

## INFECTIVE ENDOCARDITIS WITH SPONDYLODISCITIS AFTER PROSTATE BIOPSY

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Clin Biomed Res. 2014;34(1):80-83

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### ABSTRACT

Transrectal ultrasound-guided prostate needle biopsy is the ideal method of obtaining prostate specimens for histological analysis and is therefore often used in clinical practice. In most studies, prostate biopsy is considered a safe procedure with few major complications. In the present report, we describe a case of endocarditis with spondylodiscitis, two very rare complications associated with prostate biopsy.

In the present report, we describe a case of infective endocarditis (IE) with spondylodiscitis (SD), two very rare complications of prostate biopsy. Only a few cases have been described in the literature reporting IE or SD as complications of prostate biopsy.

**Keywords:** *endocarditis; discitis; prostate; biopsy; needle.*

A 75-year-old former smoker man presented with 2 days of sudden onset back pain. The pain worsened with spinal mobilization and impaired his mobility, partially improving with rest. The patient did not report any associated paresis, paresthesia or prior history of back pain.

Past medical history included diabetes, hypertension, ischemic stroke with no significant sequelae, and benign prostatic hyperplasia. The patient had undergone a prostatic biopsy 2 weeks before the onset of symptoms due to an increase in the prostate-specific antigen (19.2ng/mL), which was negative for neoplasia. He had received antibiotic prophylaxis with ciprofloxacin (oral, 500mg twice daily for 3 days, starting the morning of the procedure).

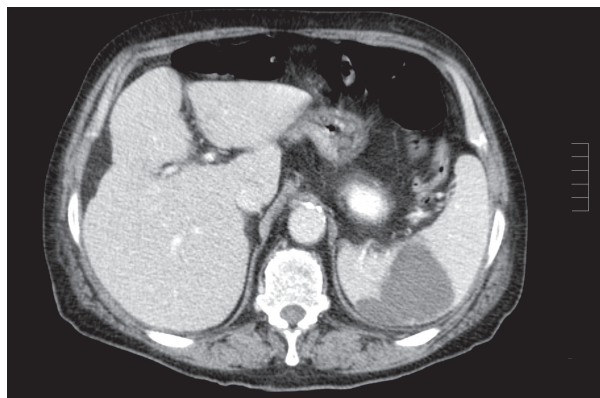
Physical examination showed pale mucous membranes, high fever (38.1°C), and tachycardia (heart rate 102bpm). There was no other significant finding. Lower back was tender on palpation.

Admission lab tests showed anemia (hemoglobin 9.8g/dL), leukocytosis (19,750/L, with 10% of bands), elevated c-reactive protein (CRP) levels

(265mg/dL, normal value: < 5mg/dL) and high erythrocyte sedimentation rate (ESR) (82mm/h, normal value: < 30mm/h). Urine sediment was normal and urine culture was negative for bacteria. Signs of chronic obstructive pulmonary disease and small bilateral pleural effusions were observed on a chest x-ray. A lumbosacral column x-ray showed only degenerative changes.

A computed tomography (CT) scan of the lumbosacral spine was performed and showed vertebral degenerative changes and an extensive splenic hypodense lesion (7.4 x 7.0cm) (Figure 1), suggestive of splenic infarction. Upon suspicion of SD, empirical antibiotic treatment was initiated (cefepime 2g three times a day plus vancomycin 1g twice daily). A magnetic resonance imaging (MRI) was performed and showed alterations suggestive of SD (Figure 2). In addition, blood cultures collected upon admission showed the growth of *Enterococcus* sp. (susceptible to ampicillin and vancomycin; resistant to high-level streptomycin and gentamicin), and antibiotic therapy was guided by the antibiogram (ampicillin 3g four times a day).

