Case Report

Two-stage posterior decompression and fusion for tuberculous spondylitis after intravesical bacillus Calmette-Guerin instillation

ABSTRACT

Intravesical bacillus Calmette-Guerin (BCG) instillation is an effective treatment for nonmuscle invasive superficial bladder cancer. BCG induces a massive influx of inflammatory cells and production of cytokines in the bladder mucosa and lumen that leads to an immune response against tumor cells, acting as an immunotherapy. Cystitis, bladder ulceration, and bladder contracture are known local genitourinary complications, whereas systemic complications such as miliary pulmonary tuberculosis, mycotic aneurysms, tuberculous spondylodiscitis, and granulomatous hepatitis are very rare. A case of tuberculous spondylodiscitis at the T8 and T9 levels following intravesical BCG instillation for bladder carcinoma is reported. The patient initially underwent decompressive laminectomy for spastic paraparesis. After reporting improvement for few weeks, the patient again presented with similar complaints and was found to have an increased kyphotic deformity, for which he underwent fixation surgery.

Keywords: Intravesical bacillus Calmette-Guerin, pott's spine, tuberculous spondylodiscitis

INTRODUCTION

Intravesical bacillus Calmette-Guerin (BCG) instillation is an effective treatment for nonmuscle invasive superficial bladder cancer. Cystitis, bladder ulceration, and bladder contracture are known local genitourinary complications, whereas systemic complications such as miliary pulmonary tuberculosis (TB), mycotic aneurysms, tuberculous spondylodiscitis, and granulomatous hepatitis are very rare.[1]

A case of tuberculous spondylodiscitis at the T8 and T9 levels following intravesical BCG instillation for bladder carcinoma is reported. The patient initially underwent decompressive laminectomy for spastic paraparesis. After few weeks, the patient again presented with similar complaints and was found to have an increased kyphotic deformity, for which he underwent fixation surgery.

CASE REPORT

A 79-year-old male presented with a 2-month history of progressive mid-back pain and mild motor weakness of both lower limbs. His past medical history included transurethral resection of bladder cancer followed by intravesical BCG instillation 5 years earlier. He had tenderness on palpation and percussion over the left side of the iliac bone. A neurological examination showed severe impairment of deep sensation and hypalgesia up to the level of T9. Initial routine laboratory

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examination showed a white blood cell (WBC) count of 4500/μL, a C-reactive protein (CRP) of 0.04 mg/dL, and an erythrocyte sedimentation rate (ESR) of 13 mm/1st h. The QuantiFERON-TB test result was negative.

Plain radiographs showed T8 and T9 compression fractures. A chest X-ray was unremarkable. A computed tomography (CT) scan showed destruction of the anterior and mid T8 and T9 vertebral bodies [Figure 1a]. Magnetic resonance imaging (MRI) showed a collapsed T8 and T9 disc space with a T1 hypointense and T2 hyperintense, nonenhancing paraspinal soft-tissue collection causing cord compression at the same level [Figure 1b and c]. Fluorodeoxyglucose (FDG) positron emission tomography-CT showed high FDG uptake in the T8 and T9 vertebrae, and an epidural soft-tissue mass lesion surrounding the T8 and T9 vertebrae.

Clinical course

A diagnosis of T8 and T9 spondylodiscitis was made, with differential diagnoses of pyogenic spondylodiscitis, Pott's spine, and metastatic tumor compression fracture. He underwent T8 and T9 decompressive laminectomy and decompression of the soft-tissue component compressing the cord. A specimen was sent for histopathological and culture sensitivity analysis.

Postoperatively, he was relieved of mid-back pain, the motor weakness was improved, and he was able to walk with the help of crutches. Impairment of deep sensation persisted.

Six weeks later, *Mycobacterium tuberculosis* was detected from the cultured specimen. The patient was started on antitubercular treatment with isoniazid, rifampin, ethambutol, and pyrazinamide.[2] At 3-month follow-up, CT and MRI showed the same size of bony destruction with no change in the thoracic kyphosis and the T8 and T9 epidural soft-tissue lesion [Figure 2a and b]. The laboratory examination showed a WBC count of 3900/μL, a CRP of 0.49 mg/dL and a persistent high ESR of 30 mm/1st h. He had mild motor weakness of both lower limbs and was able to walk without support. Deep sensory impairment persisted. Antitubercular treatment was continued for 6 months.

