ELSEVIER

CrossMark

The Spine Journal 15 (2015) e1–e6

THE SPINE JOURNAL

Case Report

Spinal tuberculosis of the lumbar spine after percutaneous vertebral augmentation (vertebroplasty or kyphoplasty)

Ming-Xiang Zou, MD, Xiao-Bin Wang, MD, Jing Li, MD*, Guo-Hua Lv, MD, You-Wen Deng, MD

Department of Spine Surgery, The Second Xiangya Hospital of Central South University, No. 139, Middle of Renmin Rd, Changsha, Hunan 410011, People's Republic of China

Received 17 September 2014; revised 20 January 2015; accepted 18 February 2015

Abstract

BACKGROUND CONTEXT: Spinal tuberculosis occurring after percutaneous vertebral augmentation has rarely been described. To date, only two such cases have been documented in the literature. Vertebral augmentation may reactivate a quiescent tuberculous lesion and promote the infective process in elderly patients with or without immunosuppression, thereby resulting in poor outcomes.

PURPOSE: The purposes of this study were to present two cases in which spinal tuberculosis occurred after vertebroplasty or kyphoplasty, to highlight the clinical features and need for early diagnosis of this pathology, and to postulate probable reasons for this association.

STUDY DESIGN: This study is based on a clinical case series and literature review.

METHODS: In this report, we review the clinical histories of two old women undergoing vertebral augmentation with subsequent spinal tuberculosis.

RESULTS: The first patient responded favorably to conservative treatment with multidrug antitubercular therapy and spinal braces. The second patient underwent surgical debridement through a posterior approach alone, without instrumentation, combined with adjuvant chemotherapy. By 1 year after treatment, both patients had experienced almost complete recovery and continued to be seen for follow-up visits.

CONCLUSIONS: Suspicion should be high, and magnetic resonance imaging is warranted in cases with deteriorating clinical symptoms and signs of acute infection after vertebral augmentation. We propose obtaining exhaustive microbiologic and histologic evidence via needle biopsy or open surgery in a timely fashion to establish an accurate diagnosis because tubercular spondylitis occurring in such a situation may progress rapidly.

Keywords: Spinal tuberculosis; *Mycobacterium tuberculosis*; Percutaneous vertebroplasty; Percutaneous kyphoplasty; Lumbar spine; Vertebral augmentation

Introduction

Percutaneous vertebral augmentation with transpedicular injection of bone cement, referred to as vertebroplasty or kyphoplasty, is now a well-established treatment for painful and osteoporotic compression fractures [1–3]. Although low complication rates are characteristic of vertebral augmentation, evidence suggests that percutaneous vertebroplasty and kyphoplasty can be associated with subsequent spinal infection [4–14]. Furthermore, several cases of osteomyelitis occurring at the site of injury have been reported [15–19]. However, spinal tuberculosis occurring

FDA device/drug status: Not applicable.

Author disclosures: ***M-XZ:*** Nothing to disclose. ***X-BW:*** Nothing to disclose. ***JL:*** Nothing to disclose. ***G-HL:*** Nothing to disclose. ***Y-WD:*** Nothing to disclose.

The authors declare that they have no conflicts of interest or sources of support. This article has not been published elsewhere in whole or in part. All authors agree to give permission to reproduce the material signed by the author(s) and publishers concerned. The study protocol was approved by the Institutional Review Board at the Second Xiangya Hospital of Central South University, Hunan, People's Republic of China, and written informed consent was obtained from the patient for publication of this study and any accompanying images.

* Corresponding author. Department of Spine Surgery, The Second Xiangya Hospital of Central South University, No. 139, Middle of Renmin Rd, Changsha, Hunan 410011, People's Republic of China. Tel.: (86) 73185295624; fax: (86) 73182654334.

E-mail address: jingli1969@126.com (J. Li)

http://dx.doi.org/10.1016/j.spinee.2015.02.032

after vertebral augmentation has been rarely described in the English language literature, with only two anecdotal cases documented to date [20,21].

