.51 (1.0-4.0 mg/L), and a positive urinalysis with 1+ leuk esterase, 1+ glucose, 1+ protein, trace Hemoglobin, and >100 WBC UF/hpf. Patients EKG was unremarkable with normal sinus rhythm, and his troponin was negative. Blood cultures and urine cultures were taken for further workup. A summary of all blood work performed for the patient can be found in Table 1.

On admission a chest x-ray was performed which showed a mildly enlarged cardiac silhouette, but did not show any evidence of pneumothorax or fracture. A computed tomography angiography (CTA) of his thorax and abdomen was performed which ruled out aortic dissection and aneurysm; it showed an enlarged prostate gland, a 1.0 cm nodular lung density in the right lung base, a 3 mm non obstructing calculus in the lower pole of the right kidney and focal rectal wall thickening with mild perirectal fat stranding. A bedside ECHO was performed in the emergency room which showed trace B lines and a

trace pericardial effusion. Patient was admitted to the hospital for IV antibiotics to treat his urinary tract infection and monitor his troponin levels as well as EKGs.

On the evening of the patient's first day in hospital, he spiked a fever to 102.2 degrees F; he was subsequently re-cultured and ceftriaxone IV was continued for the patient. His urine culture did not grow any bacteria; however his blood cultures were flagged as positive for growth of gram positive bacilli. An official echocardiogram (ECHO) was performed which showed an ejection fraction (EF) of 55-60%, PASP of 38 mmHg, moderate tricuspid regurgitation and a hypodensity on the free leaflet of the tricuspid valve. The patient was promptly started on IV Vancomycin for treatment of endocarditis, and daily blood cultures were done until the documented resolution of the bacteria. Subsequently a trans-esophageal echocardiogram (TEE) was performed to further assess the hypodensity which identified a large mobile vegetation measuring 1.72cm x 1.59cm on the tricuspid valve (Image 1), as well as a vegetation on the inferior aspect of the non coronary cusp of the aortic valve. Patient's blood cultures grew *Enterococcus Faecalis* and he was started on Ampicillin and Gentamicin, and transferred to another facility where he was evaluated by cardiothoracic surgery. Patient had a repeat ECHO at this facility which demonstrated vegetations involving all three leaflets of the tricuspid valve; a 2.3cmx1.5cm vegetation on the posterior leaflet, a 1.5cmx1.1cm on the anterior leaflet and a smaller vegetation on the septal leaflet. Patient was deemed not to need any acute surgical intervention and was discharged from the hospital with a total 6 week course of ampicillin and ceftriaxone via peripherally inserted central catheter (PICC). Patient has since completed his antibiotic course and had a repeat ECHO which demonstrated a mobile vegetation measuring 2.1cm x 1.6cm on the anterior leaflet 2.0cmx1.4cm vegetation on the posterior leaflet of the tricuspid valve; the small septal leaflet vegetation was not seen on this study. Patient is following with both infectious disease and cardiology as an outpatient and is scheduled for a colonoscopy in the coming months.

3. Discussion

The initial step in the establishment of a vegetation is endocardial injury, followed by focal adherence of platelets and fibrin. Then, the initially sterile platelet-fibrin nidus becomes secondarily infected by microorganisms circulating in the blood, either from a distant source of focal infection or as a result of transient bacteremia from a mucosal or skin source [13]. It is widely accepted that transient bacteremia which leads to the onset of IE are associated with surgical procedures in the fields of dentistry, otorhinolaryngology, urology, and obstetrics and gynecology. In fact, all major urology guidelines recommend the use of antimicrobial prophylaxis during a TURP [8,9,10]. However, antimicrobial prophylaxis are usually used to decrease the incidence of postoperative bacteriuria, rather than other significant infective parameters such as bacteraemia, bloodstream infection or severe sepsis including endocarditis [2]. In fact, urological instrumentation is associated with enterococcal endocarditis with an estimated rate of enterococcal IE of fewer than 1 endocarditis case every

4000 procedures [2], as was seen in our case. It is thought that Bacteraemia might occur as a consequence of trauma to the prostate tissue during the resection. Furthermore, there may be no concordance between urine culture results and bacteraemia [2], similar in our patient.

The Duke criteria are a collection of major and minor criteria used to establish a diagnosis of infective endocarditis. It combines major and minor clinical criteria for establishing diagnosis. Major criteria include positive blood cultures from typical endocarditis organisms and evidence of endocardial involvement often with the use of echocardiography. Minor criteria includes vascular phenomena (arterial emboli, pulmonary infarcts, Janeway lesions, conjunctival hemorrhage), immunological phenomena (glomerulonephritis, Osler's nodes, Roth's spots, Rheumatoid factor), fever >38 degrees celsius, predisposing condition (IV drug user, known cardiac lesion, etc.), or microbiological data that does not conform to fit major criteria. A diagnosis of IE is made when two major clinical criteria are met, one major and three minor criteria, or five minor criteria.

