

A definite case of spondylodiscitis caused by *Streptococcus equisimilis*

P. Richette¹, P. Pizzuti¹,
A. Quillard², L. Raskine³,
B. Naveau¹, F. Lioté¹

¹Fédération de Rhumatologie, Centre Viggo Petersen; ²Service Central d'Anatomie et Cytologie Pathologiques; ³Service de Microbiologie-Virologie, (AP-HP) Hôpital Lariboisière, Paris, France

P. Richette, MD; P. Pizzuti, MD;
A. Quillard, MD; L. Raskine, MD;
B. Naveau, MD; F. Lioté, MD, PhD.

Please address correspondence to:
Prof. Frederic Lioté, Fédération de
Rhumatologie, Hôpital Lariboisière,
2 rue Ambroise Paré, 75010 Paris, France.
E-mail: frederic.liote@lrh.ap-hop-paris.fr

Received on October 25, 2000; accepted
in revised form on May 17, 2001.

© Copyright CLINICAL AND
EXPERIMENTAL RHEUMATOLOGY 2001.

Key words: *Streptococcus equisimilis*,
spondylodiscitis, lumbar discitis,
infection.

ABSTRACT

To shed light on the role of *Streptococcus equisimilis* (SE) in the pathogenesis of intervertebral disc infection, we report here a case of lumbar spondylodiscitis in a 37-year-old male caused by SE, with identification of this strain by cultures from L4-L5 lumbar disc biopsy. Intravenous therapy with penicillin and gentamycin combined with immobilization resulted in a rapid and complete recovery. The patient did not have underlying disease and showed no obvious history of exposure to animals. We conclude that SE may be responsible for both septic arthritis and spondylodiscitis.

Introduction

Few cases of definite septic arthritis (1) and two possible cases of spondylodiscitis due to SE (2, 3) have been described so far. However, this microorganism has never been identified from the disc tissue. We report in a 37-year-old male the first case of spondylodiscitis caused by SE with identification of this strain by cultures from L4-L5 lumbar disc biopsy.

Case report

A 37-year-old white male was admitted to hospital on December 1996 for L4-L5 spondylodiscitis. His past history included episodes of acute lumbar pain in the 5 past years. He had returned to France after working for 5 years in Malaysia. His symptoms started 4 months later, with low back pain that appeared gradually without any traumatism. Pain was initially permanent and did not improve with analgesics and rest. It was associated with high fever (40°C) and shivering. He was first admitted to an internal medicine department on November 1996. Physical examination disclosed a severe stiffness of the lumbar spine. There was no sciatica nor any infectious signs at complete physical examination. Laboratory data showed an erythrocyte sedimentate rate (ESR) of 16 mm/h and a C-reactive protein (CRP) level of 40 mg/l (N < 5). Blood and urine cultures remained negative. X-rays of the lumbar spine were normal. Low back pain as well as CRP level decreased gradually with rest and

analgesics. He was not given any non-steroid anti-inflammatory or antibiotic treatment. A diagnosis of acute low back pain with concomitant viral infection was made and the patient was discharged one week later.

However, low back pain flared up again with a temperature of 38°C. Few days later, he visited a clinic for a second opinion. ESR has increased to 40 mm/h. Magnetic resonance imaging (MRI) of the lumbar spine showed a spondylodiscitis pattern at L4-L5 level with an anterior epiduritis. He was admitted in our department for further investigations and treatment. Physical examination disclosed a normal temperature but marked stiffness of the lumbar spine. The remainder of the complete physical examination was normal. Significant laboratory findings included: ESR 36 mm/h, CRP 40 mg/l, and white blood cell count 8,600/mm³ with 68 % neutrophils. Blood and urine cultures again tested negative. Chest X-rays and dental X-rays were normal. A test for HIV infection was negative. Cardiac echography showed normal valves. Roentgenograms of the lumbar spine revealed an erosion of the postero-superior corner of L5 without any narrowed disc spaces or abnormality in the soft tissues. A ^{99m}technetium bone scan disclosed increased uptake at the L4-L5 level.

Examination of a needle biopsy of the L5 vertebra confirmed the diagnosis of spondylodiscitis and histopathological examination of a stained specimen revealed many Gram positive cocci (Fig. 1). Finally, a group C *Streptococcus* typed as *Streptococcus equisimilis* was identified in the vertebral disc culture after 24 hours. The patient reported no exposure to animals.

Treatment was started with amoxycillin and gentallin given for 10 days intravenously (IV) followed by amoxycillin (IV) alone for 2 weeks, then oral penicillin for 8 additional weeks. During treatment, the patient was immobilized in dorsal decubitus. He made a rapid recovery and became pain-free after 4 weeks of treatment. After one month of antibiotics, control X-rays showed no further changes, and especially no destruction of the disc. One year after the

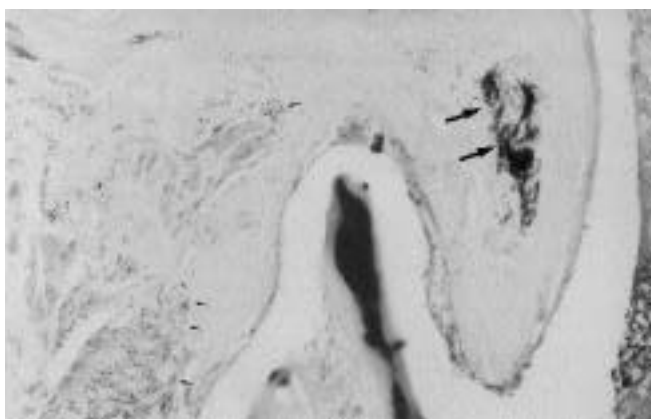


Fig. 1. Intervertebral disc biopsy specimen with Brown and Brenn staining (original magnification x125). Large amounts of Gram positive cocci (dark arrows) can be seen within a discal fibrocartilage tissue with mild alterations.

discontinuation of therapy, the patient was still asymptomatic.