Here, we report two patients with fulminant tuberculous spondylitis after lumbar vertebroplasty or kyphoplasty. We highlight the clinical features and need for early diagnosis of this pathology. We also postulate probable reasons for this association.

Case reports

Case 1

A previously healthy 68-year-old woman was admitted to our institution on February 2012 with a 3-month history of considerable pain in the lumbosacral region, without radiating leg pain. Ambulation aggravated the pain. She also complained of intermittent low-grade afternoon fever in the absence of night sweats, malaise, or weight loss. She had suffered a vertebral compression fracture because of trauma on November 2011 and underwent percutaneous vertebroplasty at the L2 level with an uneventful recovery (Fig. 1A–C). Initially, there was no evidence of active infection, including tuberculosis; therefore, the vertebral augmentation procedure was performed. A biopsy was performed during vertebroplasty, after the vertebral body had been accessed through the pedicle with the cannulas. The biopsy (Fig. 3A) and microbiologic analysis, including acid-fast bacilli culture of the specimen, failed to show any pathologic cause of the fracture.

She was afebrile on admission. Physical examination revealed light tenderness over the infected area with paravertebral muscle spasm, a positive straight leg–raising test and the Bragard sign in the right lower leg. Spinal movements were markedly restricted, but neurologic examination was largely negative. Examinations of her head, neck, and heart were normal. No rash or lymphadenopathy was found. An abdominal examination was normal, with no tenderness or hepatosplenomegaly. Laboratory studies revealed a white blood cell count of 5,900 per mm^3 with 76.8% neutrophils, erythrocyte sedimentation rate of 66.0 mm per hour, and C-reactive protein of 31.4 mg/dL. Tests for human immunodeficiency virus and hepatitis B virus were negative.

Magnetic resonance imaging (MRI) of the lumbosacral spine indicated severe spondylitis manifesting as abnormal signal changes at the L1–L2 level with bony destruction of L2 (Fig. 1D and E). These lesions had not been detected approximately 3 months previously during an MRI examination conducted for the vertebroplasty. However, this patient was not a candidate for surgery because computed tomography (CT) of the lung, performed after abnormal findings that were obtained on chest radiography, revealed exudations in the right middle and left superior lobes, suggesting acute pulmonary infection. Therefore, antimicrobial treatment with intravenous cephaloridine (2.0 g given every 8 hours) was administered. After 12 days, a review of chest CT showed that the pulmonary infection had not responded to the antibiotic therapy; furthermore, the patient's presenting complaints had worsened, and her inflammatory markers were rising.

At this stage, MRI suggested deleterious progression of the spondylitis involving the L2–L3 level (Fig. 1F and G). The antibiotic treatment was discontinued. Although subsequent blood culture yielded no pathogens, the MycoDot test was positive for tuberculosis. The patient was immunoglobulin G seropositive, leading to a tentative diagnosis of tuberculosis. Further investigations did not reveal any definitive localization of the tuberculosis, and the patient denied traveling to tuberculosis-endemic areas. Nevertheless, the patient was given adjuvant chemotherapy comprising isoniazid (300 mg/d), rifampicin (450 mg/d), pyrazinamide (1,500 mg/d), and ethambutol (750 mg/d) as treatment consistent with a diagnosis of tuberculosis. Concurrently, CT-guided biopsy of intervertebral disc L1–L2 was undertaken for culture and histopathology, with the aim of establishing an accurate diagnosis.

During the course of the treatment, the patient's clinical symptoms were alleviated unexpectedly. Histology of the

