Late-onset post-diskectomy tuberculosis at the same operated lumbar level: case report and review of literature

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Abstract Late post-diskectomy tuberculous spondylodiskitis at the operated level is an extremely rare phenomenon. We describe a unique case of a same level tuberculous spondylodiskitis with pus drainage from the operative scar 8 years after simple L4/L5 diskectomy and review the relevant literature. The therapeutic management and possible pathogenic mechanisms are discussed as well.

Keywords Tuberculosis · Diskectomy · Lumbar · Spondylodiskitis · Post-diskectomy

Introduction

Tuberculosis is a disease known in developed countries, but the problem is more serious in developing countries. Despite ongoing advancements in imaging techniques and introduction of new generation of anti-tuberculosis regimens, tuberculosis remains an international challenge [25].

The incidence of tuberculosis has been increasing worldwide [31]. Immigration from developing countries and HIV infection are the two main reasons for its

prevalence in developed countries [11, 12]. MDR-TB is also of great concern because of the increased age of the population and increased number of health-care professionals involved in the treatment [25]. Other possible factors for increased incidence include: increase in diagnosis [29], unemployment and hygiene problems [39].

The incidence of spinal TB appears to be increasing as well [2, 8]. Spinal TB constitutes 60% of bone and joint tuberculosis [1, 18]. The spinal column is affected in 1% of all TB patients [1, 12, 40]; however, Pott's disease (or spinal column TB) is the most dangerous of all [40]. In spite of the availability of diagnostic modalities, surgical techniques and effective anti-tuberculosis regimens, spinal TB is still a life-threatening disease [40], which can cause bony destruction, deformity and neurological deficits [39].

Late post-diskectomy infections are rare and have been reported to occur as long as 7 years postoperatively [36]. Malnutrition, diabetes, smoking, immunocompromised states, obesity, alcohol abuse [13] and instrumentation [36] might be related to the development of postoperative diskitis. *Staphylococcus aureus* is the leading cause in most of the cases [13, 36] and Gram-negative organisms are the next [36]. In recent years, immunocompromised states and the use of broad-spectrum antibiotics have led to an increase in the infection rate with unusual organisms including fungi and mycobacterium species [36].

The key to diagnosing tuberculosis is by considering that since TB is a multifaceted disease, definite diagnosis emerges from strong clinical suspicion. Although some typical radiological findings may exist, TB may be an excellent imitator and no single pathognomonic finding could differentiate it from other pathologies [21].

An exceptional case of late post-diskectomy TB spondylodiskitis at the operated level, 8 years after

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surgery, is presented and diagnostic and therapeutic interventions, as well as the possible pathogenic mechanism, are discussed.

Case report

History and physical exam

A 43-year-old male patient presented with chronic low back pain since the past 4 months and intermittent pus drainage from his operative scar in the lumbar region. The patient had lost weight (5 kg) over this period. He had a history of lumbar discectomy 8 years ago, which had led to a significant improvement postoperatively. Past medical history was negative for tuberculosis or other immunocompromised states; however, he had worked as a secretary in a local hospital for years.

On physical examination, no fever was detected. Odorless creamy pus drainage after compression of operative scar was evident. Other examinations including neurological examinations were all normal.

Radiological findings

Simple X-ray showed decreased L4–L5 intervertebral space and destructive changes in the surrounding end plates. MRI was performed with and without gadolinium contrast, which confirmed the destructive changes in the L4–L5 disk space and the neighboring end plates in favor of an infective process. The presence of a paravertebral abscess was also strongly suggestive for infection (Fig. 1).

Laboratory examinations

Increased ESR (80), CRP (3+) and mild chronic inflammatory anemia were found. Unfortunately, because of economical reasons, quantitative methods for CRP values are not generally performed in our country. Instead, the semi-quantitative method is used, in which CRP values in mg/l can be calculated by multiplying the dilution factor (2 for 1+, 4 for 2+, 8 for 3+ and 16 for 4+) by the detection limit (6 in our CRP kit). For our patient, 3+ was equal to 48 mg/l.

Fig. 1 a, b T1W axial MRI without and with gadolinium contrast showing the presence of an abscess in the left paravertebral space. c, d Coronal and sagittal T1W MRI depicting the simultaneous involvement of L4–L5 disk space and surrounding end plates suggestive of an infective process

Simple smear and culture from pus were negative after 48 h. PPD became positive (17 mm) after 48 h. All other investigations including biochemistry were within normal range.

