Spinal tuberculosis occurring after a closed bursting fracture of the vertebrae

Bo Huang · Chang-Qing Li · Ying Zhuang ·
Jian-Ping Xu · Yue Zhou

Received: 11 June 2010 / Revised: 10 May 2011 / Accepted: 3 January 2012 / Published online: 26 January 2012
© Springer-Verlag 2012

Abstract

Introduction Only 12 cases of musculoskeletal tuberculosis (TB) that occurred at the site of recent blunt trauma have been previously described.

Materials and methods A 43-year-old previously healthy man experienced an uncomplicated bursting fracture at the L2 vertebra without any spinal tuberculosis (ST)-associated evidence. During the conservative treatment, he was in good physical condition with back pain totally subsiding and occasional ambulation in a spinal brace, whereas 7 months after the original trauma, he presented with a characteristic manifestation of ST at the same vertebra.

Results Intraoperatively, biopsy confirmed ST of the L2 vertebra and radical debridement with an anterior fusion was performed. The patient received an 18-month course of anti-TB regimen. Excellent clinical outcomes were achieved.

Conclusion In developing countries, where TB is still widely prevalent, there must be a heightened awareness of the risk of ST at recently fractured vertebrae, as demonstrated in this case.

Keywords Spinal tuberculosis ·
Mycobacterium tuberculosis · Bursting fracture

Introduction

Although tuberculosis (TB) has almost been extinct in many developed countries, the incidence of TB is still very high and TB cases have seemed to increase over the last decade. Skeleton is the most common site of extrapulmonary involvement, with 50% of these cases being spinal [1, 2]. The term *locus minoris resistentiae* is defined as a place of less resistance, any part or organ which is more susceptible than others to the attack of a morbid agent [3]. Here we report a case which illustrates a singular manifestation of *locus minoris resistentiae* with the arising of spinal tuberculosis (ST) into an area of recent blunt trauma. On admission to our hospital, this patient presented with severe back pain, lower extremity weakness and anaesthesia as well as febris and night sweats; furthermore, imaging and laboratory examination indicated a characteristic manifestation of ST. After 3 weeks anti-TB therapy, he underwent a spinal cord decompression and lesion biopsy, finally confirming ST of the L2 vertebrae. Interestingly, 7 months prior to admission, he had experienced a blunt trauma on his back and an uncomplicated bursting fracture of the L2 vertebrae had been found, while there was no evidence of ST at that time and during the subsequent 7-month conservative treatment period. To our knowledge, it is the second time to report ST arising from vertebrae which was recently diagnosed as a closed fracture [4]. As none of the previously mentioned risk factors for ST had existed in our patient and the only agent which may facilitate the break-out of the L2 vertebrae TB was the bursting fracture of the same vertebrae caused by a blunt trauma 7 months ago, this

C.-Q. Li and Y. Zhou are co-corresponding authors.

B. Huang · C.-Q. Li (✉) · Y. Zhuang · Y. Zhou (✉)
Department of Orthopedics, Xinqiao Hospital,
The Third Military Medical University, Chongqing 400037,
People's Republic of China
e-mail: younglee881@163.com

Y. Zhou
e-mail: happyzhou@vip.163.com

J.-P. Xu
Department of Pathology, Xinqiao Hospital,
The Third Military Medical University, Chongqing 400037,
People's Republic of China

Fig. 1 Computerized tomography (CT) and magnetic resonance imaging (MRI) manifestations immediately after blunt trauma to his back. **a–c** axial, sagittal and coronal CT scan demonstrating an uncomplicated bursting fracture of the L2 vertebrae (*arrow*) and the left transverse process fracture of L2. **c** MRI sagittal T2-weighted image revealing a compression deformity at L2 (*arrow*) without any spinal tuberculosis (ST) associated appearance

case demonstrates the risk that the fractured vertebrae may be a predisposed area where ST ensues.

