

The First Case of Psoas Muscle Abscess and Sepsis Caused by *Actinobacillus ureae* in a Chronic Hepatitis B Patient in Korea

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Actinobacillus ureae, formerly known as *Pasteurella ureae*, is a rare human pathogen. Twenty-eight cases of *A. ureae* infections in humans have been reported since its first description in 1960. Various predisposing conditions such as skull fracture, alcohol abuse, neurosurgery, schizophrenia, odontal infection, diabetes, HIV infection/AIDS, Waldenström macroglobulinemia, COPD, malnutrition, rheumatoid arthritis, HCV hepatitis, etanercept, or methotrexate have been associated with infections caused by *A. ureae*. We report the first case, in the medline-based literature, of *A. ureae* psoas muscle abscess and sepsis in a HBV carrier patient.

Key Words : Psoas abscess, Chronic hepatitis B, Sepsis

INTRODUCTION

Actinobacillus (Pasteurella) ureae is a commensal of the human respiratory tract and various mammals. It is an organism of low pathogenicity and is a rare human pathogen (1). Only 28 cases of *A. ureae* infections in humans have been reported since its first description in 1960, including meningitis (2), pneumonia (3-6), hepatitis (7), sepsis (7,8), conjunctivitis (9), otitis media (10), peritonitis (11), endocarditis (12), chronic bronchitis (13), bone marrow infection (14) and septic arthritis (15). Here, we report the first case, in the medline-based literature, of *A. ureae* psoas muscle abscess and sepsis in a HBV carrier patient.

CASE REPORT

A 76-year-old male was admitted to the department of internal medicine with fever, lower back pain, and general

weakness after suffering from a slip-down injury 25 days before admission while working at his farm. His past medical history was positive for chronic hepatitis B infection, stroke, and hypertension. He had undergone appendectomy and hydrocelectomy. However, he was not a smoker nor drank alcohol.

On initial physical examination, he was acutely ill looking and showed confused mentality. His body temperature was 38.1°C, blood pressure was 140/80 mmHg, pulse rate was 96 beats/min, and respiration rate was 20 breaths/min. Positive clinical findings included tenderness and swelling on the lower back. The chest X-ray was unremarkable. The abdominal CT showed L4 vertebral body compression fracture with both psoas muscle abscess (right: 2.7×1.7×1.3 cm, left: 4.3×2.1×2.3 cm) (Fig. 1). Laboratory results were as follows: normochromic normocytic anemia (Hb = 10.2 g/dL, Hct = 30.0%); white blood cell count (WBC), 14.21×10⁹/L (polymorphs, 76.9%; lymphocytes, 9.6%; monocytes, 11.2%; eosinophils; 0.8%); platelet, 427×10⁹/L; erythrocyte sedimentation rate (ESR), 85 mm/h; C-reactive protein (CRP), 12.65 g/L; AST, 279 IU/L; ALT, 142 IU/L; ALP, 198 IU/L; total bilirubin, 0.8 mg/dL; prothrombin time, 20.6 sec (INR=1.88). Other blood biochemistry tests were normal (Table 1). His HBsAg was posi-

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tive and HBV-DNA was 393,900 copies/mL. The patient was initially treated with intravenous ceftazidime and metronidazole. On the 7th hospital day, the fusion operation of L4-5 vertebral body and incision and drainage was performed. The blood culture was positive for *A. urea*, which was susceptible to penicillin, amikacin, ampicillin, aztreonam, cefazolin, cefotaxime, ciprofloxacin, gentamicin, and cotrimoxazole. Antibiotics were changed to ceftazidime. Echocardiography showed no vegetation on cardiac valves. A few days after treatment, fever subsided and inflammatory indices such as ESR, CRP, and platelets re-

turned to normal values. Follow up abdominal CT scan after 25 days of treatment showed reduced size of right psoas abscess to 1.0×2.3×1.5 cm and disappeared left psoas abscess. A repeated blood culture performed on 14 days and 30 days after admission revealed no growth. After 30 days of intravenous antibiotic therapy, he was discharged with oral cefixime for 2 weeks. He was afebrile on control visit in outpatient clinic after 2 weeks.

