

Case Report

Spinal tuberculosis with concomitant spondylolisthesis: coexisting entities or ‘cause and effect’?

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Study design: Report of three unusual cases of coexisting spondylolisthesis and tuberculosis in the same patient.

Objectives: To document the rare occurrence and attempt to postulate the probable reasons for such an association.

Setting: Tertiary care teaching hospital in a developing country.

Methods: This communication reports the outcome of three cases where there was spondylolisthesis and spinal tuberculosis in the same patient. The probable reason for such an occurrence is discussed along with a literature review relevant to this topic.

Results: The cases responded favorably to conservative treatment with multidrug antitubercular chemotherapy and spinal braces.

Conclusions: Association of spondylolisthesis and spinal tuberculosis is extremely rare. If the tubercular process is fulminant, spondylolisthesis secondary to destruction of posterior elements by the infective process can occur. Gross destruction of anterior elements secondary to tuberculosis in some patients may place excessive stresses on the posterior elements and may precipitate a spondylolisthesis even if there was no active infection in the posterior elements. However, there remains a distinct possibility that pars defect may have existed prior to infective pathology.

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Introduction

Spondylolisthesis is defined as an anterior displacement of one vertebra on another. It may be dysplastic, isthmic, degenerative, traumatic or pathological. Pathological variety is extremely rare and has been reported in Albers–Schonberg disease, arthrogyposis, Paget’s disease, syphilitic bone disease, achondroplasia and metastatic bone diseases.^{1,2} Very rarely destruction of posterior elements or pars interarticularis due to tuberculosis may result in spondylolisthesis.^{3,4} We describe the outcome of three cases where spinal tuberculosis and spondylolisthesis co-existed in the same patient and try to explain the probable reason for the same.

Case report

Case 1

A 38-year-old female presented with a history of dull aching pain in the back since 6 months. Pain was of

insidious onset with occasional radiation to the posterior aspect of both the thighs. However, the patient was able to carry out her household activities. The pain was not accompanied by numbness, weakness or bladder involvement. She had no history of trauma, constitutional symptoms, swelling, haemoptysis or malena. She had received treatment elsewhere with no relief of symptoms. General physical examination was insignificant with no lymphadenopathy or organomegaly. Spinal examination revealed a prominence of S1 spinous process with a ‘step off’ sign. Mild local tenderness was present along with restriction of lumbar movements and paravertebral muscle spasm. She had no neurological deficit. Routine hematological and biochemical investigations were within normal limits except for a raised ESR (42 mm/1st h). Plain radiographs revealed a grade II spondylolisthesis of L5 over S1 (Figure 1a). A diagnosis of spondylolisthesis grade II (Meyerding’s classification) was made and she was advised a lumbosacral corset along with spinal flexion exercises. Prescribed treatment led to no improvement of symptoms. Rather, the pain increased in intensity over the