Case report

A 43-year-old previously healthy man experienced a blunt trauma to his back by a falling object from the building in June 2008. No neurological lesion was found except for back pain. Computerized tomography (CT) and magnetic resonance imaging (MRI) in the local hospital revealed an uncomplicated bursting fracture of the L2 vertebrae and the

left transverse process fracture of L2 (Fig. 1). As neurological function was normal and no obvious spinal canal stenosis was found, no special treatment was performed at that time and he was just prescribed lying in bed. During the conservative treatment, he was in good physical condition with his back pain totally subsiding before long, and he occasionally ambulated with brace protection. Whereas about 7 months after the original trauma, he gradually developed aggravated recurrent back pain and lower extremity weakness and anaesthesia, along with febris and night sweats. For further diagnosis and treatment, he presented to our institution in February 2009.

Fig. 2 a, b axial and sagittal CT scan revealing worm biting like destruction of the L2 vertebral body 7 months after the original blunt trauma

On admission, the temperature was 37.7°C. There was no lymphadenopathy. Obvious kyphosis of the lumbar spine and constraint of the lumbar motion were found. Physical examination revealed tenderness of the L2 spinous process and the associated bilateral paravertebral space. The neurologic assessment demonstrated lower extremity weakness with grade 4 strength for the left leg

and grade 5 for the right leg, respectively. There was hypoesthesia along his left leg extending laterally to the thigh and foot. Deep tendon reflexes were normal and Babinski's reflex was absent. Bladder and bowel function were normal. The alkaline phosphatase was 82 U/L. A human immunodeficiency virus (HIV) was negative. In addition, erythrocyte sedimentation rate significantly increased (ESR, 47 mm/h) and C-reactive protein slightly raised (CRP, 8.2 mg/L), but anti-TB antibody was negative. Further diagnostic procedures like chest X-ray and abdominal ultrasound evidenced normal findings. A lumbosacral spine radiograph revealed a destruction of L2. CT scan for the lumbar spine showed worm biting like destruction of the L2 vertebral body, which is usually seen in tuberculous spondylitis (Fig. 2). MRI scan of his lumbar spine revealed the subligamentous, intraspinal and bilateral intrapsoas major soft tissue masses as well as the destruction of the L2 vertebral body and the adjacent intervertebral discs (Fig. 3).

The patient admitted to have a healthy body before. No systemic disease in the patient's past medical history was noted. TB was also denied for him and his family. An 8-kg unintentional weight loss associated with anorexia, as well as febris and night sweats for 1 month was described.

After admission to our hospital, he was started on a daily regimen of rifampicin, armazide, ethambutol and streptomycin for anti-TB treatment. Three weeks later, the patient's back pain partially improved and febris and night sweats subsided. ESR declined to the normal level (8 mm/h), whereas lower extremity weakness and hypoesthesia did almost not improve. So surgery was performed based on the decompression and stabilization of the spine. Anterior left-sided retroperitoneal approach was chosen. Intraoperatively, purulent yellowish material was seen originating from the L2 body, which had almost completely destroyed the vertebral body and expanded into the left psoas major. In addition, the intervertebral discs of the adjacent segments were also eroded. Frozen section of the lesion showed caseating granulomas with multinucleated giant cells (Fig. 4) intraoperatively. Subsequent radical debridement of the mass in the psoas major and the L2 body as well as the adjacent necrotic discs was performed. Autologous bone graft from the left anterior iliac crest was used to bridge the defect after resection of the L2 body for stability of the spine (Fig. 5).