It is said that the formation of a focus of infection is associated with a high-speed blood flow or a direct collision with the endocardial surface, and onset is rare at a site where no abnormal high-speed blood flow exists [15]. Vegetations tend to develop at sites where blood travels from an area of high pressure through a narrow orifice into an area of lower pressure. Given that our patient likely had pre-existing moderate tricuspid regurgitation as demonstrated on the initial echo, this underlying cardiac environment likely predisposed our patient to injury allowing a sterile platelet-fibrin nidus to ultimately become infected with enterococcus bacteria as a result of a TURP procedure.

The management of infective endocarditis (IE) includes prompt diagnosis, treatment with antimicrobial therapy, and in some cases of complicated IE, surgical management. In our present case, there was no evidence of severe valvular dysfunction requiring CT surgery and the patient was appropriately treated with a 6 week course of antibiotics as mentioned in the case report. Early diagnosis and treatment of IE is essential to prevent further complications including possible septic emboli, severe sepsis, abscess formation, CHF, or conduction block [16]

4. Conclusions



Image 1. Vegetation on Aortic noncoronary cusp/TV vegetation (short axis)

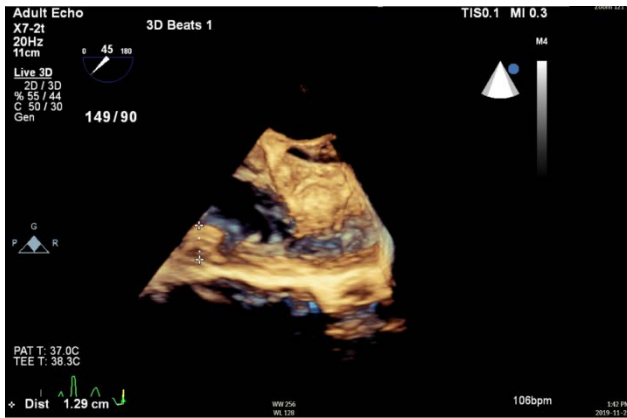


Image 2. 3D View of TV vegetation

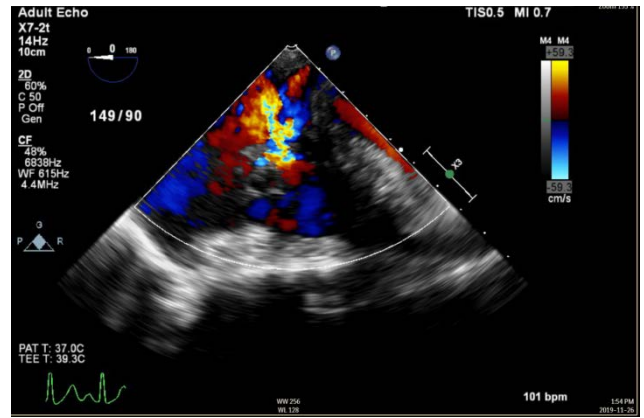


Image 6. Tricuspid Regurgitation jet in 4 chamber view

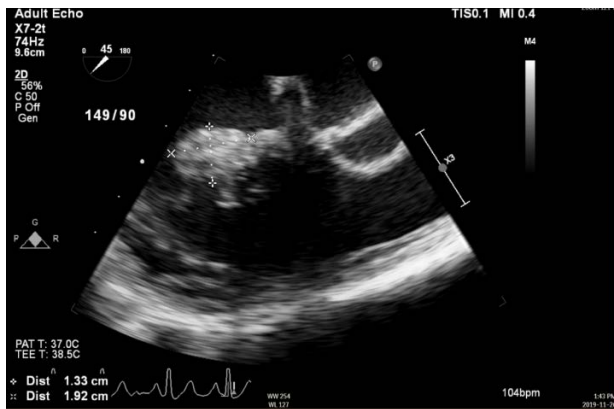


Image 3. Sizing TV vegetation

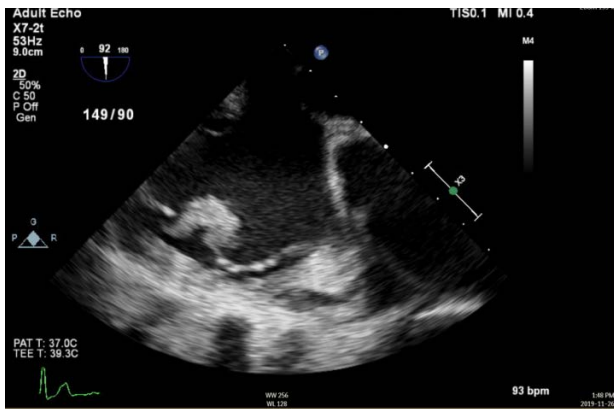


Image 4. TV vegetation



Image 5. 3D view TV vegetation

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