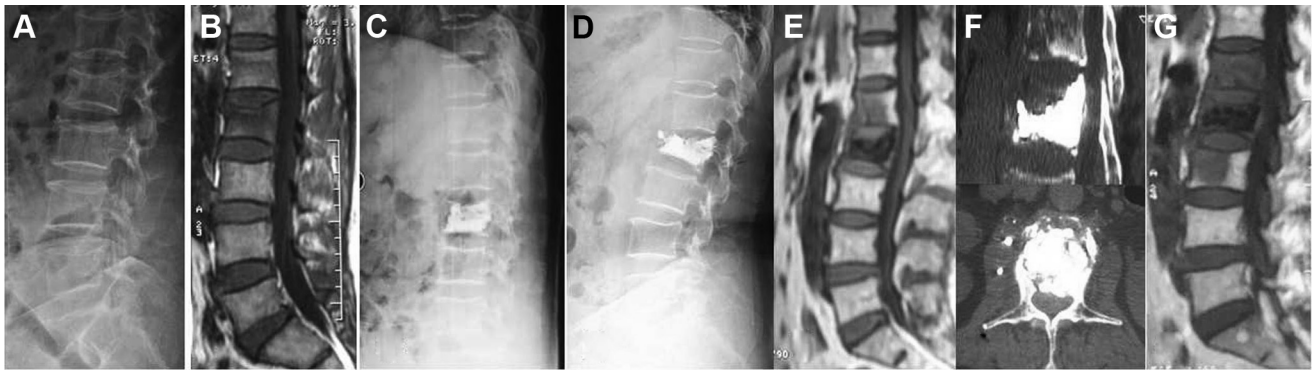

Fig. 1. (A and B) Sagittal magnetic resonance imaging (MRI) and plain radiographs of a 68-year-old woman indicating vertebral compression fracture at the L2 level. (C) Surgical vertebral augmentation was performed. (D and E) About 3 months later, plain radiography and MRI showed severe spondylitis at the L1–L2 level, with bony destruction of L2. (F and G) After 12 days, computed tomography and MRI of the same patient suggested rapid progression of infective spondylitis involving the L2–L3 level, with more severe bony destruction of L2.

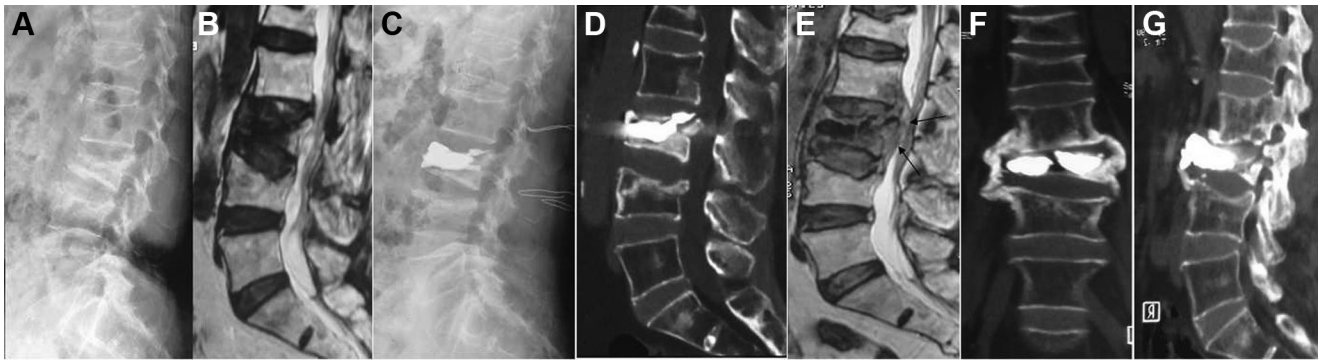

Fig. 2. (A and B) Lateral radiographs and magnetic resonance imaging (MRI) of a 67-year-old woman indicated an acute vertebral compression fracture of L3. (C) Vertebral augmentation was performed at this level. (D and E) On readmission, computed tomography (CT) and MRI showed paradiscal signal changes at the L2–L3 level with a ventral intracanal collection of fluid and bony destruction (black arrow). (F and G) One year after the initial surgery, CT of the same patient revealed a favorable radiological outcome with fibroosseous healing at the L2–L3 level.

infected tissue suggested tubercular spondylitis presenting as chronic granulomatous inflammation with a few scattered epithelioid cells (Fig. 3B). Subsequent acid-fast staining of the specimen identified *Mycobacterium tuberculosis*, which further verified the diagnosis of spinal tuberculosis (Fig. 3C). Although cultures of bronchial washings isolated no causative agent, the patient's pulmonary infection improved in parallel with initiation of the antitubercular regimen. By 10 days, the patient exhibited marked symptomatic improvement, and she was eventually discharged from the hospital. At the 1-year follow-up, she had almost recovered, and spinal immobilization with a brace allowed ambulation. Her antitubercular therapy was continued for 18 months.