Six months postoperatively, he presented with recurrence of mid-back pain and moderate paraplegia. The laboratory examination showed a WBC count of 3500/μL, a CRP of 0.14 mg/dL, and an ESR of 28 mm/1st h. MRI showed an increase in the size of the T8 and T9 epidural soft-tissue mass compressing the dural sac [Figure 2c]. He underwent laminectomy of T6–7 and T10–11, removal of scar fibrous tissue around T8 and T9, and bilateral pedicle screws and rod fixation on T5–7 and T10–11.

Histopathological findings showed only chronic inflammatory changes. There were no typical findings of TB spondylitis, such as caseous necrosis, ruling out that diagnosis [Figure 3a and b].

Two months after the second surgery, he reported improvement in back pain and motor weakness, and he was able to walk with crutches with slight sensory improvement. Six months after the second surgery, CT showed no further progression of the kyphotic deformity. Bony fusion developed, with remodeling of the vertebral body. MRI showed that the paravertebral abscess was resolving and decreasing in size [Figure 4a-b]. The antitubercular treatment was completed at 6 months according to the guidelines.[2]

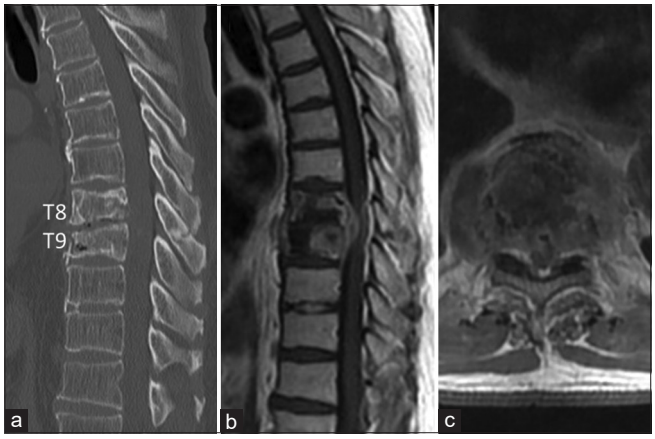

Figure 1: Preoperative images. (a) Sagittal computed tomography shows T8–9 anterior vertebral body destruction. (b and c) Sagittal and axial images (T1-weighted with contrast) showing marrow edema, collapsed T8–9 disc, and soft-tissue mass lesion enhancement with anterior dural sac compression

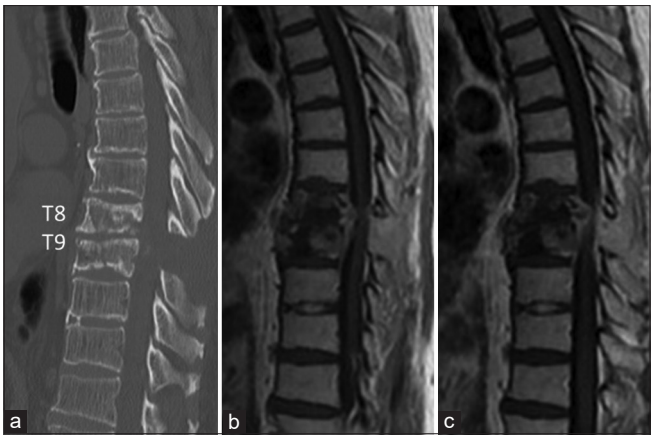

Figure 2: After first surgery, postoperative follow-up images. (a) Follow-up computed tomography scan 3 months later showing the same size of bony destruction with no change in thoracic kyphosis. (b) Three-month follow-up sagittal T1-weighted magnetic resonance imaging with contrast, showing the same size of paravertebral abscess. (c) Six months later, sagittal T1-weighted magnetic resonance imaging with contrast shows increased size of the paravertebral abscess with dural compression and increased kyphosis at the same level

In this case, after 6 months of antitubercular treatment, the laboratory examination showed a WBC count of 4500/μL, a CRP of 0.57 mg/dL, and a persistent high ESR of 18 mm/1st h.

DISCUSSION

A live-attenuated strain of *Mycobacterium bovis* is used in BCG vaccine.[3] Dr. Morales used intravesical BCG for the first time to treat, control residual tumor, and prevent recurrence and disease progression of nonmuscle invasive superficial transitional bladder carcinoma.[4] Although the local genitourinary complications are more common, cases of TB spondylodiscitis after intravesical BCG instillation were rarely reported by departments of neurosurgery.[1]

Thoracic vertebrae are affected most commonly, as in the present case. The hematogenous route through the Batson venous plexus is the most common route of spread to thoracic vertebrae.[5]

A high index of suspicion is required to diagnose Pott's spine secondary to intravesicular BCG therapy, especially in an area where the prevalence of Pott's spine is very low.

