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Tuberculous Psoas Abscess and Worsening Vascular Aneurysm; All from Bacillus Calmette-Guerin (BCG) Therapy?

Authors' Contribution:
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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 76
Final Diagnosis: Tuberculous psoas abscess
Symptoms: Fever
Medication: —
Clinical Procedure: —
Specialty: Infectious Diseases

Objective: Rare disease

Background: Intravesical bacillus Calmette-Guerin (BCG) is used in the treatment and prophylaxis of carcinoma *in situ* of the urinary bladder and for the prophylaxis of primary or recurrent stage Ta and T1 papillary tumors following transurethral resection. Significant systemic complications are rare but have been reported.

Case Report: We describe this case of *Mycobacterium bovis* psoas abscess and worsening abdominal aortic aneurysm following BCG therapy for bladder cancer. A 76-year-old male presented with a fever of a few days. He had a computed tomography (CT) scan of abdomen and pelvis that showed left iliopsoas fluid collection measuring 6.7×3.8 cm and an abdominal aortic aneurysm that had almost doubled in size from 4.9 cm to 8.5 cm. The patient underwent CT-guided aspiration of the iliopsoas collection. *Mycobacterium bovis* was isolated from the aspirate cultures. He had received intravesical BCG therapy for bladder cancer a few years prior.

Conclusions: The rapid increase in the size of the abdominal aortic aneurysm (mycotic aneurysm) in our patient was most likely due to BCG therapy. The risk-benefit assessment of this treatment should be carefully considered especially in patients with a pre-existing vascular aneurysm.

MeSH Keywords: Aortic Aneurysm, Abdominal • *Mycobacterium bovis* • Psoas Abscess • Urinary Bladder Neoplasms

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Background

Intravesical bacillus Calmette-Guerin (BCG) therapy is an effective treatment option for bladder cancers. Significant systemic complications have been reported such as *Mycobacterium* infections as well as vascular complications. We present a case of *Mycobacterium bovis* abscess and worsening abdominal aortic aneurysm in a patient who was treated with BCG therapy for bladder cancer previously.

Case Report

A 76-year-old male presented to the hospital with high grade fever and weakness for five days. Other associated symptoms included night sweats and weight loss. He reported no abdominal pain, cough, or shortness of breath.

His past medical history was significant for bladder cancer, diagnosed in 2013. The patient was treated with six weeks of intravesical BCG and valrubicin. He had recurrent carcinoma *in situ* of the bladder and left distal ureteral tumor in 2016. He received another course of intravesical BCG and interferon A. He subsequently underwent distal urethrectomy and left radical nephroureterectomy in February of 2016. He had a 4.0 cm abdominal aortic aneurysm (AAA) in 2007 that increased to 4.9 cm in 2012.

Physical examination revealed a chronically ill looking patient, who was hemodynamically stable. He was febrile with a temperature of 102°F (38.9°C). Computed tomography (CT) of the chest, abdomen, and pelvis showed stable pulmonary nodules, worsening AAA measuring 8.5 cm in size (Figure 1); a left iliopectus fluid collection measured 6.7×3.8 cm (Figure 2). The patient underwent CT-guided aspiration of the left iliopectus collection. The specimen was sent to the microbiology laboratory for bacterial, fungal, and acid fast bacilli (AFB) cultures. Initial stains revealed AFB. *Mycobacterium bovis* was later recovered after three weeks of incubation. The organism was resistant to pyrazinamide.

The patient was found to have a high preoperative cardiac risk index; surgical repair of the AAA was therefore not pursued. The patient was managed conservatively with percutaneous drainage and anti-tuberculous medications; initially isoniazid, rifampin, and ethambutol. This regimen was stepped down to isoniazid and rifampin after two months. He initially responded to treatment with resolution of the fevers and decreased size of the iliopectus abscess. He subsequently passed away about seven months into treatment.



Figure 1. Computed tomography scan with arrow head showing abdominal aortic aneurysm.



Figure 2. Computed tomography scan with arrow head showing iliopectus abscess.

Discussion

BCG is a live strain of *Mycobacterium bovis* developed by Calmette and Guerin for use as an attenuated vaccine to prevent tuberculosis and other mycobacterial infections [1]. Intravesical BCG is approved by the United States Food and Drug Administration for treatment of carcinoma *in situ*, high grade T1, or high risk Ta bladder cancer. It reduces the risk of recurrence and maintenance therapy reduces the risk of progression in patients with high grade non-muscle invasive bladder cancer [2].

The reported toxicities associated with intravesical BCG therapy are extensive and variable. Some of the more common toxicities include fever, malaise, hematuria, and cystitis. Rash, ureteral obstruction, contracted bladder, and cytopenia have been reported [3]. More severe complications have been reported: vascular aneurysms and infections such as prostatitis, orchiepididymitis, balanitis, osteomyelitis, mycobacterial pneumonias, hepatitis, nephritis, disseminated BCG, and abscesses [4,5].

The pathogenesis of vascular aneurysms and abscesses associated with BCG is not fully understood. Direct intimal colonization from hematogenous spread, metastatic implantation through the vasa-vasorum or local vascular extension from an adjacent infectious site have been proposed [6]. Disruption of the urogenital mucosa at the time of bladder instillation, traumatic Foley catheter insertion, biopsy, surgery or active cystitis are the most important known risk factors for BCG infection [6,7]. Psoas abscesses have been reported to occur nearly exclusively in patients with infrarenal aneurysms [8]. Vascular aneurysms, as with other complications of intravesical BCG therapy, may manifest long after treatment has been completed. Cases diagnosed between four months and over five years after the completion of treatment have been reported [9,10].

Conclusions

Our patient had a stable AAA for many years but it rapidly increased in size following BCG therapy. The patient's AAA was

first noted in 2007 at 4.0 cm. It increased to 4.9 cm in 2012 before he received BCG therapy. It was measured at 8.5 cm when he presented with psoas abscess three years after he first received BCG therapy. Radiologically, this was highly suggestive of mycotic aneurysm given the rapid increase in size of the AAA [11]. However, *Mycobacterium bovis* was never isolated from the blood. The development of the psoas abscess and the rapid expansion of the AAA were most likely from the BCG therapy. The pathogenesis of this is largely speculative at this time. However, until the exact cause and effect relationship of BCG therapy and the development of infectious complications are identified, the risk-benefit assessment of this treatment should be carefully considered, especially in these patients with pre-existing vascular aneurysms.

Conflict of interest

The authors declare no conflict of interest.

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