Chronic inflammatory diseases such as fungal infections, spinal brucellosis or tuberculosis were in the differential diagnoses; however, the patient denied any history of such diseases or close contact with a tuberculosis patients in the past.

Intervention

For diagnostic purposes, fluoroscopy-guided percutaneous needle biopsy from L4 and L5 vertebrae was performed under general anesthesia. After ensuring the right placement of guide catheters through pedicles into the body of L4 and L5, biopsy specimens were taken.

Acid-fast bacilli were evident in the smear of the samples. Histopathology confirmed the presence of epithelioid granuloma with caseous necrosis, and epithelioid and multinuclear giant cells (Fig. 2). The culture were positive for *Mycobacterium tuberculosis* after 6 weeks in a Lowenstein–Jensen environment. Thorough investigation for possible urinary tract co-infection was negative.

Based on negative results for possible bacterial infection and above-mentioned findings, the patient received a four-drug anti-tuberculosis regimen including isoniazid (INH), rifampin (RMP), ethambutol (ETH) and pyrazinamide (PZA) for 2 months. The regimen was restricted to INH and RMP for the next 10 months. During the 1st month of treatment, the general condition of the patient improved significantly and he gained his appetite and weight. Pus drainage diminished gradually and by the end of the 2nd

month of treatment it was totally abolished. MRI obtained at this time showed changes indicative of drug response (Fig. 3). ESR returned to normal by the end of the 3rd month. The patient returned to his job during the 5th month and spent an uneventful course of treatment for the next 8 months. One-year postoperative MRI revealed significant changes in favor of treatment without the development of deformity or restenosis (Fig. 3).

To report on this unique case, the patient's consent was taken to anonymously publish his data and imaging.

Discussion

Postoperative intervertebral diskitis might be seen in 0.2–10% of all discectomies [38]. Most reports concur that the average incidence is 1% after simple discectomy [13]; however, this figure seems to be underestimated. One probable cause may be self-limited infections in most of the cases, which remain undetected. The other cause might be the sole application of simple X-ray and tomography in most series to confirm the diagnosis, while these modalities are less sensitive than MRI [4].

Late post-discectomy infections are rare and have been reported to occur as long as 7 years postoperatively [36]. Some factors such as malnutrition, diabetes, smoking, immunocompromised states, obesity, alcohol abuse [13] and instrumentation [36] may precipitate postoperative diskitis. Responsible pathogens include bacteria and fungi [36]. *Staphylococcus Aureus* is the leading cause (60%) in most of the cases [13, 36] and Gram-negative organisms are the next [36]. In recent years, the administration of broad-spectrum antibiotics and increased number of immunocompromised patients has led to an increase in infection rate with unusual organisms [36].

Rare cases of postoperative tuberculous after procedures in other areas of the body, including hip arthroplasty and coronary artery bypass surgery have also been described [9, 37].

A complete search in Medline and Google search engine for late postdiscectomy tuberculosis at the operated level revealed two similar cases in literature [16, 19]. The first case [19], a 31-year-old male patient with a history of partial hemilaminectomies of L4 and L5 and L4/L5 discectomy presented with fever and inflamed operative scar 2 months after surgery. The operative site was opened and multiple abscess pockets were evacuated. The incision was left open and irrigated daily. Simultaneously, intravenous chloromycetin was administered to cover staphylococcus, because gram stain and cultures were negative for tuberculosis and fungi. The result was disappointing and the patient underwent two other operations in the next 2 weeks and a change in antibiotic regimen, with no complete

Fig. 2 Photomicrograph of the biopsied specimen showing accumulation of mononuclear and multinuclear inflammatory cells and also epithelioid, giant multinuclear cells, which encompass a caseous necrotizing granuloma

Fig. 3 T1W axial and sagittal MRI obtained 2 months (a, b) and 1 year (c, d) after the initialization of anti-tuberculosis regimen delineates the resolution of paravertebral abscess and increased signal intensity of vertebral body and vertebral end plates in favor of successful treatment. No sign of deformity or restenosis is evident

improvement. The patient was treated with a three-drug antituberculosis regimen after the culture became positive in 4 weeks. The response was complete in 5 months and the regimen was discontinued by the patient.