The physical status presented to be gradually stable with the muscle strength of both lower limbs recovered to grade 5 and the left leg hypoesthesia partially recovered post-operatively. Further microbiological analysis detected acid-fast bacilli (AFB). Culture results were subsequently positive for *Mycobacterium tuberculosis*. All investigations for other pathogens were negative. The patient was placed in a spinal brace for stabilization of the vertebral column

Fig. 3 MRI indicating the subligamentous, intraspinal and bilateral intrapsoas major soft tissue masses as well as the destruction of the L2 vertebral body and the adjacent intervertebral discs 7 months after the

original blunt trauma. **a** sagittal T1-weighted image. **b** sagittal T2-weighted image. **c** coronal T2-weighted image

Fig. 4 Specimen obtained from biopsy of the L2 vertebrae revealing large areas of caseating granulomas with multinucleated giant cells of Langerhan's type (arrows) (hematoxylin and eosin stain; original magnification, $\times 200$)

and discharged to receive a planned 18-month course of anti-TB regimen. When last seen in May 2010, he had no further complaints.

Discussion

Since Sir Perciville Potts first described pott disease, or ST in 1779, ST has existed for centuries [5]. Although the prevalence of TB has reduced since the advent of anti-TB

drugs, recent studies indicated the increased incidence of extrapulmonary TB in industrialized countries [6]. A retrospective case-control study including 705 patients with TB in the United States from 1996 to 2000 showed women, non-Hispanic blacks, and HIV-positive persons had a significantly higher risk for extrapulmonary TB than men, non-Hispanic whites, and HIV-negative persons [7]. In another retrospective review conducted in France from 1980 to 1994, risk factors for ST included foreign birth site, diabetes mellitus, and prior exposure to TB [8]. Whereas none of the above mentioned risk factors had existed in our patient except for the identity of Asian resident. He was not suffering from HIV infection or diabetes mellitus and was not using any immunosuppressive medication. On the other hand, exposure to TB was also denied, and TB was not found out elsewhere in the body. The only agent which may facilitate the break-out of the L2 vertebrae TB was the bursting fracture of the same vertebrae caused by a blunt trauma 7 months ago.

In our case, the pre-existing hematoma from the bursting fracture of the L2 vertebrae obviously created a local media for hematogenous seeding of *Mycobacterium tuberculosis* and subsequent formation of abscess. Potential mechanisms of infection may include local congestion or stasis, or the readily available iron contained in hemoglobin that potentiates microbial proliferation [9]. Macrophages that contained dormant *Mycobacterium tuberculosis* at the potential site of infection entered the circulation and took root in the hematoma by chemotaxis [10]. The paravertebral venous plexus may be one possible route for the deposition of organism [11]. After reaching the hematoma, the tubercle

Fig. 5 Lateral lumbar spine X-ray 1 week after surgery indicating that autologous bone graft from the left anterior iliac crest bridged the defect after resection of the L2 body for stability of the spine (arrow)

bacilli were released and initiated a new focus of infection. Many studies have proved the remarkable capacity of *Mycobacterium tuberculosis* to survive within the hostile environment of the macrophage and their ability to escape immune defense mechanisms [12]. However, the exact mechanism by which TB happens in a recent traumatized site has not been revealed yet.

Only 12 cases of musculoskeletal TB that occurred at the site of recent blunt trauma have been previously described. All those cases had no breach of the overlying skins. Two of those occurred in calcaneus; 2, in sternum; 1, in ankle; 1, in forearm; 1, in fourth metacarpal bone; 1, in olecranon process; 1, in knee joint; 1, in thigh; 1, in sternoclavicular joint; and 2, in spine [4, 10, 13–15]. However, only one case of vertebral osteomyelitis due to *Mycobacterium tuberculosis* happening after a definite closed fracture of the spine was ever reported [4]. So it is the second case that ST arised from vertebrae which had been recently diagnosed as a closed bursting fracture. In the above 12 Cases of musculoskeletal TB, 6 cases had evidence of tuberculosis elsewhere in the body by *M. tuberculosis* inspection in sputum or pulmonary infiltration in chest X-ray or computerised tomography. The other six had

normal chest X-rays and at the time of their injury had been in good health. In our case, he had a good physical condition before initial trauma and in the 7-month interval, in addition, his chest X-ray was negative, which demonstrated no evidence of tuberculosis elsewhere in his body.