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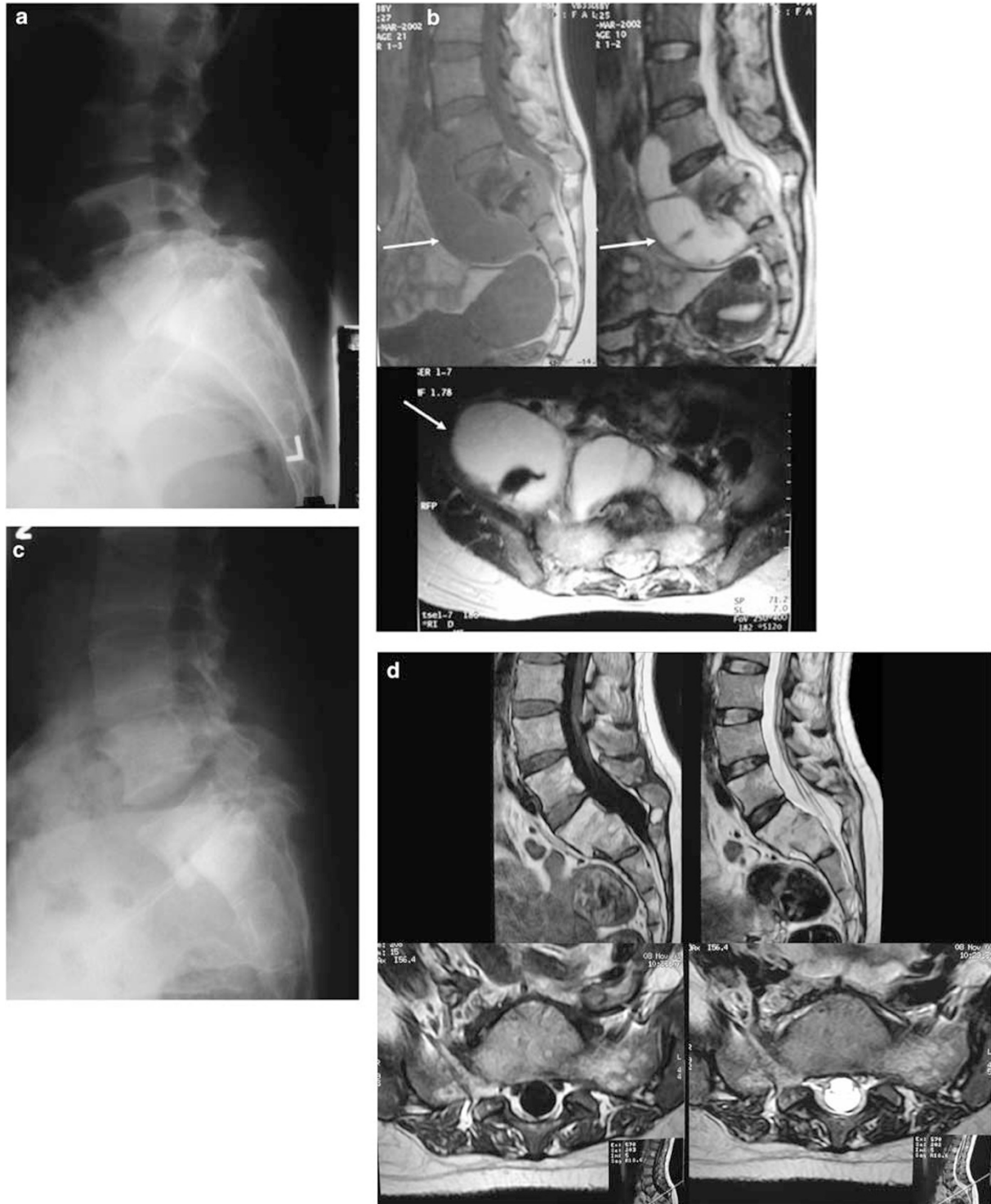


Figure 1 (a) Plain radiograph lateral view showing grade II spondylolisthesis of L5 over S1. (b) MRI suggested panvertebral tuberculosis of L5 and S1 vertebral bodies with involvement of posterior elements. There was a prevertebral abscess with characteristic hypointense signal in T1-weighted image (top left, white arrow) and hyperintense signal in T2-weighted image (top right, white arrow) along with a right-sided psoas abscess (bottom, white arrow). (c) Radiographs at 18 months post antitubercular treatment showed partial collapse of L5 and a markedly reduced disc space between L5 and S1. (d) Sagittal and axial views on T1-weighted images (top left and bottom left) and T2-weighted images (top right and bottom right) following 18 months of antitubercular treatment showing complete resolution of the prevertebral and right-sided psoas abscess

