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LATE-ONSET SPINAL IMPLANT INFECTION DUE TO STREPTOCOCCUS SALIVARIUS PROBABLY TRIGGERED BY A BLOOD SPLASH DURING A DENTAL PROCEDURE

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ABSTRACT

Introduction: Spinal implant infections (SIIs) are a rare but well-known complication of spinal surgery. Early SIIs are usually caused by *Staphylococcus aureus*, β-haemolytic streptococci, or Gram-negative bacilli. On the other hand, late-onset SIIs are usually caused by coagulase-negative staphylococci or *Cutibacterium acnes*.

Case description: Here, we present a rare case of a late-onset SII in a 24-year-old dentist, caused by oral streptococcal flora *(Streptococcus salivarius)* that was probably inoculated into his conjunctival mucous membrane by a blood splash during a dental procedure, seven years after spinal fusion. He was initially treated broadly with empiric antibiotics as the organism was not identified, until it was later isolated from an intraoperative tissue culture. After that, he was given targeted antimicrobial therapy along with implant removal.

Discussion: The conjunctival mucosa is a well-established route of entry for blood-borne pathogens, particularly viral organisms, in health-care settings.

Conclusion: This treatment strategy resulted in significant improvement of his symptoms and general condition.

KEYWORDS

Streptococcus salivarius, spinal implant infection, occupational exposure, blood splash, dentists

LEARNING POINTS

- In culture-negative or late-onset implant infection, a deliberate occupational and bloodborne-exposure history can reveal a source that cultures do not.
- Prior antimicrobial exposure markedly lowers culture yield and withholding antibiotics before intra-operative, or aspiration sampling can be decisive.
- Eye protection is essential for dental and other healthcare workers, as the conjunctiva is a viable portal for bacterial as well as viral transmission.

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INTRODUCTION

SIIs are usually classified by when they occur relative to surgery: early-onset SIIs occur within 30 days, while chronic or late-onset SIIs develop after 30 days[1–3].

SIIs can be caused by various organisms, mainly depending on when the infection occurs. Although the causative organisms may overlap, early-onset infections are usually caused by *Staphylococcus aureus*, β-hemolytic streptococci, and aerobic Gram-negative bacilli. By contrast, late-onset infections can be caused by coagulase-negative staphylococci and *Cutibacterium acnes* (formerly *Propionibacterium acnes*)[1]. These infections may result from direct contamination during surgery or, less frequently, from bloodstream spread, especially with organisms such as *S. aureus* and *Streptococcus spp.*[4].

A large proportion of late-onset infections do not yield organisms on culture. An analysis of 45 cases of late-onset implant-associated infections following corrective surgery revealed that 82% of cases had no detectable microorganisms[5]. Furthermore, oral streptococci are infrequent causes of these infections in spinal implants, particularly in late-onset infections[6].

This report describes a complex late-onset spinal implant infection caused by oral *Streptococcus salivarius* in a dentist. The organism was inoculated into the eye by a blood splash during a dental procedure performed without eye protection. This case illustrates the diagnostic challenges and the importance of personal protective equipment (PPE) during dental procedures especially for healthcare workers with spinal implants.

CASE DESCRIPTION

A 24-year-old male with a previous history of thoracolumbar fusion surgery for congenital scoliosis, performed seven years prior, presented to the hospital complaining of a throbbing headache with photophobia for one week duration. Several days later, he developed subjective fever, chills and rigors along with severe upper and lower back pain associated with lower limb weakness. At that time, he presented to a peripheral hospital and was admitted for possible meningitis.

Soon after hospitalization, two sets of blood cultures were sent which turned out to be negative, and a cerebrospinal fluid (CSF) cell count revealed a white blood cell (WBC) count of 113 cells/µl, however, no CSF cultures were obtained at that time. He was started on ceftriaxone and vancomycin for the suspicion of meningitis and was transferred to our tertiary center for further management and care.

Upon admission to our hospital, he was hemodynamically stable and afebrile, with a temperature of 37°C, RR 19/min, BP 110/69 mmHg, SpO_2 98% on ambient air. He had full strength throughout except left-predominant weakness, left hip flexion 5-/5, left knee extension 4+/5. On back examination, he had a posterior midline surgical scar that is well-healed with paraspinal tenderness at the cervicothoracic and thoracolumbar junctions.