In conclusion, this is a fairly interesting and exceptional case. Seven months ago before admission to our hospital, this patient only had a blunt trauma on his back and all the imaging examinations just demonstrated an uncomplicated bursting fracture at the L2 vertebrae without any ST-associated evidence. In addition, after this, there was an about 7-month interval in which he had a good physical condition. While 7 months later after encountering the blunt trauma on his back, ST-related symptoms such as severe back pain, lower extremity weakness and anaesthesia as well as febris and night sweats emerged. Imaging and laboratory examination as well as specimen culture result finally confirmed the diagnosis of ST at L2. So we believe that in developing countries, where TB has still been very popular, the heightened awareness for the risk that ST occurs at recent fractured vertebrae should be maintained, as demonstrated in this case.

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

References

1. Turgut M (2001) Spinal tuberculosis (Pott's disease): its clinical presentation, surgical management, and survey study on 694 patients. *Neurosurg Rev* 24:8–13
2. Weaver P, Lifeso RM (1984) The radiological diagnosis of tuberculosis of the adult spine. *Skeletal Radiol* 12:178–186
3. Miller RA (1990) Locus minoris resistentiae and the Koebner phenomenon. *Int J Dermatol* 29:223–224
4. Govender S, Charles RW, Ballaram RS, Achary DM (1988) Vertebral osteomyelitis after a closed fracture of the spine. A case report. *S Afr Med J* 73:124–126
5. Arriaza BT, Salo W, Aufderheide AC, Holcomb TA (1995) Pre-columbian tuberculosis in northern chile: molecular and skeletal evidence. *Am J Phys Anthropol* 98:37–45
6. Gautam MP, Karki P, Rijal S, Singh R (2005) Pott's spine and paraplegia. *JNMA J Nepal Med Assoc* 44:106–115
7. Yang Z, Kong Y, Wilson F, Foxman B, Fowler AH, Marrs CF, Cave MD, Bates JH (2004) Identification of risk factors for extrapulmonary tuberculosis. *Clin Infect Dis* 38:199–205
8. Pertuiset E, Beaudreuil J, Liote F, Horusitzky A, Kemiche F, Richette P, Clerc-Wyél D, Cerf-Payrastré I, Dorfmann H, Glowinski J, Crouzet J, Bardin T, Meyer O, Dryll A, Ziza JM, Kahn MF, Kuntz D (1999) Spinal tuberculosis in adults. A study of 103 cases in a developed country, 1980–1994. *Medicine* 78:309–320
9. Chan ED, Kong PM, Fennelly K, Dwyer AP, Iseman MD (2001) Vertebral osteomyelitis due to infection with nontuberculous *Mycobacterium* species after blunt trauma to the back: 3 examples of the principle of locus minoris resistentiae. *Clin Infect Dis* 32:1506–1510
10. Weir WR, Muraleedharan MV (1983) Tuberculosis arising at the site of physical injury: eight case histories. *J Infect* 7:63–66

11. Korovessis P, Sidiropoulos P, Piperos G, Karagiannis A (1993) Spinal epidural abscess complicated closed vertebral fracture. A case report and review of literature. *Spine* 18:671–674
12. Pieters J (2008) *Mycobacterium tuberculosis* and the macrophage: maintaining a balance. *Cell Host Microb* 3:399–407
13. Sendi P, Friedl A, Graber P, Zimmerli W (2008) Reactivation of dormant microorganisms following a trauma. Pneumonia, sternal abscess and calcaneus osteomyelitis due to *Mycobacterium tuberculosis*. *Neth J Med* 66:363–364
14. Simon GL, Worthington MG (1982) An unusual case of pleural, epididymal and sternoclavicular tuberculosis. *J Infect* 4:259–261
15. Stead WW, Bates JH (1971) Evidence of a “silent” bacilleemia in primary tuberculosis. *Ann Intern Med* 74:559–561