Acute spinal cord compression due to epidural lipomatosis complicated by an abscess: magnetic resonance and pathology findings

Giulio Zuccoli · Nicolò Pipitone · Nicola De Carli ·
Luigi Vecchia · Stefano C. Bartoletti

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Abstract A 68-year-old male presented with rapidly progressive paraplegia. MR images of the thoracic spine were interpreted as being consistent with an abscess within an epidural lipomatosis compressing the spinal cord. Laminectomy was performed, and a large amount of pus was drained from the epidural lipomatosis, from which *Staphylococcus aureus* was isolated. This is the first reported case of an abscess involving an epidural lipomatosis.

Keywords Epidural lipomatosis · Spinal abscess · Magnetic resonance imaging

Introduction

Spinal epidural abscess (SEA) is a rare condition associated with high morbidity and mortality if untreated. Its incidence has been estimated at approximately one to two cases per 10,000 subjects [1]. SEA is characterized by a collection of pus within the epidural space. Predisposing factors include diabetes mellitus, glucocorticoids and non-steroidal immunosuppressive drugs as well as intravenous heroin use [2]. Other common predisposing factors are a history of trauma and/or of spinal surgery [2]. SEA usually affects the thoracolumbar level, arguably because of the extensive venous (Batson's) plexus located at this site [3]. Spinal epidural lipomatosis (SEL) is characterized by an excessive deposit of fatty tissue in the epidural space [4]. Obesity and glucocorticoid use are known risk factors for SEL [5, 6]. Herein, we describe the MR and pathological findings of a patient presenting with rapidly progressing paraplegia, which MR revealed to be due to spinal cord compression caused by a *Staphylococcus aureus* abscess in an epidural lipomatosis. The peculiarity of this case is discussed.

Case report

A 68-year-old man presented at our institution with progressively developed paraplegia having suffered from pain in the thoracic spine and slight fever during the last 5 days. ASIA impairment scale showed preservation of the sensory but not of the motor function (ASIA-B). Review of systems disclosed normal heart and lung findings including auscultation, no organomegaly or (rebound) tenderness on abdominal palpation, normal pulses, no skin rash, no focal neurological deficit other than the paraplegia. Complete

G. Zuccoli
Radiology Department, Arcispedale Santa Maria Nuova,
Reggio Emilia, Italy

G. Zuccoli (✉) · S. C. Bartoletti
Department of Pediatric Radiology, University of Pittsburgh
Medical Center, Children's Hospital of Pittsburgh,
One Children's Hospital Drive 4401 Penn Avenue,
Pittsburgh, PA 15224, USA
e-mail: giulio.zuccoli@gmail.com
URL: <http://www.chp.edu>

N. Pipitone
Department of Internal Medicine,
Arcispedale Santa Maria Nuova, Reggio Emilia, Italy

N. De Carli
Microbiology Unit, Arcispedale Santa Maria Nuova,
Reggio Emilia, Italy

L. Vecchia
Pathology Institute, Hospital S. Giuseppe, Empoli, Italy

blood count showed $10.12 \times 1,000/\text{mm}^3$ leukocytes, 12.9 g/dl hemoglobin, 125 mg/dl glucose. ESR and C-reactive protein were raised at 34 mm/1st hour and 10.1 mg/l (normal range <5), respectively. D-dimer, prothrombin time and partial thromboplastin time were 443 ng/ml, 14.5 and 29.6 s, respectively. The remainder of the blood workup was unremarkable. Past medical history included prostate cancer, hypertension, and hyperlipidemia. The patient underwent a CT scan of the abdomen, chest, and brain. Abdominal CT ruled out vascular aneurysms of the abdominal aorta, while chest CT showed centrilobular emphysema without signs of infection. Brain CT was also negative for infection. To evaluate spinal cord abnormalities, MR images were obtained, including sagittal T1-weighted images (TR, 450 ms; TE, 6 ms), axial T1-weighted images (TR, 450 ms; TE, 8 ms), sagittal T2-weighted images (TR, 3,500 ms; TE, 115 ms), sagittal Short Tau Inversion Recovery images (TR, 2,500 ms; TE, 160 ms, TI, 60), sagittal Spectral Presaturation with Inversion Recovery images (SPIR) (TR, 450 ms; TE, 6 ms). After intravenous contrast injection of 0.1 mmol/kg of gadoteric acid, sagittal Spectral Presaturation with Inversion Recovery images were acquired. The MR imaging study was performed using a 1T system. MR revealed a mass within the posterior epidural space extending from the third to the eight thoracic vertebra that compressed the dorsal medulla. The high signal intensity component of the mass seen on T1-weighted images (Fig. 1) was completely suppressed by both pre-contrast SPIR and STIR images (not shown). T2-weighted sagittal