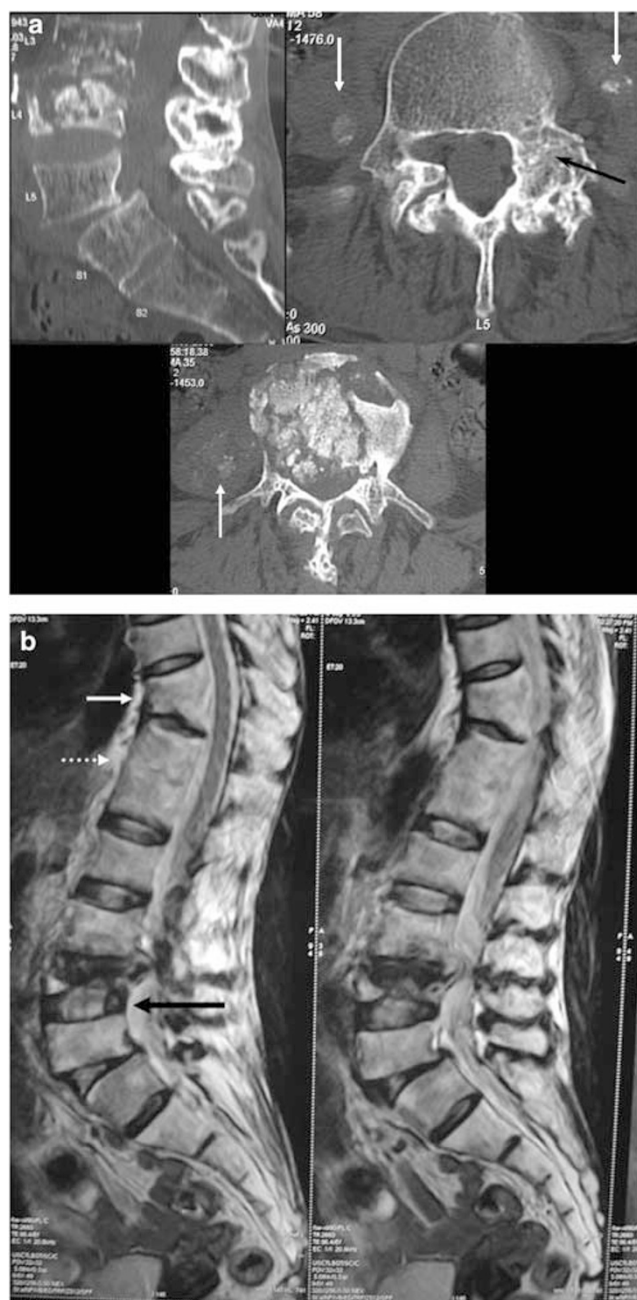


Figure 2 (a) CT scan revealed a reduced disc space L3–4 (top left), collapse with fragmentation of L4 vertebral body (top left and bottom) with irregular erosions and destruction of L3 vertebral end plate (top left). The posterior elements of L5 showed irregularity, sclerosis along with secondary degenerative changes in the facet joints (top right). There was grade II spondylolisthesis of L5 over S1 (top left). Adjacent psoas muscles appeared bulky with calcification in their substance (top right and bottom, white arrows). The left-side pedicle of L5 showed a pathological stress fracture (top right, black arrow). (b) MRI revealed block vertebrae at D10–11 (solid white arrow) and D12–L1 (dotted white arrow), decreased disc space between L3 and L4 with paravertebral involvement of the bodies of L3 and L4 (black arrow). Some anterior epidural space collection at the lower lumbar region suggested active disease

