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Candida dubliniensis spondylodiscitis in an immunocompetent patient. Case report and review of the literature

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ABSTRACT

We describe what appears to be the first case of spondylodiscitis due to *Candida dubliniensis*. Our case adds to the current literature of the importance of *C. dubliniensis* as a cause of fungemia and subsequent deep seated infections. It highlights the importance of taking fungal as well as bacterial culture from biopsy specimens in patients with spondylodiscitis. We also review the literature covering the reported cases of *Candida* spondylodiscitis, which amount to about 100 over the last 5 decades.

1. Introduction

Spondylodiscitis is usually due to pyogenic bacteria (e.g. *Staphylococcus aureus*), but *Mycobacterium tuberculosis* and fungi are also occasionally involved [1,2]. Early recognition and timely intervention are important for successful management of osteomyelitis and spondylodiscitis. Blood cultures yield the etiological agent of pyogenic spondylodiscitis in more than a half of the cases. However, the yield may be significantly lower in non-pyogenic spinal infections such as fungal spondylodiscitis where fungemia may have been present only several months previously [3,4]. Surgical or needle biopsy specimens from the affected vertebral body or adjacent tissues are the cornerstone on etiological diagnosis. The importance of isolation of the etiological agent is underscored by the fact that treatment regimens for pyogenic bacteria, mycobacteria and fungi are completely different. Also, narrow-spectrum treatment options should be preferred to ameliorate the resistance problems related to the long treatments required.

No more than 5% of the cases of spondylodiscitis are caused by fungi; of these, *Candida* species are the most frequent agents [2]. *Candida* spondylodiscitis usually affects immunocompromised patients after hematogenous dissemination [5]. We report what appears to be the first case of spondylodiscitis caused by *Candida dubliniensis* in an immunocompetent patient.

2. Case

A 37-year old male who was an intravenous drug addict with chronic hepatitis C infection presented in May 2011 with severe lumbosacral pain. The pain radiated intermittently to both lower limbs but was not associated with any consistent changes in physical examination. Radiography of the lumbosacral spine in June 2011 was normal. As the pain escalated and became daily, new radiographs were obtained 1 month later, and now narrowing of the presacral space and a blurry edge adjacent to the fifth lumbar vertebral body were observed. This finding raised a suspicion of an infectious process and the patient was referred to our clinic. On admission (day 0) the erythrocyte sedimentation rate (ESR) was 45 mm/h, the C-reactive protein (CRP) 13 mg/l, the leukocytes 6.5×10^9/l and the hemoglobin 135 g/l. Magnetic resonance imaging (MRI) of the lumbosacral region on day 1 showed spondylodiscitis in the presacral space with bone edema, blurry edges of the end plates of the presacral discus, which was narrowed, and a presacral phlegmon 1 cm by depth (Fig. 1).

On day 3 the patient was prepared for a needle biopsy in general anesthesia. After induction of anesthesia with midazolam, fentanyl, propofol and rocuronium bromide the patient sustained an anaphylactic reaction and the procedure was discontinued.

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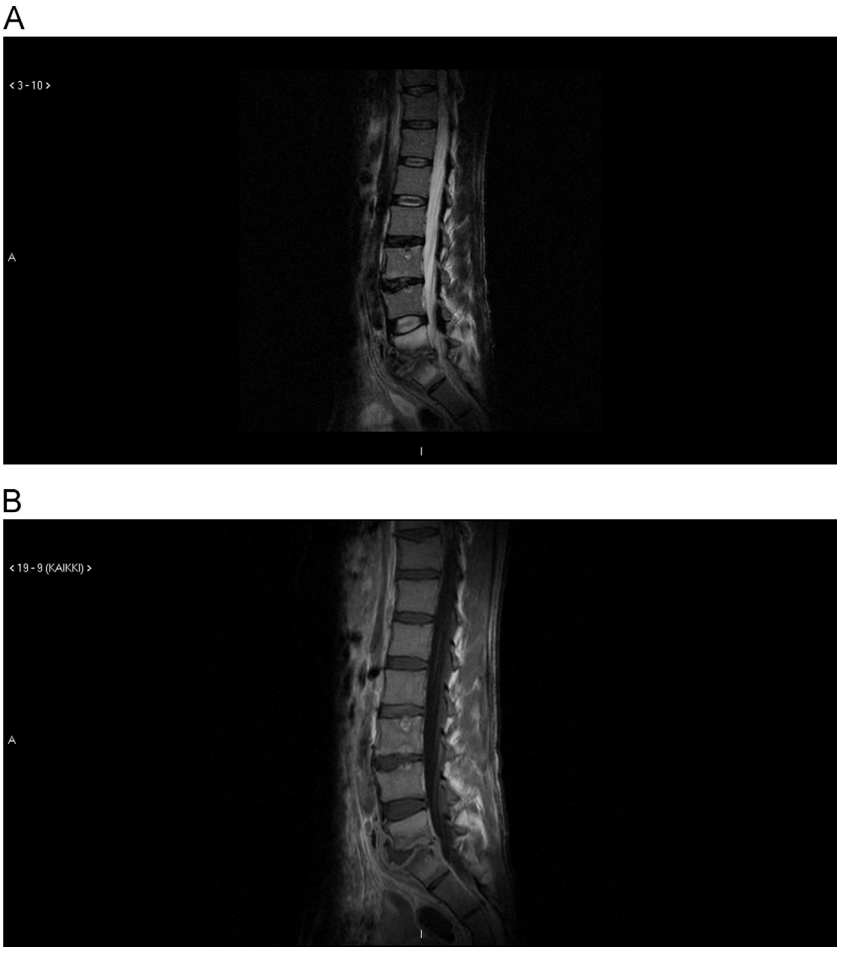