The second case [16] had a very complex course because tuberculosis was not considered as a possible cause, which led to delay in diagnosis and multiple invasive procedures. This patient underwent L4–L5 discectomy and intervertebral fusion. The patient remained asymptomatic until 3 months after surgery and then complained of fever and low back pain. MRI was normal and the patient received antibiotic therapy. After 3.5 months of therapy, the patient had a normal MRI, but was still in pain. Multiple facet blocks were performed and vertebroplasty was done for three levels. Six months later, spondylodiskitis appeared on MRI and needle biopsy revealed no bacteria. The patient received antibiotics for the next 5 months. The second biopsy was positive for bacteria, but negative for tuberculosis. Due to antibiogram, antibiotics were administered for 6 months, but had no effect on symptoms. Finally, surgical debridement and instrument removal were performed. Polymerase chain reaction was positive for

tuberculosis and the patient was treated with appropriate anti-tuberculosis regimen.

The spinal column may be affected by tuberculosis. The most common site of involvement is the thoracic region [11, 25, 40] and mostly the vertebral body is affected [1, 18]. Lower lumbar and lumbosacral junctions are rare sites of involvement [31]. Any delay in diagnosis and appropriate therapeutic intervention contribute to neurological deficits, spinal column deformity and resultant decreased recovery [40].

The source of infection in cases of spinal involvement is usually outside the spinal column [1]; however, it remains undetected in most of the cases [3]. The disease usually spreads hematogenously from indolent foci in the lungs [3]. Some authors [14, 21] believe that the spinal column is involved after lymphatic spread from the primary foci of infection; which remain mostly indolent. Others believe that TB spreads through the vertebral venous system, as described by Batson [40], namely the Batson venous plexus. Numerous anastomoses and lack of valves in this plexus might be the main explanation. Mulleman et al. [24] argued that the reactivation of indolent foci is the main

mechanism of spinal column involvement. Three mechanisms of spinal involvement were suggested for our patient. First, we hypothesized that it might be secondary to hematogenous spread from a primary focus such as the lung. After entering the lungs, the bacilli infiltrate lymph nodes and cause a low level of bacteremia. This leads to the spread of bacilli into remote tissues including paravertebral lymph nodes. If the disease is controlled at this stage, the bacilli remain latent in these areas [35]. The possibility was considered high remembering the occupation of the patient as a secretary in a local hospital; however, CXR revealed no abnormality in favor of tuberculosis. Enhanced chest CT scan is the imaging of choice in evaluating inactive pulmonary foci; however, we cannot totally exclude the possibility of activated pulmonary foci, because this was not requested for our patient. Moreover, we were in an endemic area for tuberculosis and these foci might be seen incidentally in CXR and chest CT scan.

Second, we thought of the possibility of spread from adjacent involved urinary tracts. This was also ruled out with a thorough investigation including urine smear and culture, KUB, IVP and contrast-enhanced CT scan. Enhanced abdominal and pelvis CT scan is the best imaging modality to show lymph node involvement.

Third, we thought of the possibility of instrumental infection during the first surgery and the resultant paravertebral lymph node involvement. This may have been indolent for years, until a flare-up due to decreased immunity. HIV infection was suspected as a cause, but the serology was negative.

Rajini et al. [32] reported the transfer of a fast growing mycobacterium, *Mycobacterium chelonae*, to their patient through laparoscopy. Although direct transfer of *Mycobacterium tuberculosis* seems to be unusual, this needs further investigation. A high suspicion of tuberculosis, polymerase chain reaction and appropriate culture for TB might be clues for similar cases. Although we are unable to document other sources of decreased innate immunity, the role of the latter mechanism seems to be stronger in our patient.

Gradual onset of the disease makes the diagnosis difficult [25]. Various clinical presentations of TB spondylodiskitis have been described [2]. Depending on the site of involvement, age at diagnosis and the number of vertebrae involved, the disease may appear as a simple back pain or severe deformity of the vertebral column [21]. In a study by Nas et al. [25], the clearest symptoms included focal pain, fever, paraparesis and sensory loss.

Pain is the main symptom in spinal TB [2, 24]. Fever and systemic symptoms may not be present until the late stages of musculoskeletal TB [2]. Hence, the diagnosis is often difficult and long-standing symptoms may persist before a definite diagnosis [14].