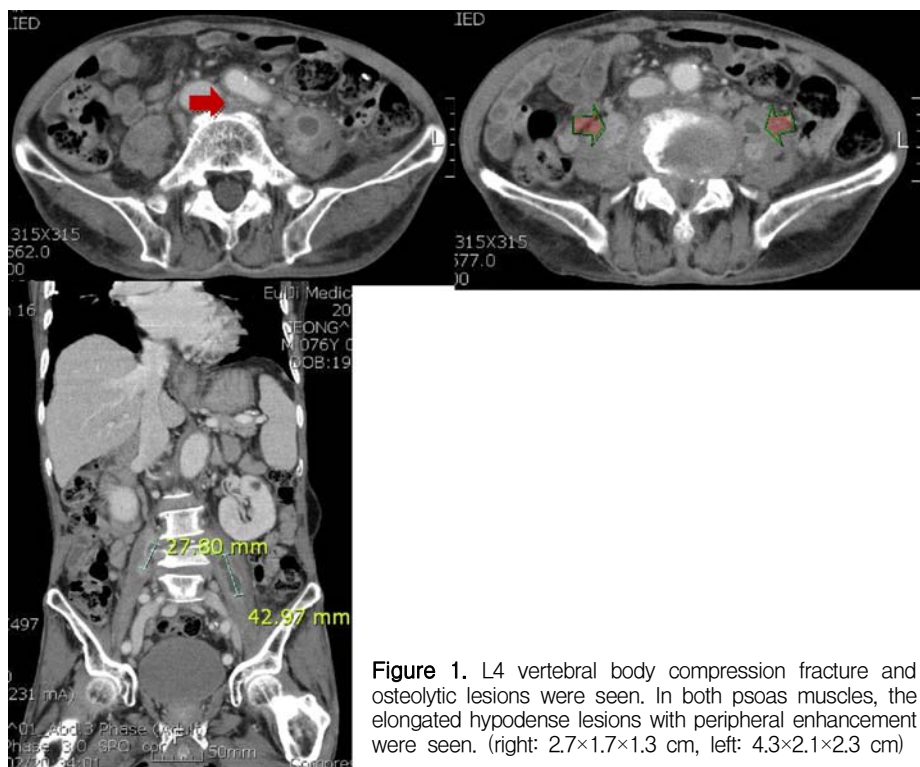


Figure 1. L4 vertebral body compression fracture and osteolytic lesions were seen. In both psoas muscles, the elongated hypodense lesions with peripheral enhancement were seen. (right: 2.7×1.7×1.3 cm, left: 4.3×2.1×2.3 cm)

Table 1. The Laboratory findings of the Patient

Laboratory findings	Hospital day 1	Hospital day 54
White blood cell ($\times 10^9/L$)	14.21	7.09
polymorphs (%)	76.9	71.2
lymphocytes (%)	9.6	13.8
monocytes (%)	11.2	8.5
eosinophils (%)	0.8	0.7
Platelet ($\times 10^9/L$)	427	334
Erythrocyte sedimentation rate (mm/hr)	85	10
C-reactive protein (g/L)	12.65	0.5
AST/ALT (IU/L)	279/142	56/25
ALP (IU/L)	198	95
Total bilirubin (mg/dL)	0.8	0.9
Prothrombin time (sec)	20.6 (INR=1.88)	12.9 (INR=1.04)

or impossible to culture in vitro. In addition, analysis of DNA sequences has been used to name organisms that were unidentifiable by phenotypic testing (20).

Of the documented cases in the literature, this is the first case, to our knowledge, of a serious *A.ureae* infection associated with psoas muscle abscess that has been documented in HBV carrier without progression to liver cirrhosis. When a patient with psoas muscle abscess is found, the possibility that uncommon organism could be the cause of infection should always be seriously considered and effort should be made to identify species and obtain antibiotic susceptibility. This will lead to early initiation of appropriate antibiotic therapy resulting in successful treatment outcome.

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