Pott's Disease in Children: a Case Report and Review of Current Practices

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Abstract

Spinal tuberculosis, also called Pott's disease, is a prevalent disease in low- and middle-income countries but can also be seen in Europe. Back pain is the main presenting complaint, and the majority of patients do not experience systemic symptoms. Complications include spinal deformation and medullar compression with associated neurological symptoms. In many cases there is a significant delay between the appearance of symptoms and diagnosis, which may make the outcome worse and underlines the importance of a good understanding of this pathology. Treatment includes tuberculostatic therapy as well as surgery in selected cases. We describe the case of a 15 year old girl presenting with respiratory complaints and spinal tuberculosis, and we review and discuss available knowledge about this important disease.

Introduction

First described by Sir Percival Pott in 1779, spinal tuberculosis, also called Pott's disease, represents half of musculoskeletal tuberculosis, which represents 10% of extrapulmonary tuberculosis, and 1% of all tuberculosis cases (1, 2). Around 1300 children were affected by tuberculosis in 30 countries of the European Union and European Economic Area countries in 2022 according to the last Surveillance Report of the World Health Organization (3).

Pott's disease most frequently involves the thoracic vertebrae and is multi-focal in 51% of cases (of which 8% involving adjacent vertebrae) (1, 4, 5). In 90-95% of cases, spinal tuberculosis involves the anterior part of the vertebral body (2).

The management of spinal tuberculosis in children is subject to debate. This report describes a 15-year old girl who was admitted to the hospital with chest pain and dyspnoea. She was diagnosed with pneumonia, pleural effusion and thoracic Pott's disease. We aim to discuss current knowledge and review available guidelines for this important disease.

Clinical case

A previously well 15 year old girl of Congolese origin and living in Mayotte for 2 years presented with a 4 month history of lateral thoracic pain and exertional dyspnoea. There was no fever and her general condition was good. Her vaccination status was unknown. There were no known tuberculosis (TB) contacts. On clinical examination, she had a dry cough and decreased breath sounds at the right lung base.

The blood tests performed showed mild inflammation (CRP 30 mg/L [N < 5.0 mg/L], with normal leucocyte count) and a microcytic anaemia (Hb 11.6 g/dl [N 12-16 g/dL], mean corpuscular volume 76.8 fL [N 78-100 fL]). HIV serology was negative. The chest X-ray showed a right basal pneumonia with pleural effusion. There was no adenopathy on examination or chest X-ray. Pleural fluid examination showed inflammation with a lymphocytic predominance. Direct examination, culture and PCR for *Mycobacterium tuberculosis* were negative. The 3 gastric aspirates performed were also negative. Her tuberculin skin test showed an induration of 15 mm and the interferon-gamma release assay was also positive. A CT scan of the chest, abdomen and pelvis was performed, which showed a middle lobe consolidation and a significant right pleural effusion, as well as a small lytic lesion of the antero-superior corner of the T10 vertebral body, with an anterior abscess measuring 10 mm in antero-posterior diameter at the T9-T10 level. MRI confirmed a spondylodiscitis at T9-T10 level and a prevertebral abscess, and showed damage to the right sacroiliac joint, presumably from TB. There was no spinal cord compression (see Figure 1).

She was referred to a tertiary hospital, where an ultrasound-guided vertebral biopsy was performed, which revealed acid-fast bacilli on microscopy. The diagnosis of tuberculosis was confirmed by GeneXpert, with a strain susceptible to rifampicin. She was started on a standard four-drug oral regimen of isoniazid 5 mg/kg, rifampicin 12 mg/kg, pyrazinamide 30 mg/kg and ethambutol 20 mg/kg, plus pyridoxine. Gene sequencing confirmed a fully sensitive strain. The index case was not identified.