Fig. 1 Axial T1-weighted image at T6 level demonstrates abnormal epidural fatty tissue deposition representing epidural lipomatosis (white arrow). A collection of hypointense tissue, consistent with an abscess, is seen within the epidural lipomatosis (asterisk). Please note the compressed spinal cord (arrowheads)

images showed high signal intensity in the posterior epidural fat consistent with fatty tissue including a well-demarcated high signal intensity abscess (Fig. 2a). The content of the mass did not enhance, while the T3–T4 inter-spinous space showed peripheral enhancement (Fig. 2b) with abnormal signal extending to the T3–T5 left paraspinal space where STIR images showed high signal intensity (not shown). MR images were interpreted as being consistent with an abscess within epidural lipomatosis. Based on the clinical symptoms and of the MR findings, the patient underwent urgent laminectomy from T4 down to T8. At surgery, a large amount of pus was drained from the epidural lipomatosis, from which *S. aureus* was isolated. Pathology showed mature adipose un-encapsulated tissue consistent with lipomatosis containing blood vessels and fragments of fibrous tissue and striated muscle (Fig. 3). Based on the radiological, surgical, pathological, and microbiological findings, SEL complicated by SEA was diagnosed. Treatment with ceftriaxone 1 g twice per day given intravenously commenced. The patient's paraplegia gradually regressed starting from day 1 after surgery, and resolved without clinical sequels. Two weeks after the admission, the patient was discharged home with intramuscular ceftriaxone 1 g die until attainment of remission. One month MR follow-up showed expected post-surgical changes without vertebral malalignments and an unremarkable spinal cord thus avoiding the need of fixation (Fig. 4).

Discussion

SEA is a rare infectious condition, associated with significant morbidity and mortality. Hematogenous spreading is thought to be the main route of the infection in SEA. Contiguous contamination of the epidural space from of an osteomyelitis or paravertebral abscess represents the other sources of SEA [7]. In our patient, signal intensity alterations were observed at the T3–T4 inter-spinous space extending to the T3–T5 left para-spinal space, suggesting that the infection may have spread from this area to the epidural fat, although the converse is also possible. There is some evidence suggesting that the incidence of SEA has increased due to spinal interventional procedures including steroid epidural injection, catheterization, and surgery [8, 9]. Predisposing factors for SEA are similar to those reported for other serious infections. In particular, diabetes mellitus, intravenous drug use, immunosuppressive therapy, cancer, HIV/AIDS and renal failure have all been described as risk factors [10]. Among bacterial infections, *S. aureus* is most frequently involved, being found in one-half of patients [2], but other infectious agents have also been reported [11–13]. However, in our patient, none of the

Fig. 2 The epidural lipomatosis (arrows) and the abscess (arrowheads) show high signal intensity on sagittal T2-weighted images (a). Sagittal SPIR image (b) after gadolinium injection. The epidural fatty tissue enhances (arrowheads). Please note the presence of inter-spinous space abscess at T3–T4 level (arrow)

Fig. 3 Histology shows mature un-encapsulated adipose tissue incorporating blood vessels and fragments of fibrous tissue and striated muscle. Dense inflammatory infiltrate consisting of granulocytic polymorphonucleate cells with foci of coagulative necrosis is seen. Transmural inflammatory infiltrate of the vessel wall showing polymorphonucleate cells and foci of coagulative necrosis is also present

above predisposing factors was present. Furthermore, the patient had no skin lesions at the time of the diagnosis.

SEL, a condition related to abnormal non-encapsulated fatty tissue deposition in the epidural space at thoracolumbar level [4], is rarely associated with neurological symptoms. Known predisposing factors are glucocorticoid therapy and Cushing's syndrome [5, 6, 14], while the idiopathic form has mostly been described in obese patients [6]. Our patient suffered from hyperlipidemia, but he neither showed abnormal fatty distribution at physical examination, nor he had impaired glucose tolerance, which

Fig. 4 One month MR follow-up shows post-surgical changes about the posterior aspect of the thoracic spine (asterisks). The physiological dorsal kyphosis is preserved. The cord and its canal are within normal limits

has been reported together as predisposing factors in one patient with symptomatic epidural lipomatosis [15]. Spinal cord compression caused by epidural abnormal fatty tissue deposition has rarely been described [16, 17]. To our knowledge, there are no previous reports describing the coexistence of both SEA and SEL.

Conclusions

This case is peculiar for a number of reasons. First, our patient had no known risk factors for spinal infection. Second, there were no classical predisposing factors for spinal lipomatosis either. Third, lipoma is usually not prone to become infected. At the same time, it should be borne in mind that lipomas are usually well vascularized, although the vascular network is often compressed by the distended adipocytes [18]. It may be thus hypothesized that in our patient an infection spread hematogenously to the epidural lipomatosis via the epidural plexus, and was able to invade it because of its rich vascular network. However, it is equally possible and perhaps more probable that the infection localized first to the T3-T4 inter-spinous space and subsequently spread to the epidural fat [19]. This case also highlights the pivotal role of MR in the identification of the epidural abscess and in guiding further investigations.

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

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