Discussion

We describe the first case of infectious spondylodiscitis due to *Streptococcus equisimilis* (SE) in which the specific microorganism was identified both by staining and culture of specimens from the intervertebral disc. SE is a strain Gram positive cocci classified as a beta haemolytic streptococci of the Lancefield group C (4). Traditionally, group C Streptococci are divided into four species (*S. equi*, *S. zooepidermicus*, *S. dysgalactiae* and *S. equisimilis*) which can be differentiated by their biochemical properties (5). They are a common cause of infections in animals, but are rarely pathogenic in humans (6). These organisms can be identified in the throat, skin and genital tract of healthy adults, especially in Europe and North America (7). The pharynx is a common reservoir.

Streptococci cause approximately 15% - 20% of all cases of non-gonococcal septic arthritis. Group A Streptococcus is the type most frequently isolated (8). In group C Streptococcus septic arthritis, almost every joint can be involved and the infection is frequently polyarticular (5, 9). Steinfeld *et al.* (1) reviewed the literature on joint infections caused by SE and found only 8 cases - involving the knee (n=2), the ankle (n=2), the hip (n=1), the acromioclavicular joint (n=1) and two polyarticular infections. In these 8 cases, SE

was isolated in the synovial fluid. Moreover, in 5 out of the 8 cases blood cultures were positive. In 3 cases immunodeficiency was observed (breast cancer, carcinoma of penis associated with diabetes, AIDS and corticosteroids). Furthermore, Parola *et al.* (10) recently reported another case of SE septic arthritis of an ankle.

Asplin *et al.* (3) reported a case of bacteremia due to a Streptococcus C identified as SE complicated one month later by a C6-C7 spondylodiscitis which was presumed to be due to the same strain, but needle biopsy was not performed in this case. Recently, a case of T8-T9 spondylodiscitis caused by SE was reported (2) in an 83-year-old man with diabetes mellitus, although the cultures of the specimens sampled from the disc space remained sterile. The strain was isolated from blood cultures and the source of infection was assumed to be the oral cavity. These two cases share with our report the same typical spondylodiscitis clinical features. However, an important difference is the absence of narrowed disc space on X-rays in our case. Bradley *et al.* (6) reviewed 88 cases of group C streptococcal bacteremia and found no source of infection in 38%. In the other cases, the onset of infection was observed in the upper respiratory, urinary tracts, intestinal tracts and the skin. The majority of infections was community acquired. A contact with animals was noted in 25 % of the cases.

Spondylodiscitis caused by group C

Streptococci are extremely rare, especially those due to SE. This bacteria does not appear to affect joints with pre-existing rheumatological conditions (only 1 case out of 8 in the series of Steinfeld *et al.*) but the immune status of the patient could play a role in the onset of arthritis (1). The source of infection is often unknown, as observed in our case.

In most cases, these strains are sensitive to penicillin. Although penicillin G is the most active antibiotic, group C Streptococci are also sensitive to cephalosporins, vancomycin and erythromycin. The combination of gentamycin and beta-lactams or vancomycin results in bactericidal activity (5, 7).

References

1. STEINFELD S, GALLE C, STRUELENS M *et al.*: Pyogenic arthritis caused by *Streptococcus equisimilis* (group-C streptococcus) in a patient with AIDS. *Clin Rheumatol* 1997; 16: 314-6.
2. GOMEZ-RODRIGUEZ N, FERREIRO-SEOANE JL, IBANEZ-RUAN J, SEVILLANO-CASTANO J: Spondylodiscitis caused by *Streptococcus equisimilis*. *Br J Rheumatol* 1998; 37: 1030-2.
3. ASPLIN CM, BEECHING NJ, SLACK MP: Osteomyelitis due to *Streptococcus equisimilis* (group C). *Br Med J* 1979; 13: 89-90.
4. ROTT J: Pyogenic hemolytic streptococci. In SNEATH PHA, MAIR NS, SHARPE ME, HOLT JG (Eds): *Gergey's Manual of Systematic Bacteriology*, Vol. 2, Baltimore, Williams and Wilkins 1986, 1047-54.
5. ORTEL TL, KALLIANOS J, GALLIS HA: Group C streptococcal arthritis: Case report and review. *Rev Infect Dis* 1990; 12: 829-37.
6. BRADLEY SF, GORDON JJ, BAUMGARTNER DD, MARASCO WA, KAUFFMAN CA: Group C streptococcal bacteremia: Analysis of 88 cases. *Rev Infect Dis* 1991; 13: 270-80.
7. SALATA RA, LERNER PI, SHLAES DM, GOPALAKRISHNA KV, WOLINSKY E: Infections due to Lancefield group C streptococci. *Medicine* 1989; 68: 225-39.
8. GOLDENBERG DL: Bacterial arthritis. *N Engl J Med* 1985; 21: 764-71.
9. COLLAZOS J, ECHEVARRIA MJ, AYARZA R, DE MIGUEL J: *Streptococcus zooepidemicus* septic arthritis: Case report and review of group C streptococcal arthritis. *Clin Infect Dis* 1992; 15: 744-6.
10. PAROLA P, BROUQUI P, MAURIN M, BOURGEADE A: A new case of *Streptococcus equisimilis* septic arthritis. *Clin Rheumatol* 1998, 17: 71-2.