Despite good compliance, her condition worsened, with persistent back pain and an increase in abscess size and vertebral signal on T2 sequences on a subsequent MRI. A second biopsy was performed after 5 weeks of treatment, which showed the persistence of *M.tuberculosis*, which was still sensitive to rifampicin by GeneXpert. It was thought that her evolution could be explained by reduced diffusion of the drugs to the lesion. Immune reconstitution, which occurs during the first weeks of TB therapy, could also explain this paradoxical reaction and is a likely cause in the context of good adherence.

The pleuropulmonary disease resolved with treatment. An immunologic work-up showed no abnormality. The prevertebral abscess was drained by minimally invasive surgery. MRI after surgical drainage showed an almost complete drainage of the abscess, and a stability of the signal in vertebrae T9-T10 and of the anterior part of T11. The presacral collection was stable. The 4 drug combination therapy was continued for 7 months, followed by isoniazid and rifampicin for 1 month (total duration of 8 months). This long duration of 4 drug therapy was justified by concerns about initial treatment failure. She was asked to wear a rigid brace. The further evolution was satisfactory. 15 months after initiation of treatment, she occasionally complains of back pain due to discopathy, but her life has returned to normal.

Discussion

Pathology of Pott's disease

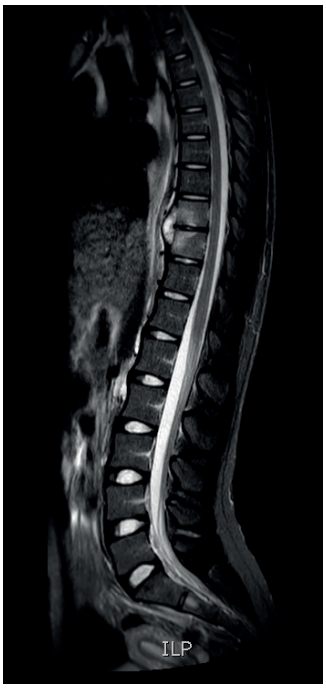
M.tuberculosis affecting the spine typically spreads by haematogenous route, facilitated by vertebral vascularisation. The primary focus is pulmonary in the majority of cases (4, 6). In the retrospective paediatric study performed by Benzagmout et al, a concomitant pulmonary infection was found in half of the cases (4). Other routes of dissemination include direct inoculation after a trauma or surgery and contiguous dissemination from adjacent tissues, but these are not commonly seen in children (6).

M.tuberculosis is deposited by terminal arterioles on the anterior part of the vertebral body, which is the usual initial site of infection (7). The common blood supply of adjacent vertebral bodies by segmental arteries explains multifocal disease (1). Infection then spreads to the central part and to the cortex of the vertebral body. Eventually it can disseminate below the anterior or posterior longitudinal ligaments, as well as through the periosteum, causing bulging of the vertebral surface and devascularisation of the periosteum (1, 4, 8). The formation of a cold abscess around the lesion is a characteristic feature of spinal tuberculosis (1).

Infection tends to disseminate subligamentary, but can also spread to the adjacent soft tissues and cause paravertebral or epidural abscesses, which can compress the spinal cord and cause neurological complications. Motor deficit is the first manifestation, as motor fibres are more sensitive to compression than sensory fibres, which are more sensitive to ischaemia (2). Collateral circulation prevents ischaemia of neural fibres (2).

Intervertebral disc lesions can occur in children, whose discs are still vascularised (1, 4, 7). Destruction of the disc and adjacent vertebrae leads to spinal deformity in the form of gibbus (hump-shaped deformity involving 2-3 vertebrae) and kyphosis (convex curvature of the spine when many vertebrae are involved) (9). A flat vertebra may be seen in cases of severe compression (1).

Figure 1: Initial MRI, T2 sequence. Hypersignal of the vertebral bodies T9-T10 and pre-vertebral abscess.



Clinical findings

The clinical picture includes back pain, systemic symptoms and symptoms due to complications. Some features are specific to certain localisations.