Case 2

A 67-year-old woman presented to our department after having suffered unrelenting low back pain for the last 3

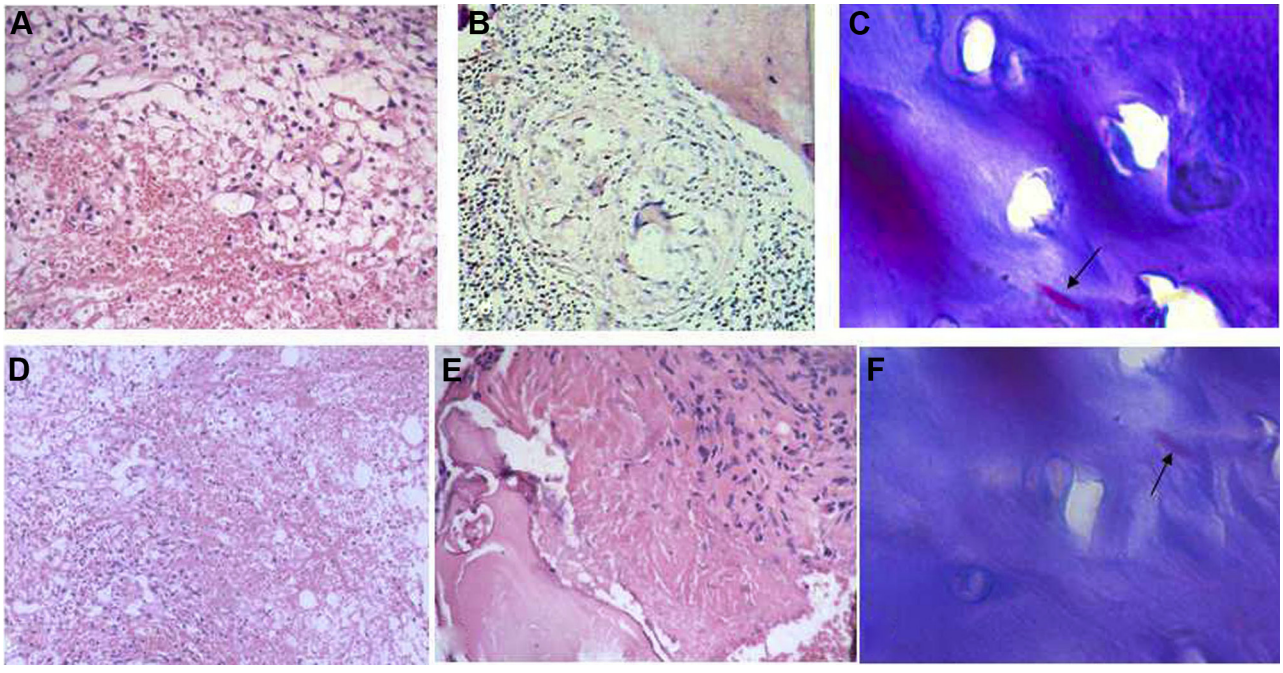

Fig. 3. Histopathology and microbiological findings. (A) Biopsy of the specimen in the first patient showed acute inflammatory cells infiltration and hemorrhage (×200, hematoxylin and eosin [H&E] staining). (B) Histology of infected tissue from this patient revealed a granulomatous formation with a few scattered epithelioid cells, suggesting the diagnosis of tuberculosis (×200, H&E staining). (C) Acid-fast staining of infected tissue identified causative organism consistent with *Mycobacterium tuberculosis* and further confirmed the diagnosis of tuberculosis (oil microscopy with ×1,000, black arrow). (D) Biopsy of the specimen from the second patient showed acute inflammatory cells infiltration and hemorrhage without any pathologic cause of the fracture (×100, H&E staining). (E) Histology confirmed tuberculous spondylitis presenting as chronically granulomatous inflammation with caseous necrosis (×100, H&E staining). (F) Microbiological analysis of the specimen using acid-fast staining identified *M. tuberculosis* (oil microscopy with ×1,000, black arrow).

