CASE REPORT

Spondylodiscitis presenting as pleural effusion in a geriatric female: A case report

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Abstract

Pleural effusion is a frequently seen medical problem caused by pulmonary and non-pulmonary diseases. Spondylodiscitis is a very rare cause of pleural effusion and is typically diagnosed based on clinical, laboratory, microbiological and radiological findings. The low incidence and different clinical presentations of Spondylodiscitis make its diagnosis and treatment challenging. We present the case of a 78-year-old female who was initially admitted due to chest pain and upon chest radiography, was found to have pleural effusion; and eventually diagnosed with spondylodiscitis.

Keywords: Spondylodiscitis, exudative pleural effusion, geriatrics, vertebra, infection

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Introduction

Pleural effusion is a widespread medical problem and has more than 50 known causes, including pulmonary and non-pulmonary diseases. The most common causes of exudative pleural effusion are malignancies, pneumonia and tuberculosis.[1] Spondylodiscitis is a very rare cause of pleural effusion.[2] In such a case, the diagnosis of spondylodiscitis is often delayed, since the initial diagnostic tests for pleural effusion typically focus on pleuropulmonary diseases. Early diagnosis and treatment are critical in the management of spondylodiscitis and its consequent critical, fatal complications.[3-5] Therefore, spondylodiscitis must be considered in the differential diagnosis of pleural effusion. Here, we present the case of a geriatric female who came with complaint of chest pain, was noted to have pleural effusion upon chest radiography, and was eventually diagnosed with spondylodiscitis.

Case Report

A 78-year-old woman presented with right-sided chest pain and dyspnoea for five days to the emergency service of Yedikule Chest Diseases and Thoracic Surgery Training and Research Hospital in May 2017. She had a two-month history of back pain and was admitted for physical therapy owing to these complaints. She was referred to our hospital two days later because of right-sided pleural effusion, as revealed by chest radiography (Figure 1). She also had a history of ischaemic heart disease, hypertension and type 2 diabetes mellitus. She had smoked for 50 packs/years and consumed 200 ml alcohol per day four days a week. Her body temperature was 37°C, breathing sounds were absent in the right basal hemithorax, her leukocyte count was normal, C-reactive protein (CRP) level was 412.4 mg/dL and erythrocyte sedimentation rate (ESR) was 122 mm/h. Her arterial blood gas pH was 7.47, partial oxygen pressure was 65 mmHg and partial carbon dioxide pressure was 29 mmHg. Her D-dimer level was increased at 6.78 mg/L (0–0.060), while blood and urine cultures were negative. Electrocardiography and echocardiography revealed normal findings. Levofloxacin (500 mg/day, intravenously) and Enoxaparin 0.6 mL 2 × 1 (subcutaneously) were started empirically. Chest computed tomography (CT) angiography ruled out pulmonary embolism; however, right-sided pleural effusion and degenerative changes in bone structures were noted. Thoracentesis yielded a fluid exudate (pleural fluid pH 7.26, the pleural fluid-to-serum albumin ratio >0.5, the pleural fluid-to-serum LDH ratio >0.6) with an adenosine deaminase level of 10.4 IU/L, a lymphocyte percentage of 40% and a neutrophil percentage of 60%. No acid-fast bacilli and malignant cells were detected in the pleural fluid. The pleural fluid cultures grew Methicillin-sensitive Staphylococcus aureus.

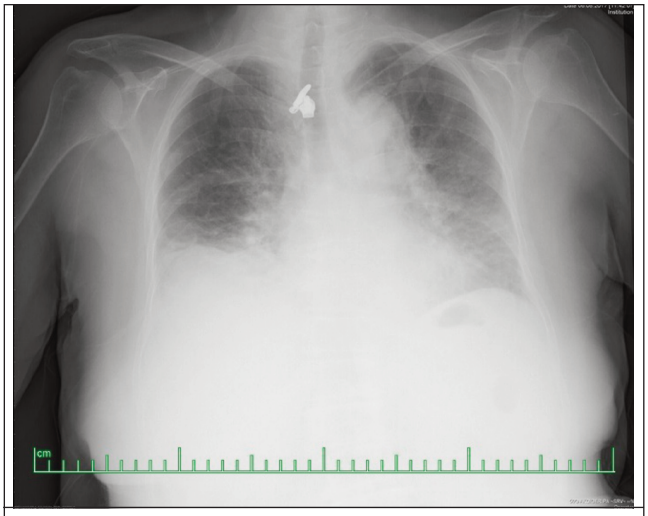

Figure-1: The initial chest radiograph showing pleural effusion on the right side.