Based on these findings, diagnosis of IE was considered. A transesophageal echocardiography was carried out and revealed a vegetation of 1.6 x 1.0cm adhering to the aortic valve coronary leaflet, with leaflet perforation causing a mild to moderate eccentric regurgitation. The patient developed heart failure, requiring valve replacement, and completely recovered after four weeks of intravenous antibiotic therapy (standard treatment duration for native valve IE with negative valve culture). At 12-month follow-up, there were no signs of clinical or laboratory relapse.



**Figure 1:** Splenic cystic wedge-shaped lesion. There is peripheral enhancement of the splenic capsule. These findings suggest splenic infarction.



**Figure 2:** T2-weighted image shows hyperintense signal of the L5-S1 intervertebral disc, which is suggestive of SD. There are associated degenerative changes.

## DISCUSSION

Pyogenic SD is a primary infection of the nucleus pulposus of the intervertebral disc (10). In most SD cases, the primary route of infection seems to be hematogenous spread secondary to bacteremia (15). Due to the avascular nature of the intervertebral disc space in adults, SD is rarely observed (16), with incidences ranging from 0.4 to 2.4 per 100,000 individuals each year (10).

Often there is a relatively recent history of an infective focus elsewhere, usually treated with antibiotic therapy. Recent history of an invasive procedure or surgery is important (3), as was the case with our patient. Although clinical presentation of SD varies, it typically presents as insidious onset of localized back pain combined with non-specific symptoms (17).

Laboratory findings may vary depending on the grade and causative agent, often revealing elevated ESR, white blood cell count and CRP values (3). Blood cultures should be collected on admission, and ideally a CT-guided biopsy of the infected disc should be performed in order to confirm diagnosis, identify the causal agent, precisely determine antibiotic sensitivity, and initiate the appropriate treatment (18). Biopsy was not performed in our patient, and diagnosis was made based on the

whole clinical picture. Diagnosis of SD can be confirmed by a bone scintigraphy, CT scan or MRI, with the latter being the most sensitive technique in the acute phase (19). Contrast enhancement is the earliest sign and pathognomonic in the acute inflammatory episode (3).

Treatment includes conservative treatment and surgery. Antibiotics and immobilization constitute the conservative treatment. Surgery may be indicated for spinal cord and radicular compression, biomechanical instability, severe persistent pain, or abscess (3). Antimicrobial therapy alone is appropriate for most cases (20). Empirical therapy should cover *Staphylococcus aureus* and Gram-negative organisms, taking into account local susceptibility rates and the likelihood of colonization with resistant organisms (21). Most authors recommend 4 to 6 weeks of intravenous therapy followed by 3 to 6 months of oral therapy (20). The overall rates for mortality and recurrence of infection have been reported as 2-11%. Recurrence is usually within six months, rarely up to one year (3).

SD may sometimes be the presenting sign of an IE. Early diagnosis of IE as the source of the SD is often difficult because clinical and radiologic patterns are similar to those present in SD alone. In all patients with SD, IE should be excluded, particularly in patients with a history of heart valve disease (22).

The frequency of this association is still controversial, ranging from 0.6-2.2% (23,24) when patients with known SD were screened for IE to 10-15% (22,26) when patients with known IE were screened for SD in retrospective studies. The most common clinical picture of this association is musculoskeletal symptoms preceding diagnosis of IE (19).

Except for duration of treatment and hospitalization, a previous study (26) did not find any significant differences between IE with and without SD, especially regarding patient age and gender, clinical and laboratory features, and evolution of disease. The prolonged treatment of patients with IE and SD can be explained by the lengthy treatment of SD. Although prognosis depends mainly on the valvular disease, appropriate determination of this association has important consequences, e.g., need for a longer antibiotic therapy course and a correct interpretation and target investigation of persistent elevated inflammatory markers (19).

We presented a case of SD with IE following a prostate biopsy. It was successfully treated with four-week ampicillin monotherapy and valve replacement. SD and IE together are a very rare complication of this procedure, and both diagnoses should be considered even when only one of them presents symptoms.

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Received: 15/12/2013

Accepted: 05/02/2014