A repeat lumbar puncture revealed a CSF WBC count of 320/µl with a negative Gram stain and culture. His erythrocyte sedimentation rate (ESR) was 12 mm/h, C-reactive protein (CRP) 3.6 mg/l with all blood cultures being negative including dedicated *Brucella* cultures.

Post-contrast T1-weighted fat-suppressed magnetic resonance imaging (MRI) showed *(Fig.1)* a right-sided paraspinal collection at the cervicothoracic junction with another separate thoracolumbar paraspinal collection.

During this admission, he was started on meropenem with good clinical response, he also underwent ultrasound-guided aspiration of both the cervical collection and thoracic collection. Aspirate Gram stains and cultures were negative. He completed a six-week course of meropenem and was subsequently discharged on day

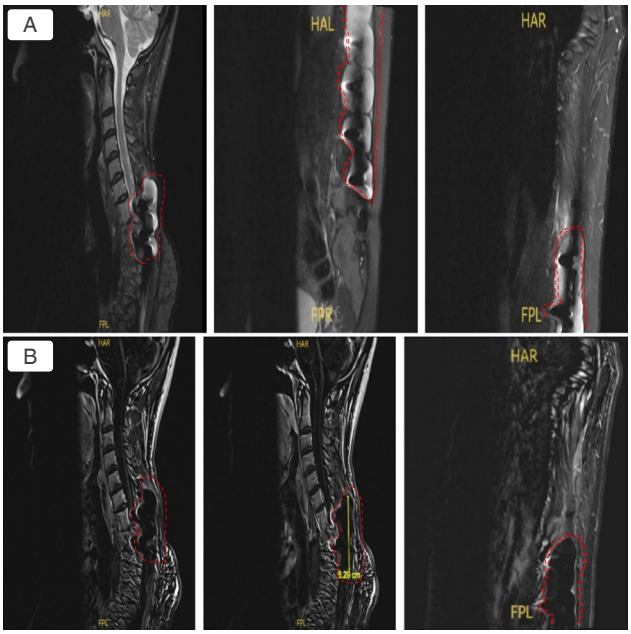


Figure 1. *Whole spine magnetic resonance imaging on initial presentation.*

A) Sagittal short tau inversion recovery (STIR) sequence: there are multiple multiloculated paraspinal fluid collections seen around the screws of the fixation device, mainly at the cervicothoracic and thoracolumbar junction regions, demonstrating slightly low signal intensity on T1 and high signal intensity on fluid-sensitive sequences (T2 and STIR).

B) Subtraction sagittal T1 turbo spin echo post-contrast sequence: the collections show peripheral thin enhancement on post-contrast sequences. On the axial planes, the largest collection at the cervicothoracic junction is seen mainly at the right side of C7 vertebral body level, measuring around 1.8 × 5.8 cm in maximum anteroposterior and transverse dimensions, respectively. The largest collection at the thoracolumbar junction is seen mainly at the right side of L2 vertebral body level, measuring around 3.3 × 5.2 cm in maximum anteroposterior and transverse dimensions, respectively. No definite intraspinal collections were identified. Red dashed outlines delineate the fluid collections.

46 on oral ciprofloxacin. Two weeks after discharge, he presented to his follow-up with the infectious disease clinic and complained of recurrent headache, back pain, and malaise. He also disclosed that he worked as a dentist and informed the treating team of his occupational exposure that happened seven days prior to the onset of initial illness, where he had a patient's blood splashed into his right eye during a dental procedure. This exposure caused transient conjunctival irritation without visual changes and resolved spontaneously. The source patient was not tested, and the incident was not reported to the infection control department. At the time of the incident, he was wearing gloves, a gown and a surgical mask but he was not wearing eye protection.

During this follow-up visit the decision was made for hardware removal given his recurrent symptoms, the newly disclosed blood-splash exposure, and repeated negative cultures on antibiotics, therefore, he was re-admitted.

On day one of the re-admission, he was started on empirical linezolid and metronidazole that was subsequently held after two weeks. The antibiotics were held for two weeks prior to hardware removal to maximize the yield of intra-operative cultures. During the antibiotic-free interval, ESR rose to 49 mm/h and CRP to 8.04 mg/l.