ensuing 4 weeks to the extent that even performing routine household activities became difficult. Magnetic resonance imaging (MRI) performed subsequently revealed a prevertebral collection (Figure 1b, top left and top right) and a large right-sided psoas abscess (Figure 1b, bottom). There was evidence of grade II spondylolisthesis of L5 over S1 vertebral body with reduced disc space at this level. The paradiscal region of L5 and S1 showed low signal intensity on T1-weighted images and high signal intensity on T2-weighted images. Similar changes of signal intensity were also seen in the posterior elements of L5 (Figure 1b, bottom). There was evidence of intraspinal collection of fluid from L5 to S1 (Figure 1b, top left and top right). MRI findings were consistent with panvertebral tuberculosis with concomitant spondylolisthesis. The psoas abscess was aspirated, yielding pus, which was subjected to Ziehl–Neelsen (Z–N) staining. However, no acid-fast bacteria could be demonstrated. A clinico-radiological diagnosis of tuberculosis L5–S1 with associated spondylolisthesis was made. The patient was put on bed rest for 12 weeks along with multidrug antitubercular chemotherapy. The regimen followed was of four drugs (INH, Rifampicin, Pyrazinamide and Ethambutol) for the intensive phase lasting 2 months and two drugs (INH and Rifampicin) for the maintenance phase lasting 16 months (RHEZ₂ + RH₁₆). By 12 weeks, the patient had marked symptomatic improvement and was mobilized using a Gold–Waith brace. At completion of 18 months of antitubercular chemotherapy, she was relieved of her local spinal tenderness and paravertebral spasm. Her ESR decreased to 22 mm/1st h. Radiographs taken at this stage showed partial collapse of L5 with markedly reduced L5/S1 disc space (Figure 1c). Flexion, extension views revealed no evidence of instability. Repeat MRI revealed complete resolution of the prevertebral (Figure 1d, top left and top right) and psoas abscess (Figure 1d, bottom left and bottom right) with fatty marrow replacement of the involved vertebral bodies. There was no significant canal compromise.

Case 2

A 60-year-old female presented with backache radiating to both her thighs for the last 18 months. The pain was present even at rest and was associated with occasional fever. She was treated for spinal tuberculosis about 30 years back, details of which were obscure. On examination, she had tenderness in the lumbosacral region along with paravertebral muscle spasm. Spinal movements were markedly restricted and there was no neurological deficit. Her routine blood investigations were essentially normal except for an elevated ESR of 55 mm/1st h. Radiographs revealed block vertebrae at D10–11, D12–L1, along with paradiscal destruction and reduced disc space between L3 and L4 vertebrae, suggestive of multiple level involvement secondary to tuberculosis and spondylolisthesis of L5 over S1. Computerized tomography (CT) scan revealed collapse with fragmentation of L4 vertebral body (Figure 2a, top left and



Figure 3 (a) Radiographs showed a grade II spondylolisthesis of L4 over L5 with a reduced disc space and fuzzy margins of L4 and L5 (black arrow). (b) After 18 months of antitubercular treatment, plain radiograph, lateral view, showed sclerosis and improved definition of the paradiscal regions of L4 and L5 vertebrae suggestive of healing (black arrow). (c) CT scan after treatment. A defect in the pars interarticularis of L4 (black arrow) without any evidence of involvement of posterior elements by the disease process. (d) MRI after treatment suggested healed tuberculosis L4–5 with spondylolisthesis L4 over L5 with fatty replacement of the marrow suggested by hyperintense signal in both T1- and T2-weighted images (left and right, respectively) (white arrows)

bottom) and destruction of the adjoining L3 vertebral end plate (Figure 2a, top left). Posterior elements of L4 vertebrae also showed sclerosis (Figure 2a, bottom). Adjacent psoas muscles appeared bulky with calcification in their substance (Figure 2a, top right and bottom, white arrows). The posterior elements of L5 showed irregularity and sclerosis with partial obliteration of the facet joint on the left side (Figure 2a, top right) along with spondylolisthesis of L5 over S1 (Figure 2a, top left). The left side pedicle of L5 showed a pathological stress fracture (Figure 2a, top right, black arrow) and the L5/S1 facet joints at that level showed marked degenerative changes with possibly facet subluxation (Figure 2a, top right). MRI revealed block vertebrae at D10–11, D12–L1 along with decreased disc space L3–4

suggestive of multiple level involvement due to the tubercular process (Figure 2b). There was some anterior epidural space collection at the lower lumbar region suggesting active disease. She was diagnosed clinico-radiologically as a treated case of spinal tuberculosis with multiple level involvement with recrudescence and started on antitubercular treatment (RHEZ₂ + RH₁₆). By 2 months she had symptomatic improvement and was mobilized with a Goldthwaite brace, antitubercular treatment being continued for a total of 18 months.