Back pain is the most common symptom and is present in 90-100% of the cases (8). Back pain at rest at the level of the lesion is characteristic (9). Its intensity varies with the level of bone destruction and spinal instability and is higher in thoracic lesions (9). Aggravating factors include movement, coughing and weight bearing. Axial pain is due to bone destruction, mass effect from abscesses, and spinal instability, whereas radicular pain is due to mass effect or vertebral collapse (7, 9). Back rigidity and muscle spasm may contribute to the pain (1).

Systemic symptoms occur in only 20-30% of cases and are more common in cases associated with pulmonary tuberculosis (1, 8, 9). They include asthenia, fatigue, fever (most often in the evening), sweating, loss of appetite and weight loss (4). They start insidiously, which explains the usually long diagnostic delay. In adults, Batirel et al found a median diagnostic delay of 78 days, while Khanna et al. found a median delay of 3 to 6 months (5, 7).

Complications include abscess in 69% of cases, neurological deficit in 40% of cases, and spinal deformity in 16% of cases (5).

Abscesses are cold, painless and grow slowly. Their subligamentous spread causes a mass effect with specific characteristics depending on their location.

Neurological deficit is caused by spinal cord compression and gives the related symptoms: radicular pain, motor deficits ranging from paresia to paraplegia (or even tetraplegia in the case of cervical spine lesion), sensory loss and continence disorders. Paraplegia may occur at any time and at any stage of the vertebral disease (1). The prognosis of early paraplegia is better than that of late paraplegia (1). Kumar et al. proposed a classification of paraplegia in spinal tuberculosis, reflecting spinal cord compression. Other classifications also exist (2).

Spinal deformity is particularly pronounced in children as their spine is very flexible, ossification is still in progress, and vertebral lesions cause impairment of anterior growth. Risk factors for spinal deformity include age less than 10 years, lesion of more than 3 vertebrae, and a thoracic location (4). Furthermore, a spinal deformity is less well tolerated when it affects the thoracic region (4, 9). Khanna et al. classify kyphosis reconstruction into three types according to severity (7). Rajasekaran et al. define 4 "spine at risk" signs, namely subluxation, retropulsion of the vertebral body, lateral translation and toppling, which have a prognostic value (9). According to the authors, the presence of more than 2 signs is an indication for surgery, while more than 3 signs indicate a risk of progression to severe kyphosis (7, 10). Childhood Pott's disease presents the particularity of ongoing deformity even after healing of the disease due to the growing nature of the spine (9).

Diagnostic features

Isolation of *Mycobacterium tuberculosis* by culture or molecular assays is required to make a definitive aetiological diagnosis. CT-guided biopsy is the gold standard to collect the sample. If there is a surgical indication, it can also be obtained surgically (1, 7).

Radiological findings

Plain radiographs are abnormal in 99% of cases and may show a volume loss of the vertebral plate and disc, anterior osteopenia and lytic lesions, osteosynthesis, the presence of an abscess, and spinal deformity in late disease (1, 7). Of note, osteolysis is visible on plain radiographs when half of the bone density has already been lost, which implies late diagnosis (1, 4, 7). Concomitant pulmonary lesions are present in 50-70% of the cases (4, 6).

CT scan detects bone lesions earlier and more accurately than plain radiographs. The exact bony extent of lesions, the condition of the posterior column and joints, and the stability of the spine can all be assessed. As stated by Khanna et al., it may detect smaller lesions, the presence of calcifications in abscesses and epidural lesions with bony fragments, and show the aetiology of spinal cord compression. Most of all, it is used to perform CT scan-guided biopsy (7).