Spinal cord compression may happen due to pus or abscess formation during the acute stage of TB. Delayed cord compression is also possible due to fibrosis, caseous materials, increased kyphotic deformity (gibbous) and, uncommonly, reactivation of tuberculosis [21].

Leukocyte count has a limited value in diagnosis and may be within normal range or slightly increased [24]. ESR is a useful guide to assess therapeutic response in spondylitis [12]; however, it may remain unchanged even in the presence of an abscess [19]. In most patients, ESR decreases to half the value by the end of antibiotic therapy [12].

CRP is a more constant laboratory finding than ESR and decreases earlier than ESR after initiation of antibiotics [12]. In general, it is more important than ESR in the early diagnosis and follow-up of patients with spinal infection.

Skin tuberculin test (PPD) has lost its specificity especially in endemic areas, where BCG vaccination is routine, and in areas where subclinical exposure is common to tuberculosis [21].

Simple X-rays might show decreased intervertebral disk space and end plate reaction, bony destruction [30], increased paravertebral soft tissue and abscess formation [17], scalloping of the anterior vertebral body [7] and calcification within the abscess [33]. In the cervical and upper thoracic spine, increased paravertebral soft tissue might increase the space between the vertebral body and the trachea [15]. These changes along with relevant clinical findings suggest further investigation for the diagnosis. Some authors believe that MRI and CT help in the diagnosis of TB spondylitis [10, 40]. Although these have facilitated diagnosis to a great extent [6, 28], biopsy is the preferred diagnostic modality [28]. MRI is the best imaging method [2, 24], and shows typical diskitis, as well as epidural and paraspinal abscesses, better than any other method [24]. Administration of gadolinium increases the resolution and helps to distinguish an infective process from degenerative changes in end plates and intervertebral disks [36].

Because various possible responsible organisms make detection of a specific organism and appropriate antibiotic initiation critical for a successful treatment, histological diagnosis is necessary [22]. Endoscopic methods, CT-guided biopsy or fluoroscopy-guided biopsy help a lot to make a definite diagnosis; however, one should bear in mind that because of low bacterial load in spinal TB, the possibility of detecting mycobacterium may be less than 50% [21]. Despite indefinite biopsy results in these cases, some authors suggest the onset of treatment in the presence of high clinical and radiological suspicion [26].

Considering these limitations, polymerase chain reaction has been recently suggested as an accurate diagnostic tool. Its sensitivity, specificity, and positive and negative predictive

values are 94.7, 83.3, 94.7 and 83.3%, respectively [6, 27, 34, 41].

The main treatment of TB spondylitis is with medication [8]. Spinal TB can be treated conservatively with medication and spinal column immobility during the first stages of infection and in patients without neural compression [17]. In most cases, TB abscesses have been completely cured with medical therapy [23].

After the onset of treatment, inflammatory symptoms should subside gradually and can be monitored by laboratory examinations. Clinically, this depends on the patient's general condition, neurological status and response to treatment. The inability to achieve this level is regarded as treatment failure or the possibility of complications [25].

Based on the WHO protocol, all cases of spinal TB should be treated medically for at least 6 months; on the other hand, CDC (centers for disease control and prevention) suggest that osteoarticular TB, including TB spondylitis, should be treated at least for 12 months [32]. In our case, we preferred to follow the CDC guidelines.

There is no accepted protocol for surgery; however, it may be indicated for abscess drainage, neurological deficits, biopsy specimens, drug resistance, spinal instability [5, 20], progressive vertebral destruction and deformity [8, 23]. Surgery is necessary for progressive paraplegia [33]. It should be emphasized that in all patients undergoing a surgical procedure, complete medical therapy should also be considered [17, 33].

Conclusion

Although extremely rare, tuberculosis should be included in the differential diagnosis of post-discectomy diskitis. Lymphatic or hematogenous spread from indolent foci or reactivation of TB in immunocompromised patients may be the possible explanations for this exceptional phenomenon. This case provides evidence in favor of the fact that surgical intervention is not always the primary acute treatment option. Obviously, acute neural compression with deficit would be an exception, as would late stenosis from scar formation or deformity. This case is also unique in that the patient developed infection at the site of prior surgery 8 years later without any other areas of potential infective sources throughout the body. Thus, we hypothesize that he may have had latent organisms from the time of surgery that in combination with some immunocompromise led to his late infection.

Conflict of interest statement None of the authors has any potential conflict of interest.

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