On day 29 of this admission, he underwent removal of all spinal hardware. Intra-operatively, serous fluid was noted, and tissue specimens were sent for culture and subsequently turned out to be negative. In the early post-operative period, he started to develop orthostatic headache that worsened with ambulation and was accompanied by neck stiffness. A whole-spine MRI was done and revealed paraspinal collections tracking along the prior screw trajectories at the cervicothoracic and thoracolumbar junctions with intraspinal extension at C7-T1 indenting the right dorsolateral thecal sac with a suspected dural defect.

On day 39, he underwent a dural repair surgery for the dural defect, with an active CSF leak identified intra-operatively at T1-T2 and was repaired. During the surgery, three CSF specimens and one implant-bed tissue specimen were collected intra-operatively. Gram stain revealed Gram-positive cocci in clusters from the CSF and revealed Gram-positive cocci in chains from the tissue sample. Two out of the three cultures grew *Staphylococcus haemolyticus* after more than 48 hours of incubation. This was considered a likely contaminant based on clinical and microbiological correlation. The tissue culture grew *S. salivarius*.

Both isolates were identified by VITEK MS matrix-assisted laser desorption/ionization-time-of-flight mass spectrometry (VITEK MS: bioMérieux SA, Marcy-l'Étoile, France). The *S. salivarius* isolate was susceptible to cefotaxime, ceftriaxone, and vancomycin, with intermediate susceptibility to penicillin.

Empirical broad-spectrum linezolid and meropenem were started for the post-operative clinical worsening which was continued after review of the microbiological findings. On day 54 the antimicrobials were narrowed down to ceftriaxone 2 g every 12 hours.

On day 63, he was discharged on oral cefuroxime 500 mg twice daily to complete a 12-week course of targeted therapy, counted from day 54. He was followed up in the infectious disease clinic at approximately five and 11 weeks after discharge, and at each visit he was asymptomatic with no new neurologic symptoms. Inflammatory markers normalized by the fifth week visit *(Fig. 2)*.

DISCUSSION

This case reports a late-onset spinal surgical implant infection due to *S. salivarius* that was probably transmitted to the patient via a blood splash while he was performing a

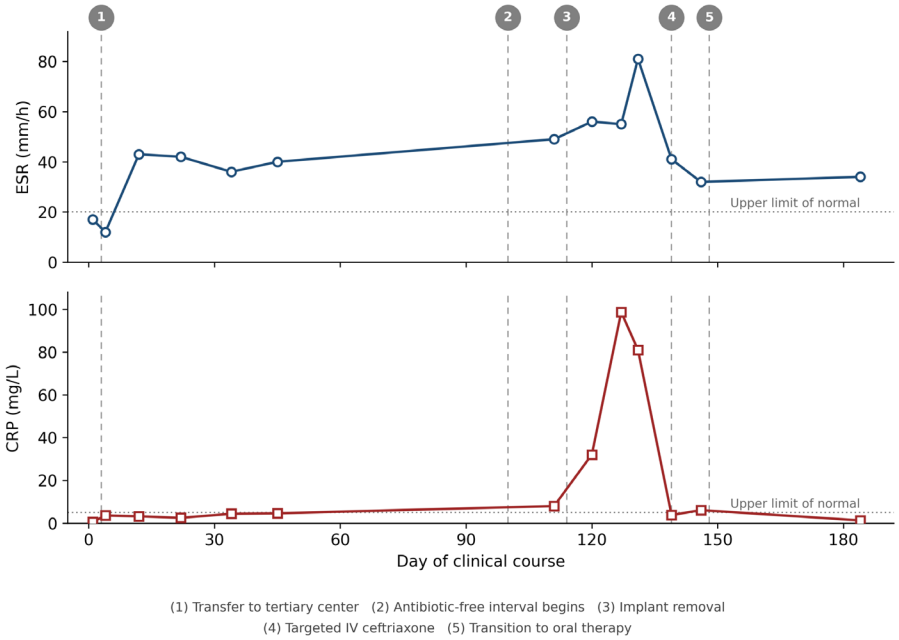


Figure 2. *Trajectory of inflammatory markers across the clinical course. Erythrocyte sedimentation rate (ESR) (top) and C-reactive protein (CRP) (bottom) values are plotted against the day of the clinical course. Numbered events along the upper margin indicate: 1) transfer to the tertiary center, 2) start of the planned antibiotic-free interval before hardware removal, 3) implant removal, 4) initiation of targeted intravenous ceftriaxone after full susceptibility data, and 5) transition to oral therapy. Dotted reference lines indicate the local upper limits of normal.*