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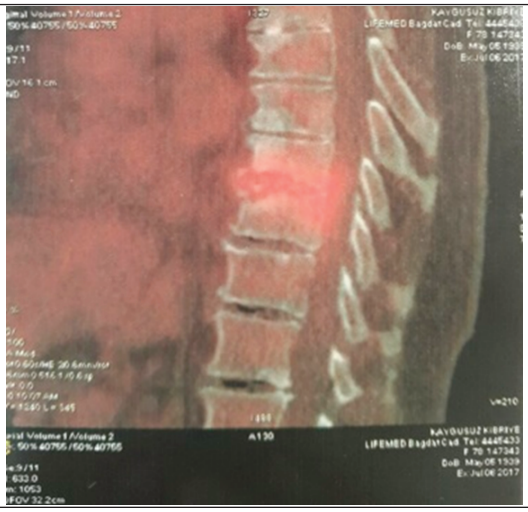

Figure-2: PET/CT showing high FDG uptake in the right paravertebral region accompanied by heterogeneous density in the vertebral endplates at the T9-T10 vertebral level.

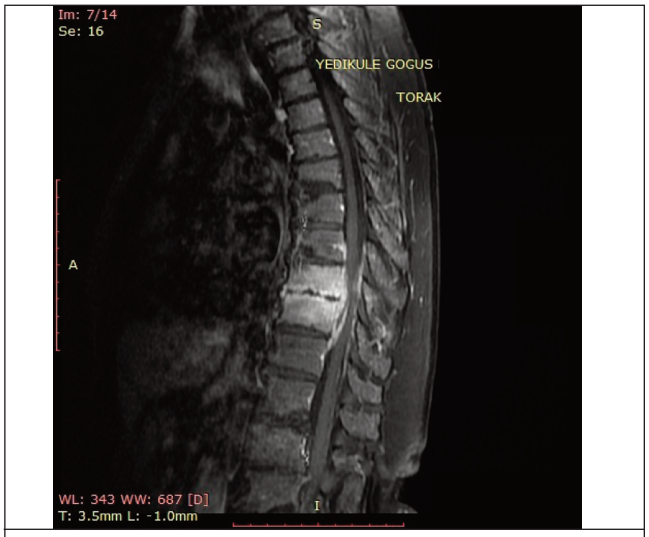

Figure-3: Post-contrast MRI of the spine showing contrast enhanced at the level of T9-T10 and loss of disc space height.

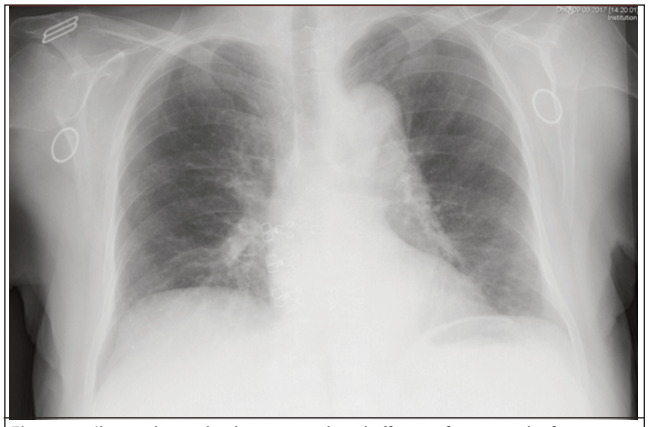

Figure-4: Chest radiography showing no pleural effusion after 1 month of treatment.

Treatment was modified to Levofloxacin (500 mg/day, intravenously) and Piperacillin-Tazobactam (3 × 4.5 g/day, intravenously) for 14 days. On partial regression of pleural effusion the patient was discharged. One week later, the patient was readmitted to our clinic due to persistent back pain and right-sided pain. Physical examination revealed slightly diminished breathing sounds in the right basal hemithorax and localised tenderness in the right paravertebral area of the lower thoracic spine. Her ESR was 114 mm/h and CRP level was 182 mg/dL, and thoracic ultrasonography revealed a 2.5-cm-thick pleural effusion in the right pleural space. The blood and urine cultures were still negative. Thoracentesis yielded haemorrhagic pleural fluid with a lymphocyte percentage of 80% and a neutrophil percentage of 20%. The pleural fluid cultures were negative for bacteria, acid-fast bacilli and malignant cells; however, malignancy was suspected because of severe pain and haemorrhagic pleural effusion. 18F-fluoro-D-deoxyglucose (FDG) positron emission tomography (PET)/CT revealed high FDG uptake in the right paravertebral region accompanied by heterogeneous density in the vertebral endplates at the T9-T10 vertebral level (maximum standardised uptake value is 10.3) indicating spondylodiscitis (Figure 2). The diagnosis was confirmed using magnetic resonance imaging (MRI) (Figure 3). The patient was treated with Fusidate Sodium (3 × 500 mg/day, orally) in combination with Teicoplanin (400 mg/day, orally) for six weeks, Hyperbaric oxygen therapy and spinal immobilisation using a thoracolumbar corset. A month later, the pleural effusion had resolved completely (Figure 4), CRP was 36 mg/dL and ESR was 57 mm/h. After the resolution of pleural effusion, the patient was followed by a neurosurgeon for one year for the possibility of recurrence. Control MRI was unnecessary because of improvement of inflammation markers as well as clinical symptoms.