days. She also had primary biliary cirrhosis and received symptomatic treatment irregularly, although the details of this treatment were obscure. She had undergone kyphoplasty at the L3 level because of a painful vertebral compression fracture caused by trauma about 1 month previously and had experienced an uneventful recovery (Fig. 2A–C). Initially, there was no evidence of active infection, including tuberculosis. Therefore, the vertebral augmentation procedure was performed. Her movement was markedly restricted, and she complained of intermittent mild pain in the right foot that was spontaneously alleviated. A routine biopsy (Fig. 3D) and microbiological analysis, including acid-fast bacilli culture, performed during the kyphoplasty did not show any pathologic cause of the fracture.

On admission, she was afebrile and had no constitutional symptoms, such as weight loss, chills, night sweats, or fatigue. Tests for human immunodeficiency virus and hepatitis B virus were negative. There was neurologic compromise (Grade D according to the Frankel grading system) but no apparent tenderness or percussion pain over the infected region on physical examination. Laboratory tests revealed elevated erythrocyte sedimentation rate (69.0 mm/h) and C-reactive protein (20.8 mg/dL). Although the patient had no previous history of tuberculosis and denied traveling to tuberculosis-endemic areas, plain radiography of the chest revealed lesions of fibrosis and calcification consistent with the diagnosis of obsolete pulmonary tuberculosis. Plain radiography of the abdomen was normal. Although her inflammatory markers were elevated, they were nonspecific and a definitive diagnosis could not be made. Whole body bone scintigraphy showed multifocal abnormal bone metabolism involving the extremities and spine, leading to a tentative diagnosis of bone metastasis from cancer, with an emphasis on management with analgesia. Unfortunately, the patient's clinical condition deteriorated with progressively rising inflammatory markers, and further management was required.

Despite negative blood culture, subsequent MRI of the lumbosacral spine revealed paradiscal signal changes at the L2–L3 level, with a ventral intracanal collection of fluid and bony destruction (Fig. 2D and E), suggesting infective spondylitis. Given the potential risk for increased neurologic deficit and progressively worsening clinical symptoms, posterior surgery comprising radical debridement, decompression via hemilaminectomy at the L2–L3 level, and drainage was performed. Although subsequent culture of intraoperative pus and infected tissue yielded no organisms, histology confirmed tubercular spondylitis presenting as chronic granulomatous inflammation and caseous necrosis with positive acid-fast staining for *M. tuberculosis* (Fig. 3E and F). Further investigations did not find any other definitive localization of tuberculosis.

An antitubercular regimen of four drugs, comprising isoniazid (300 mg/d), rifampicin (450 mg/d), pyrazinamide (1,500 mg/d), and ethambutol (750 mg/d), was commenced. Eighteen days postoperatively, the patient was discharged from the hospital with obvious symptomatic improvement. At the 1-year follow-up, she had returned to normal daily activity with favorable clinical and radiological outcomes (Fig. 2F and G).

Discussion

Percutaneous vertebral augmentation is an efficacious procedure for the treatment of unrelenting pain and the stabilization of vertebral body compression fractures. Although vertebroplasty and kyphoplasty have been demonstrated to be safe treatment options, there is evidence suggesting that spinal infection can occur after percutaneous vertebral augmentation, especially in patients with comorbidities that compromise their immune capacity [4–14]. Furthermore, several cases of osteomyelitis occurring at the site of injury have been reported [15–19].