Fig. 1. Magnetic resonance imaging (MRI) of the lumbosacral region on July 12th showed LV-SI spondylodiscitis and presacral phlegmon. (A) T2 image and (B) T1 image with gadolinium contrast.

After epinephrine and norepinephrine and corticosteroid injections he was admitted to intensive care unit. The procedure was postponed until after he had been tested for allergies and he was referred home for 3 weeks. Now the ESR was 9 mm/h, the CRP 6 mg/l and the liver function tests within reference ranges.

On day 32 the patient was readmitted to the clinic for a new needle biopsy. Two days before admission his ESR was 26 mm/h and the CRP was 9 mg/l. Blood cultures were drawn on nine different occasions between 11th July and 13th August (Bactec 9240 system, Becton Dickinson, Sparks USA), and were negative for bacteria and fungi. Specific fungal vials (Bactec Mycosis) were not used. Interferon gamma testing for mycobacteria was negative.

After needle biopsy samples for bacterial, fungal and mycobacterial cultures had been drawn an intravenous treatment regimen (cloxacillin 2 g every 4 h and oral levofloxacin 500 mg once daily) was instituted. Bacterial cultures of the needle biopsy specimen turned out negative as well as PCR testing for bacterial 16S ribosomal RNA. Importantly, a fungal culture of the needle biopsy specimen grew *Candida* species. Isolate identification by the Vitek 2 Yeast identification system (ID-YST, bioMérieux, Marcy-l´Etoile) showed *C. dubliniensis* (excellent confidence level, bionumber 6102546061125771). The isolate was sensitive to amphotericin B, fluconazole, flucytosine and voriconazole. Antibacterial treatment was stopped and intravenous liposomal amphotericin B at a dose of 3 mg/kg was started on day 35. After 4 weeks the liposomal amphotericin B was discontinued and the patient was put on oral fluconazole 400 mg once daily. After 2 months of the start of antifungal therapy, on day 92, a lumbosacral MRI showed decreased edema in the LV vertebra, presacral disc space and prespinal soft tissues. After 5 months of antifungal therapy, on day 190, MRI showed only minimal residual edema and the dose of fluconazole was decreased to 200 mg once daily. Fluconazole was stopped after a total duration of 32 weeks of antifungal treatment. Three months later, the patient was symptomless and ESR was 2 mm/h and CRP 4 mg/l.

3. Discussion

Invasive fungal infections including nosocomial bloodstream infections due to *Candida* species have increased significantly over the last years. *Candida* infections are common among immunocompromised patients and intravenous drug users, but spondylodiscitis caused by *Candida* species is infrequent. Over the past 47 years 94 cases of *Candida* spondylodiscitis (the vast majority of them caused by *Candida albicans*) have been reported. Miller and coworkers (1966–2000) and Moon and coworkers (2006) found together 82 cases [3,6]. We searched the published literature through the PubMed for *Candida* spondylodiscitis from 2007 through 2012 and found 12 additional cases.

Candida spondylodiscitis is a uniquely rare condition and a high level of clinical suspicion is needed to identify it among patients presenting with low back pain. Important clues are the presence of risk factors for fungal infections and a history of preceding