Full-spine and gadolinium-enhanced MRI is the examination of choice and allows early detection of lesions (4). Its sensitivity is 93% and its specificity is 96% (1). MRI is useful to evaluate the spinal cord, abscesses, soft tissues, vertebral anomalies and collapse, intervertebral discs, the presence of a tuberculoma or other lesions, and spinal deformity (1, 4, 7). It may also be used for follow-up. Lesions appear hypointense on T1 sequences and hyperintense on T2 sequences. Lesions of concern in spinal TB include multifocal disease with respect to the intervertebral disc, enhancement of the adjacent soft tissues, and abscesses or collections of granulation tissue in the peri-vertebral area. Subligamentary abscesses with thin and smooth walls have 90% specificity for TB (4, 8).

FDG-PET scan is not very specific, causes irradiation and is expensive. It can help identify a hypermetabolic abscess in order to perform a biopsy, but is generally not used to diagnose spinal TB (2, 9).

Scintigraphy has good sensitivity but low specificity. Tc99m scintigraphy, used for bone, has 35% false negatives, while gallium scintigraphy, used for inflammatory processes, has 70% false negatives. It can detect disease before MRI, but as there is no pathognomonic image for spinal TB, scintigraphy is of limited use for the work-up (1, 2, 8).

In terms of neuroimaging guidance for biopsy, ultrasound has been proven to be safe and effective and has several advantages, including the absence of ionising radiation, fast acquisition time, and good structural and vascular characterisation (11).

Laboratory testing

Detection of acid-fast bacilli by Ziehl-Neelsen staining has low sensitivity. Culture on Lowenstein's medium also has poor sensitivity (around 75%) in spinal TB due to paucibacillary disease, with the risk of false negatives, and has a long turnaround time of 6 to 8 weeks (12).

Due to the limitations of culture, histopathology is of value in the diagnosis of spinal TB. Characteristic findings include epithelioid cell granuloma, granular necrosis, lymphocytic infiltration and Langerhans cells (7).

Molecular assays are increasingly being used for diagnostic purposes. Their advantages include rapid turnaround time, detection of paucibacillary disease, and testing for resistance. A recent Cochrane review reported a sensitivity range of 96-100% and specificity of 53-100% for Xpert MTB-RIF (Cepheid) in bone aspirate, and sensitivity of 88-96% and specificity of 97% for the newer Xpert MTB-RIF Ultra (Cepheid) (13).

The erythrocyte sedimentation rate (ESR) is elevated above 20 mm/h in 60-83% of the cases and normalises with treatment (14). A full blood count may show inflammatory anaemia; the leucocyte count is normal in more than half of the cases (8).

Tuberculin skin test (TST) and Interferon-Gamma Release Assay (IGRA) may be helpful, but do not differentiate between latent and active tuberculosis. The TST is positive in 63-90% of cases of spinal TB (8).

Treatment

A. Medical

Drug-sensitive spinal TB is treated with standard anti-tuberculosis drugs, the intensive phase consisting of isoniazid (H), rifampicin (R), pyrazinamide (Z) and ethambutol (E) and the continuation phase consisting of isoniazid (H) and rifampicin (R) (15-18). These drugs have a good bone penetration (except in lesions with sclerotic wall). Drug-resistant spinal TB is less common and its management is more complex and beyond the scope of this review (19). The duration of treatment for drug-sensitive spinal TB is controversial, as there is no clear biological or radiological marker of cure. Shorter regimens, as long as they are effective, reduce unnecessary drug exposure. The main risk is recurrence of disease if treatment is incomplete.

The main guidelines (listed in Table 1) vary in their recommendations for the duration of treatment, with total durations ranging from 6 to 12 months. Longer durations are justified by the risk of severe complications if spinal TB is insufficiently treated, and by the difficulty of assessing treatment response. However, a recent systematic review and meta-analysis including only randomised controlled trials in adults and children, comparing short course of 6 months with a longer course of at least 9 months, with a follow-up period of at least 12 months after completion of chemotherapy, found that the healed status of spinal TB was equivalent in both groups, suggesting that shorter course of treatment may be considered in spinal tuberculosis, although more homogeneous and specific paediatric studies are needed (20). Studies with longer follow-up periods are reassuring regarding the efficacy of 6-month regimens (21-23).