However, reports on the association between spinal tuberculosis and vertebral augmentation are rare. As summarized in the Table, only two pertinent cases focusing on active spinal tuberculosis after vertebral augmentation have been documented in the literature to date [20,21]. In these two reports, the tuberculosis developed in an elderly patient with premorbid conditions that were similar to those in our two cases. Osteoporotic fractures are a hallmark of old age, and immunosuppression and the relatively poor immune system in this population permit organisms to inoculate the operative site. These aspects may explain the similarities among our cases and the previously reported ones.

To our knowledge, vertebral involvement by tuberculosis often results from the hematogenous spread of tuberculous bacilli from a distant focus, most commonly the lung, or as a result of direct dissemination from a proximal paraaortic lymph node. In the context of penetrating trauma requiring vertebral augmentation, however, the underlying mechanisms through which tuberculosis can occur remain unclear. It is our impression that tuberculosis may be provoked via hematogenous inoculation of the causative agent at the site of injury in patients with active pulmonary tuberculosis. This situation appears more plausible in the first case described here. Despite our failure to identify the organism responsible for this patient's infective pulmonary disorder, signs of recovery after the initiation of diagnostic chemotherapy contributed to the diagnosis of active pulmonary tuberculosis. One hypothesis is that a new tuberculous lesion may be initiated by local reactivation of quiescent bacteria or by the release of mycobacteria from macrophages infected by tuberculous bacilli that have migrated to the injury site [16]. This explanation may account, to some extent, for the break out of tuberculosis in our second patient.

We cannot completely exclude the possibility that spinal tuberculosis may arise at the site of previous trauma by chance, as spinal tuberculosis may mimic a compression fracture [22]. In contrast with the indolent course that

Table
Summary of previous reports focused on active spinal tuberculosis occurring after vertebral augmentation

Reference	Age (y)	Gender	Type of vertebral augmentation	Risk factors	Presenting symptoms	Time to therapy	Mode of therapy	Outcome	Follow-up
Ivo et al. [20]	70	Male	Kyphoplasty	Acute exacerbation of chronic obstructive pulmonary disease, Type II diabetes mellitus and esophagitis	Backache, fever, and general weakness	2 wk	Ineffective antimicrobial treatment with ceftriaxone and flucloxacillin followed by posterior instrumentation of Th11–L3 and anterior debridement, corporectomy of L1 and interposition of a titanium mesh cage filled with autologous rib graft and antitubercular treatment	Died from septic multiple organ failure	None
Bouvresse et al. [21]	69	Male	Vertebroplasty	Liver transplantation, hereditary hemochromatosis, and long-term use of immunosuppressive drugs	Low back pain, left L5 sciatica, and acute urinary retention	1 mo	Posterior decompression via L5–S1 laminectomy and adjuvant antitubercular chemotherapy at standard dose	Recovered and discharged from hospital 2 wk later	Not available

spinal tuberculosis usually takes, our cases progressed rapidly, requiring effective management in a timely fashion. This inconsistency may suggest that the true causal relationship between vertebral augmentation and spinal tuberculosis is elusive. We tend to believe that vertebral augmentation leading to the initiation of a locus minoris resistentiae may act as a trigger for the reactivation of an already-present inactive tuberculous focus. Furthermore, trauma may alter the local tissue response, precipitating the infective process of spinal tuberculosis.

Early diagnosis requires a high index of clinical suspicion. However, the nonspecific clinical symptoms and signs of this pathology preclude early diagnosis in most cases. Although MRI is the most useful diagnostic modality for spinal tuberculosis, a confident diagnosis from the results often cannot be made because of the nonspecific radiologic findings. Serology or hematology for tuberculosis before invasive diagnostic procedures are undertaken is recommended, but confirmation of the diagnosis is largely dependent on microbiologic and histologic evidence. In addition, detailed history taking focusing on previous tuberculosis provides invaluable information that can hasten the diagnosis.

Our present criterion is to obtain infected tissue by percutaneous needle biopsy or open surgery for bacteriologic and histocytologic examination to identify the causative pathogen in cases of suspicious infection after vertebroplasty [23]. Usually, percutaneous needle biopsy is adequate for determining the correct diagnosis, but open biopsy is necessary when needle biopsy fails, or it can be performed during definitive surgery [24]. In our patients, the diagnosis was made on the basis of pathologic examination together with positive culture outcomes of infected specimens collected by needle biopsy or surgery.