Case 3

A 42-year-old female patient presented with back pain for past 4–5 years. She also gave a history of low-grade

fever, malaise and weight loss for the past 1 year. The pain was insidious to begin with but had worsened rapidly for the past 1 year. By the time she presented to us it was present even at rest and increased on turning in bed, prolonged standing or walking. On examination there was tenderness in the lower lumbar region, marked reduction of movements and spasm. Fullness was present in the right iliac fossa, suggestive of a psoas abscess. Radiographs showed grade II spondylolisthesis of L4 over L5 with reduced disc space and fuzzy margins of L4 and L5 (Figure 3a). Aspiration of the fullness in the right iliac fossa revealed pus. However, no acid-fast bacilli could be demonstrated on Z-N staining. ESR was elevated to 35 mm/1st h with mild anemia (hemoglobin 9.0 gm%). A clinico-radiological diagnosis of tuberculosis was made and antitubercular treatment started (RHEZ₂ + RH₁₆).

At completion of the 18-months course of antitubercular treatment, the patient had symptomatic improvement and was able to carry out routine activities without significant discomfort. There were no constitutional symptoms and she gained weight. There was no local tenderness. At this stage the radiographs showed sclerosis of paradiscal regions of L4 and L5 vertebrae suggestive of healing (Figure 3b). CT scan revealed a defect in the pars interarticularis of L4 without any evidence of involvement of posterior elements by the disease process (Figure 3c, arrow). MRI suggested healed Koch's L4–5 with spondylolisthesis L4 over L5 with secondary degenerative changes (Figure 3d). There was no evidence of any marrow edema, epidural or psoas collection to suggest any residual disease activity.

Discussion

Of all cases of tuberculosis in the body, spinal tuberculosis accounts for about 2% cases.⁵ It usually affects the dorsal and dorsolumbar regions. The lower lumbar region is involved in only 10–15% of all spinal tuberculosis cases, the lumbosacral junction being affected in only 2–3%.⁶ Several large series have focused on tubercular lesion of the lumbosacral region (Bhojraj *et al*, 66 cases; Moon *et al*, 56 cases; Rajasekaran *et al*, 53 cases; Pun *et al*, 26 cases).^{5–8} However, none of them have reported any case of spondylolisthesis either secondary to or coexisting with tuberculosis in the same patient. Pathological spondylolisthesis secondary to tuberculosis, or coexisting spondylolisthesis and spinal tuberculosis is an extremely rare occurrence. The only cases on record are those by Ratliff (1956), Nissen-Lie (1941) quoted by Ratliff, Newman (1963) and Tuli (1997).^{1,3,4} In the case reported by Ratliff, spondylolisthesis was present before the tubercular process started.³ The case by Nissen-Lie had no radiological evidence of defect in the pars interarticularis before tuberculosis. He believed that extensive vertebral destruction by disease probably produced a 'stress lesion' of the neural arch facilitating listhesis.³ The details of two cases by Newman and six cases of Tuli are obscure.^{1,4} Tuli described occurrence of such cases in

middle-aged patients. The infective tubercular pathology was suspected in these cases because of destructive changes in the posterior elements in X-rays. The tubercular etiology was proved later, on subsequent surgery and histopathological examination of tissue.⁴ There is no evidence in the literature to suggest that spondylolisthesis predisposes to any infection. Any spine, normal or anomalous, can be equally affected by tuberculosis, and the spondylolisthetic segment cannot be a predilection site of tuberculosis. Of the three cases that we have presented, there was suspicion of tubercular involvement of posterior elements in two (case 1 and 2). We believe that the tubercular process involving the posterior elements may have caused the secondary spondylolisthesis in these cases. In case 1, the spondylolisthesis was probably due to active panvertebral tuberculosis at L5/S1 level, while in case 2 it could be either due to posterior element involvement due to recrudescence or as a result of secondary degeneration with a stress fracture caused due to old healed tuberculosis at multiple levels, which also involved the posterior elements of L5. The CT scan did reveal a pathological stress fracture of the left-sided pedicle of L5 (Figure 2a, top right, black arrow) with gross degenerative changes and possibly facet subluxation (Figure 2a, top right). To categorically say that there was no pre-existing spondylolisthesis in these two cases is impossible, although considering the radiological picture and the relatively short history it seems unlikely. However, in case 3, the infective process seems to have been confined to the usual anterior site (paradiscal) without involving the posterior elements. The defect in the pars in this case seems to be unrelated to the infective pathology and could have existed before the infection manifested, similar to the case reported by Ratliff.³ Suspicion of an infective pathology coexisting or contributing to the listhesis should be entertained in all patients who have significant local spinal tenderness and muscle spasm especially in endemic countries along with telltale radiological signs of a diminished disc space and paradiscal regional osteoporosis. These clinical and radiological signs would be the same as those found in isolated spinal tuberculosis. There was no progress of slippage in any of the three cases we reported. The lumbar lordosis and lumbosacral angle showed the following change.