Author	Patient and comorbidities	Clinical presentation	Spinal infection type and location	Diagnostic methods	Treatment	Outcome
Abel et al.[10]	84F; HTN, CAD, AS	Sharp lower back pain and bilateral leg weakness	Vertebral osteomyelitis/ discitis L3-L4 (MRI)	ESR 77 CRP 5.88 Blood cultures were positive for *S. salivarius* TEE negative	Ceftriaxone 2g IV q24hrs × 6 weeks	Pain improved with normalization of ESR/CRP at 12-day follow-up
Ahmad et al.[11]	73M; no PMH	Hip/neck/ low-back pain that later developed into lower extremity weakness	Vertebral osteomyelitis/ discitis L4-L5	MRI lower spine Blood cultures positive for *S. salivarius* Vegetations on TTE/TEE	Ceftriaxone IV x 6 weeks + gentamicin × 2 weeks	Death (septic shock/multiorgan failure with persistent vegetations)
Murillo et al[12]	NR (combined report)	NR	Streptococcal vertebral osteomyelitis	Viridans (including salivarius) vertebral osteomyelitis linked with endocarditis	NR	NR
Our case	24M; congenital scoliosis s/p posterior thoracolumbar fusion 7 years prior; no other comorbidities	Headache, back pain, left-predominant lower extremity weakness; brisk DTRs and bilateral Hoffmann sign	Late-onset SII with multiloculated paraspinal collections at cervicothoracic (C7) and thoracolumbar (L2) junctions	MRI paraspinal collections; intra-operative tissue culture grew *S. salivarius*; CSF grew *S. haemolyticus*; Gram stain Gram-positive cocci in chains	Hardware removal + IV ceftriaxone 2 g q12h (10 days), then PO cefuroxime (12 weeks)	5- and 11-week follow-up

Table 1. *Reported cases of Streptococcus salivarius spinal infections.*
Abbreviations: HTN, hypertension; CAD, coronary artery disease; AS, aortic stenosis; PMH, past medical history; TEE, transesophageal echocardiogram; TTE, transthoracic echocardiogram; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; NR, not reported; MRI, magnetic resonance imaging.

dental procedure on a patient while not wearing eye protection. Although this cannot be confirmed without further microbiological evaluation of the source patient, it remains highly probable that this incident triggered his infection, since the onset of his illness had a well-documented temporal relationship with the incident, and *S. salivarius* is a known normal oral flora, which was the source of the blood splash.

The conjunctival mucosa is a well-established route of entry for blood-borne pathogens, particularly viral organisms, in health-care settings[7]. In this case, the patient was not wearing eye protection at the time of the incident, which highlights the importance of adhering to personal protection equipment practices. The presence of metallic spinal implants provided a susceptible environment for bacterial adherence, and if a transient bacteraemia soon occurred after his conjunctival exposure to the blood splash, hematogenous seeding of the implant could have ensued. The isolate presented in this report showed intermediate susceptibility to penicillin but was susceptible to cefotaxime, ceftriaxone, and vancomycin, which guided the treatment regimen.

In the current case, many initial microbiological samples did not reveal any growth.

One possible explanation is the prolonged broad-spectrum antibiotic exposure prior to obtaining the correct specimens, which is known to drastically decrease the yield of cultures[8], when antimicrobials were held, implant tissue specimens finally revealed the causative pathogen.

Initially, implant retention was tried because the patient improved on meropenem. However, symptoms recurred after discharge and implant removal was necessary. After

the organism was identified, the patient was started on meningeal dosing of ceftriaxone for 10 days, followed by oral cefuroxime as the organism is susceptible to cephalosporins[9].

Only a few cases of *S. salivarius* spinal infections have been reported *(Table 1)*. Our patient was young, had no other comorbidities, and had his spinal hardware implants removed. This report also represents, to our knowledge, the first reported case of *S. salivarius* infection transmitted via blood splash.

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