Normally, treatment should be initiated after the causative agent has been identified. Based on clinicoradiologic evidence, however, one may institute chemotherapy and look for a clinical response in cases occurring in an endemic area or with a documented history of tuberculosis [25,26]. In the previous reports, the first-line treatment of tuberculous vertebroplasty or kyphoplasty has been surgical debridement. However, this infection may respond favorably to antitubercular medication, and conservative treatment may be feasible, especially in patients without significant neurologic deficits, extensive bony destruction, or sequestration [24,27].

Recently, An et al. [28] reported satisfactory results for 36 patients (aged 60–85 years) with spinal tuberculosis, who were treated with antitubercular therapy alone. Likewise, we obtained a favorable outcome in our first case. However, surgical intervention is occasionally indicated in patients with neurologic injury, progressive cold abscess, or therapeutically refractory disease [29]. Our second patient underwent posterior-only debridement and drainage because of her worsening clinical condition and, most importantly, in an attempt to identify the pathogen to adjust

the medication according to susceptibility results. Instrumented stabilization was not undertaken because of reliable spinal stability, because of sparing of the bilateral facet joints intraoperatively, and in consideration of her age. Additionally, the presence of single-segment tuberculosis with no need for kyphosis correction and no palpable paravertebral abscess also dictated our choice of procedure [30]. This strategy may be indirectly justified by the subsequent satisfactory outcome.

Conclusions

Vertebral augmentation may reactivate a quiescent tuberculous lesion and promote the infective process in an elderly patient with or without immunosuppression. Suspicion should be high, and MRI is warranted in cases with deteriorating clinical symptoms and signs of acute infection after vertebral augmentation. We propose obtaining exhaustive microbiologic and histologic evidence via needle biopsy or open surgery in a timely fashion to establish an accurate diagnosis because tubercular spondylitis occurring in such a situation may progress rapidly.