	Lordosis (Initial/ last follow-up)	Lumbosacral angle (Initial/last follow-up)
Case 1	53/56°	40/55°
Case 2	55/60°	44/47°
Case 3	05/33°	10/38°

Although we anticipated a difficulty in achieving spontaneous fusion in cases with coexisting tuberculosis and listhesis due to segmental instability, by following the 'middle path regime' of treating spinal tuberculosis

advocated by Tuli,⁹ we did not encounter any major problems. The end result was bony fusion in case 1 and a fibroosseous healing in cases 2 and 3. The association of listhesis with tuberculosis in the same patient has been reported only rarely in the literature. Based on our experience in treating three such cases, we advocate a conservative approach ('middle path regime') in low-grade listhesis without neural deficit with spinal tuberculosis. An initial period of 3-months bed rest along with antitubercular treatment minimizes the forces that would tend to cause a progress of slippage and subsequently the patient can be mobilized with the help of a brace. We observed that in these patients symptoms improved and there was no demonstrable instability at the end of the treatment. However, in situations where there is a neural deficit or if the patient is having a high-grade listhesis, then radical debridement with/without decompression, instrumentation and fusion would be more appropriate. Tuberculosis has been reported with ankylosing spondylosis⁹ and listhesis. We did not find any other spinal pathology reported as coexisting with tuberculosis. Once tuberculosis occurs at the same level as a listhesis, healing is by fibro-osseous or osseous fusion, which in a way would help in stabilizing the listhesis and prevent further slippage. Owing to erect human posture and exaggerated lumbar lordosis, forward vertebral slipping is a constant tendency. The bony architecture counteracting the above tendency is the facet articulations, intact pedicles and neural arches, normal bone structure and integrity of soft tissue.² These restrains are collectively called 'bony hook' or 'articular bolt'.¹⁰ Any pathology compromising this 'bony hook' will allow forward slip of a vertebra on the one below it. Gross destruction of anterior elements secondary to tuberculosis in some patients may place excessive stresses on this 'bony hook' and may precipitate a spondylolisthesis even if there was no active disease in the posterior elements. This would be a definite possibility in cases where the 'bony hook' was already weak as in osteoporosis or a pre-existing spondylosis. CT/MRI are excellent radiological investigations to assess patients with spondylolisthesis and diagnose pathological spondylolisthesis in early stages since posterior element involvement cannot be easily evaluated in plain radiographs. The mainstay of diagnosis rests on high clinical suspicion. Under the best of circumstances, a positive acid-fast bacteria staining or culture can be obtained in only 30% cases of osteoarticular tuberculosis.⁴ In endemic countries,

with a clinicoradiological diagnosis, one can initiate the patient on antitubercular therapy and look for clinical response.^{9,11} A new and promising modality for investigation is the polymerase chain reaction test for tuberculosis, which, if facilities are available, will help greatly in diagnosis. It is an extremely sensitive method (~100%) and can detect as low as 10 fg DNA (two bacteria). The results are available within 24 h.¹² The clinical course of the disease may be indolent and the patient may be apparently healthy without any associated constitutional symptoms. The results of conservative treatment of tubercular spondylolisthesis with spinal braces and multidrug antitubercular chemotherapy are satisfactory and surgical stabilization is seldom needed.^{2,5,6,8}

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