B. Surgical

The aim of surgical treatment is to drain the abscess if present, decompress and remove dead tissue from infected areas, improve spinal stability and prevent or correct spinal deformity (9). Surgical indications suggested by most studies include worsening neurological deficit, significant abscesses (especially with psoas involvement), severe kyphosis, absence of response to conservative treatment, and lesions with a sclerotic rim (1, 4). The choice of the surgical approach depends on factors such as age, comorbidities, location and number of lesions, severity of kyphosis, and surgeon expertise (9). The details of surgical management are a controversial topic and are beyond of the scope of this review.

C. Supportive

Traditionally, bed rest and back bracing were advocated in all cases. These measures are no longer recommended in the absence of surgical treatment, with the exception of cranio-occipital and in some cases cervical disease (1, 2, 7). Back bracing is considered unnecessary, as it has not been shown to be effective in preventing the progression of kyphosis and spinal stability is rarely compromised. There is no guideline on back bracing for surgery and the decision is left to the surgical team.

Tuberculosis Immune Reconstitution Inflammatory Syndrome (TB-IRIS)

TB-IRIS, as stated by Lanzafame et al., is an excessive immune response against *M. tuberculosis* that may occur in both HIV-infected and uninfected patients, during or after completion of anti-TB therapy (24). Three forms have been described. Paradoxical IRIS is defined as recurrent, new or worsening symptoms in a treated case after initiation of anti-TB drugs. Unmasking IRIS is an antiretroviral therapy (ART)-associated form of tuberculosis in which subclinical infection becomes apparent after ART initiation, usually in the first 3 months after ART initiation (25). IRIS of the central nervous system has been individualised from previous categories due to its specific features (25).

Lanzafame et al. propose 4 criteria to define TB-IRIS: initial clinical and radiological improvement with the start of TB treatment, secondary worsening during or after TB treatment, no condition reducing the efficacy of anti-TB drugs, and no alternative explanation for the clinical worsening (24). The localisation of TB-IRIS is independent of the primary localisation and most commonly involves the lymph nodes and the lungs (24).

Table 1: Summary of guidelines concerning treatment of paediatric spinal TB.

	Target population	Duration of quadritherapy (months)	Duration of bitherapy (months)	Total duration of treatment (months)
American Thoracic Society / Centre for Disease Control and Prevention / Infectious Diseases Society of America 2016 (15)	Children	2	7-10	9-12
British Infection Society 2009 (16)	Children and adults	2	10 if CNS involvement	12 if CNS involvement
WHO 2022 (17)	Children	2	10	12
NICE 2016 updated 2024 (18)	Children and adults	2	4 10 if CNS involvement	6 12 if CNS involvement

CNS=Central nervous system.

No specific diagnostic test has yet been found to differentiate IRIS from other causes of clinical worsening. Clinicians should be aware of this and not assume that exacerbation of symptoms and signs is due to wrong diagnosis, poor adherence, malabsorption of medication, treatment failure, resistance, adverse effect of drugs or immunodeficiency.

Anti-TB drugs should be continued in most cases. Steroids are beneficial, as shown in an RCT performed by Meintjes et al. (26). Other anti-inflammatory or immunomodulatory agents have been used anecdotally, and surgical intervention may be necessary (25, 27-29).

Conclusion

Spinal tuberculosis is a disease with insidious onset, with back pain often being the only sign. Delayed diagnosis increases the risk of complications, which can be severe. The duration of medical treatment and surgical management is still subject under debate, with shorter drug regimens showing promise. Reserving surgery for complicated cases and adapting the duration of chemotherapy to the clinical, biological and radiological response may be the best approach, but more studies are needed, notably large RCTs including children, as paediatric evidence is still scarce. This case review highlights the need to include Pott's disease in the differential diagnosis of a child presenting with thoracic or back pain.

The authors have no conflicts of interest to declare.

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