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Pott's disease post intravesical BCG

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ABSTRACT

This report describes a unique case of an 89-year-old-male who presented with a right psoas abscess and new erosive changes in the L3/L4 vertebral bodies. This is on a background of non muscle invasive bladder cancer (NMIBC) for which he received intravesical Bacillus Calmette-Guerin (BCG). Pott's disease is a rare complication of BCG therapy and there is paucity of data about the risk factors for systemic spread of intravesical BCG.

1. Introduction

Pott's disease is defined as tuberculosis (TB) of the spine. Bacillus Calmette-Guerin (BCG) is used in the treatment of bladder cancer and are attenuated strains of Mycobacterium bovis, which is part of the *Mycobacterium tuberculosis* complex.

2. Case report

An 89- year old man was diagnosed with high grade non muscle invasive bladder cancer (NMIBC) in 2017. The patient had pathological findings of high-grade urothelial carcinoma (HgTa) with micropapillary differentiation. He subsequently underwent a transurethral resection of bladder tumour (TURB-T) demonstrating residual disease of HgTa and carcinoma in situ (CIS). The patient was commenced on induction and maintenance intravesical Bacillus Calmette-Guerin (BCG) therapy, with cessation 12 months later due to poor tolerance in 2018. During his follow up in October 2020, 2 years post BCG therapy, his surveillance cystoscopy was clear however computed tomography (CT) scan of his abdomen and pelvis demonstrated a 34 × 53 × 73mm right psoas muscle collection extending anteromedially involving the infrarenal aorta (Fig. 1). There was also evidence of new erosive changes of the L4 vertebral body consistent with osteomyelitis (OM).

The patient was subsequently referred to ED. He was afebrile and had no neurological findings on physical examination. He had a normal full blood count and electrolytes, except for a mildly elevated c-reactive protein (CRP) of 51. Magnetic resonance imaging (MRI) of his cervical, thoracic and lumbar spine further demonstrated cortical destruction of L3/L4 but no cord impingement. The patient was commenced on intravenous ceftriaxone and flucloxacillin as per infectious disease (ID) advice for treatment of OM. A CT guided drain was inserted into the multiloculated right psoas abscess and fluid was sent for microscopy and culture. The fluid from the aspirate confirmed a positive polymerase chain reaction (PCR) for mycobacterium. There were no other organisms that grew in culture.

The orthopaedic spine team was consulted, however given that there was no evidence of cord or root compression they advised that there was no indication for invasive management. Pending susceptibility results, isoniazid, rifampicin, pyrazinamide, ethambutol and pyridoxine was commenced, all once daily for 2 months of induction therapy. Microbiology team later confirmed the growth of mycobacterium bovis BCG using genomic deletion analysis of the fluid aspirate. As a result, pyrazinamide was discontinued, as it is resistant to that organism.

The hospital course was prolonged because of immobility and delirium. Repeat CT brain and CT lumbar spine were unremarkable for new lesions. The patient was eventually discharged and completed 12 months of isoniazid, rifampicin and pyridoxine. His CTAP showed complete resolution of the right psoas muscle collection and he remains well with no further symptoms.

3. Discussion

Pott's disease is a vertebral infection caused by mycobacterium tuberculosis. In the literature, only 24 cases of tuberculous spondylitis, vertebral osteomyelitis and/or Pott's disease following intravesical BCG were reported.[1] In most of the reported cases, presentation of disease occurred soon after intravesical BCG therapy but has been reported up to 12 years later. Management of all reported cases was with completion of 12-month course of isoniazid, rifampicin and pyridoxine, after an initial two months of ethambutol, in addition to the other two antituberculosis

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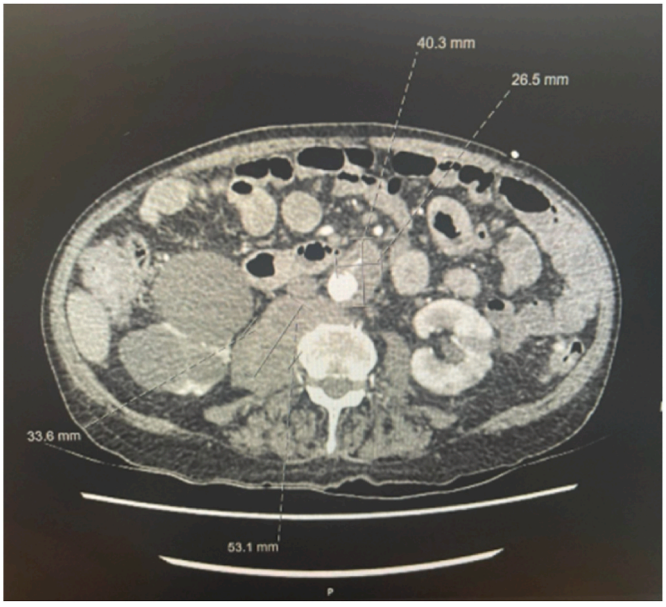

Fig. 1. Right psoas muscle collection extending anteromedially involving the infrarenal aorta.

drugs. There is a paucity of data given the rare nature of the disease and therefore long-term outcomes of such patients in unknown.

BCG is a live attenuated strain of Mycobacterium bovis and has strong evidence for use as intravesical therapy for high grade NMIBC. Although its exact mechanism is not fully understood, it is believed to induce an immune response that eradicates bladder cancer through anti-BCG-induced cell-mediated immunity.[2] Its effects are generally localized to the bladder surface, suggesting that it is the local immune response and the inherent antineoplastic properties of the BCG itself that are primarily responsible for its activity.[2]

Local complications post BCG account for 62.8% of cases with BCG-induced cystitis being the most common.[3] Frequency and dysuria, macroscopic hematuria, bladder contracture, ureteral obstruction, prostatitis and epididymo-orchitis can all occur. Rarely, serious systemic complications can arise in the form of sepsis, pneumonitis, granulomatous hepatitis, reactive arthritis and even death from disseminated BCG in less than 5% of cases.[3] These complications may be classified as early or late depending on the timing from intravesical instillation. Early complications present within 3 months while late complications occur after 3 months. Interestingly, our patient presented with disseminated BCG 2 years following his last intravesical BCG therapy. Late presentations may result from reactivation of mycobacteria after successful control of early dissemination.

The risk of BCG sepsis increases in the presence of traumatic catheterization, macroscopic hematuria, urinary tract infection, instillation within 30 days of TURBT, and concomitant use of immunosuppressive therapy.[4] The majority of these factors represent a breach of the urothelial barrier enhancing hematogenous spread and dissemination of the Mycobacteria.

The role of prophylactic antituberculosis therapy accompanying intravesical BCG therapy is debated. Rawls et al. recommended 3 days of prophylactic isoniazid therapy beginning the morning before treatment.[5] However, Fishman et al. reported BCG spondylitis even with prophylactic isoniazid coverage.[5] A larger prospective study is needed to assess whether prophylactic agents reduce intravesical BCG therapy complications.

4. Conclusion

Pott's disease is a rare complication of intravesical BCG therapy. Further understanding of the risk factors predisposing patients to disseminated BCG following therapy for bladder cancer may help optimize patient selection for this treatment and inform its optimal use.

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