CT scan-guided biopsy is required to confirm the diagnosis in patients with mild symptoms such as only mid-back pain, weight loss, or night sweats. Patients with clinical features of compressive myelopathy may require initial decompressive surgery along with instrumentation.

Empirical antitubercular treatment is required as per the guidelines in all patients. Regular follow-up with MRI to follow the size of soft-tissue mass lesions, spinal alignment, and fusion is necessary.

Radiological improvement in the form of regression of a paravertebral abscess, the resolution of marrow edema, fatty replacement of marrow, improvement in vertebral lucencies, and endplate fusion may lag behind the clinical signs of response such as the absence of fever, weight gain, and pain relief to antitubercular medications. Follow-up MRI may show flaring up of the lesion though the patient shows signs of clinical improvement with continuing antitubercular therapy. Thus, MRI should be assessed very carefully, especially during the initial months of follow-up.[6,7] Posterior instrumentation with debridement of a paravertebral abscess leads to higher fusion rates and early healing. Wang *et al*.,[8] Lee *et al*.,[9] and Broner *et al*.[10] also reported that instrumentation leads to the suppression of infection, since mycobacteria have less affinity to adhering to titanium biomaterials having an active oxide surface, which provides a stable environment for healing and enabling neurological recovery. The most important factor to prevent neurological deficits is to provide early stabilization with decompression and fusion. Only decompression without stabilization, especially in Pott's spine with more than one level involved, may lead to progression of disease and a kyphotic deformity in future, which may lead to an additional neurological deficit after a short period of improvement following decompressive surgery alone, as in the present case. Short segment fixation should be avoided when only a posterior approach is used. Fixation of at least two spinal segments adjacent to the lesion is required to provide stability and prevent the progression of deformity.[9,10]

CONCLUSION

Tuberculous spondylodiscitis after intravesical BCG instillation is rare. A high degree of suspicion is required to diagnose it early. The standard antitubercular treatment according to the culture report is advisable at the earliest

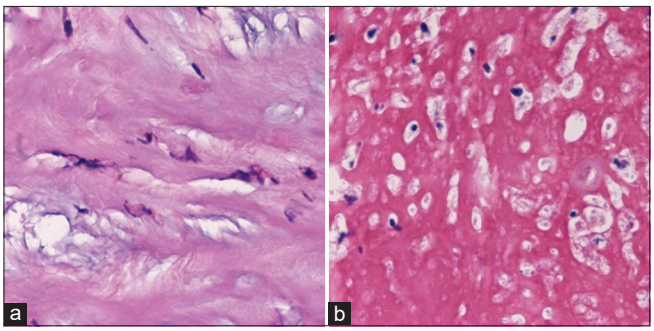

Figure 3: Histology. Histological findings of the vertebral body (a) and intervertebral disc (b) with hematoxylin eosin stain, ×40. Macrophage and lymphocyte infiltration is seen, suggesting chronic inflammatory disease. These findings do not show caseous necrosis, which is common in tuberculosis

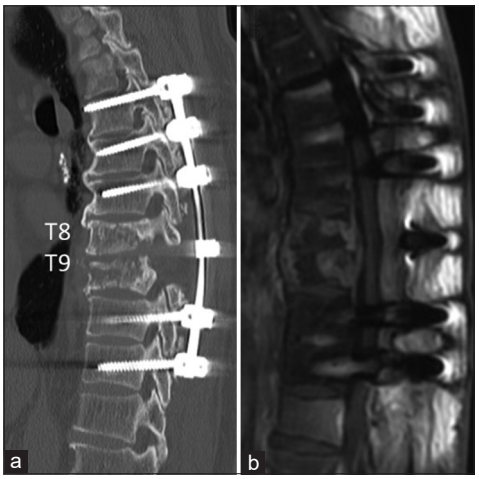

Figure 4: After the second surgery, postoperative follow-up images. Two months after the second surgery, postoperative images: (a) Sagittal computed tomography shows no further progression of kyphotic deformity. (b) T1-weighted contrast magnetic resonance imaging shows decreased size of the paravertebral abscess

possible. Patients showing signs of compressive myelopathy require decompression, debridement, and deformity correction requiring multiple level fixation with emphasis on the correction of sagittal balance.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient (s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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