References

[1] Chiras J, Sola-Martinez MT, Weill A, Rose M, Cognard C, Martin-Duverneuil N. Percutaneous vertebroplasty. Rev Med Interne 1995;16:854–9.
[2] Rao RD, Singrakhia MD. Painful osteoporotic vertebral fracture: pathogenesis, evaluation, and roles of vertebroplasty and kyphoplasty in its management. J Bone Joint Surg Am 2003;85:2010–22.
[3] Garfin SR, Yuan HA, Reiley MA. New technologies in spine: kyphoplasty and vertebroplasty for the treatment of painful osteoporotic compression fractures. Spine 2001;26:1511–5.
[4] Abdelrahman H, Siam AE, Shawky A, Ezzati A, Boehm H. Infection after vertebroplasty or kyphoplasty. A series of nine cases and review of literature. Spine J 2013;13:1809–17.
[5] Yu SW, Chen WJ, Lin WC, Chen YJ, Tu YK. Serious pyogenic spondylitis following vertebroplasty: a case report. Spine 2004;29:E209–11.
[6] Walker DH, Mummaneni P, Rodts GE Jr. Infected vertebroplasty. Report of two cases and review of the literature. Neurosurg Focus 2004;17:E6.
[7] Schmid KE, Boszczyk BM, Bierschneider M, Zarfl A, Robert B, Jaksche H. Spondylitis following vertebroplasty: a case report. Eur Spine J 2005;14:895–9.
[8] Söyüncü Y, Ozdemir H, Söyüncü S, Bigat Z, Gür S. Posterior spinal epidural abscess: an unusual complication of vertebroplasty. Joint Bone Spine 2006;73:753–5.
[9] Vats HS, McKiernan FE. Infected vertebroplasty: case report and review of literature. Spine 2006;31:E859–62.
[10] Gaye M, Fuentes S, Pech-Gourg G, Benhima Y, Dufour H. Spondylitis following vertebroplasty. Case report and review of the literature. Neurochirurgie 2008;54:551–5.
[11] Lin WC, Lee CH, Chen SH, Lui CC. Unusual presentation of infected vertebroplasty with delayed cement dislodgment in an immunocompromised patient: case report and review of literature. Cardiovasc Intervent Radiol 2008;31(2 Suppl):S231–5.
[12] Hong HS, Chang MC, Liu CL, Chen TH. Is aggressive surgery necessary for acute postoperative deep spinal wound infection? Spine 2008;33:2473–8.
[13] Shin JH, Ha KY, Kim KW, Lee JS, Joo MW. Surgical treatment for delayed pyogenic spondylitis after percutaneous vertebroplasty and kyphoplasty. Report of 4 cases. J Neurosurg Spine 2008;9:265–72.
[14] Schofer MD, Efe T, Timmesfeld N, Kortmann HR, Quante M. Comparison of kyphoplasty and vertebroplasty in the treatment of fresh vertebral compression fractures. Arch Orthop Trauma Surg 2009;129:1391–9.
[15] Stuart D. Local osteo-articular tuberculosis complicating closed fractures: report of two cases. J Bone Joint Surg Br 1976;58:248–9.
[16] Weir WR, Muraleedharan MV. Tuberculosis arising at the site of physical injury: eight case histories. J Infect 1983;7:63–6.
[17] Sendi P, Friedl A, Graber P, Zimmerli W. Reactivation of dormant microorganisms following a trauma. Pneumonia, sternal abscess and calcaneus osteomyelitis due to Mycobacterium tuberculosis. Neth J Med 2008;66:363–4.
[18] Ferris BD, Goldie B, Weir W. An unusual presentation of tuberculosis—‘‘injury TB.’’. Injury 1987;18:347–9.
[19] Kumar S, Agarwal A, Arora A. Skeletal tuberculosis following fracture fixation. A report of five cases. J Bone Joint Surg Am 2006;88:1101–6.
[20] Ivo R, Sobottke R, Seifert H, Ortmann M, Eysel P. Tuberculous spondylitis and paravertebral abscess formation after kyphoplasty: a case report. Spine 2010;35:E559–63.
[21] Bouvresse S, Chiras J, Bricaire F, Bossi P. Pott’s disease occurring after percutaneous vertebroplasty: an unusual illustration of the principle of locus minoris resistentiae. J Infect 2006;53:e251–3.
[22] Dass B, Puet TA, Watanakunakorn C. Tuberculosis of the spine (Pott’s disease) presenting as ‘‘compression fractures.’’. Spinal Cord 2002;40:604–8.
[23] Allen RT, Kum JB, Weidner N, Hulst JB, Garfin SR. Biopsy of osteoporotic vertebral compression fractures during kyphoplasty: unsuspected histologic findings of chronic osteitis without clinical evidence of osteomyelitis. Spine 2009;34:1486–91.
[24] Cheung WY, Luk KD. Clinical and radiological outcomes after conservative treatment of TB spondylitis: is the 15 years’ follow-up in the MRC study long enough? Eur Spine J 2013;22(Suppl 4):S594–602.
[25] Tuli SM. General principles of osteoarticular tuberculosis. Clin Orthop 2002;398:11–9.
[26] Nene A, Bhojraj S. Results of nonsurgical treatment of thoracic spinal tuberculosis in adults. Spine J 2005;5:79–84.
[27] Dai LY, Jiang LS, Wang W, Cui YM. Single-stage anterior autogenous bone grafting and instrumentation in the surgical management of spinal tuberculosis. Spine 2005;30:2342–9.
[28] An JY, Li DW, Cui X, Ma YZ. Analysis of clinical characteristics of elderly patients with spinal tuberculosis and its clinical effects with conservative treatment. [in Chinese]. Zhongguo Gu Shang 2013;26:210–3.
[29] Jain AK. Tuberculosis of the spine: a fresh look at an old disease. J Bone Joint Surg Br 2010;92:905–13.
[30] Jain AK, Jain S. Instrumented stabilization in spinal tuberculosis. Int Orthop 2012;36:285–92.