Discussion

Spondylodiscitis is an infrequent and serious infection of the intervertebral disc and the adjacent vertebrae, usually seen in adults in their fifth to seventh decades of life.[6] Spondylodiscitis affects more men than women[4,5] and has a prevalence of 4.8/100,000 individuals per year.[4] Advanced age, diabetes mellitus, immunosuppressive medication, intravenous drug use, surgical interventions, urinary tract infections, infective endocarditis, human immunodeficiency virus infection, malnutrition, malignancy, heart diseases, alcoholism, chronic hepatitis and renal failure are the principal risk factors.[3,6] The prevalence is constantly increasing due to an increase in intravenous drug users, haemodialysis patients, immunocompromised hosts[3,4] and geriatric population.[5,6]

This patient is an atypical case of spondylodiscitis and was initially misdiagnosed. Advanced age, diabetes mellitus and chronic alcoholism were risk factors for the development of spondylodiscitis in the present case. Pathogens affect the spinal column through haematogenous, external inoculation or contiguity. Haematogenous spread from a distant focus (urinary tract infection, infective endocarditis, skin and soft tissue infection) is the most common route of infection.[3,6] S. *aureus* is the most frequent causative pathogen.[3,5,6] Back pain, fever, paravertebral muscle tenderness and spasms are the predominant symptoms and physical findings.[3] Based on the causative pathogen and the affected spine areas, spondylodiscitis may clinically manifest in various forms, such as pleural effusion, empyema,[2] confusion, meningitis and tetraparesis;[7] furthermore, it may mimic other diseases, such as lymphoma, hepatic or biliary diseases.[8] This patient initially presented with chest pain and pleural effusion and spondylodiscitis was masqueraded. Leucocyte count may be normal or elevated, but ESR and CRP levels are typically elevated.[3] Microbiological tests are essential for choosing the appropriate therapeutic agent. In approximately half of the patients, blood culture is the simplest method of identifying the causative pathogens.[6] If blood culture is negative and no clinical response to treatment is noted, CT-guided biopsy of the intervertebral disc is recommended;[3] however, this strategy may delay the treatment. According to the clinical presentation, the causative pathogen can be determined in the urine or pleural fluid[2,9] and intervertebral disc biopsy may not be necessary. In the present case, *S. aureus* was detected in the pleural fluid and treatment was initiated for this pathogen. The mean diagnosis time is 2–4 months.[3] In this patient, the initial investigation was focussed on the lungs because of pleural effusion which delayed the actual diagnosis by 41 days. MRI is the most sensitive and specific radiological technique for diagnosis and CT plays only a minor role. When there is uncertainty in diagnosis, positron emission tomography/CT can provide additional information.[6] Treatment is based on antibiotic therapy but surgery may be necessary for some patients.[3,5] The optimal duration of antibiotic therapy is debatable. Six weeks of parenteral or highly bioavailable oral antimicrobial therapy seems adequate for most patients; however, the duration should never be less than that.[3] Advanced age, infections with Methicillin-resistant S. aureus, diabetes mellitus, renal failure or paravertebral abscesses are the risk factors for recurrence.[10] In this patient, although the initial antibiotic of choice was appropriate, the short treatment duration, advanced age and diabetes mellitus may have led to treatment failure. It may be questionable whether pleural effusion or spondylodiscitis has an initiation role. Probably the primary source of pleural effusion is spondylodiscitis in this patient. This is supported by clinical course and follow-up period of the patient — initially, she had back pain two months before right-sided pain, then pleural effusion was exudate from the beginning and empyema was never observed. And lastly, since the focus was on pleuropulmonary diseases, routine CT scans of the thorax, which was only one centimetre slice thickness, were initially performed and no skeletal windows or level adjustments were used at the first admission of the patient. Therefore, at that time, changes in vertebral bodies could not be clearly evaluated and spondylodiscitis was masqueraded.

Conclusion

The diagnosis of spondylodiscitis is typically difficult. Infrequency of disease, nonspecific symptoms and widespread back pain in geriatric population often lead to delayed or incorrect diagnosis. Furthermore, the manifestation of the disease with atypical clinical symptoms and findings makes the diagnostic process extremely complicated, as it happened in this case. Although spondylodiscitis is rare, it should be considered in the differential diagnosis of pleural effusions, especially in elderly patients with risk factors and suffering from back pain.

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