CASE REPORT

Streptococcus gordonii prosthetic joint infection in the setting of vigorous dental flossing

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SUMMARY

A 65-year-old woman with osteoarthritis, who underwent knee replacement 5 years prior, developed sudden onset knee pain and swelling. She had voluntarily starting a vigorous dental flossing regimen prior to the onset of symptoms. The patient underwent right knee arthrotomy, irrigation and debridement of right total knee arthroplasty and exchange of polyethylene with retention of the prosthesis. Intraoperative cultures grew *Streptococcus gordonii*. She was treated with 6 weeks of ceftriaxone and was later placed on oral antibiotic suppression.

BACKGROUND

Streptococcus gordonii is a commensal member of the human oral flora and a member of the viridans streptococci group. While not directly pathogenic in the oral cavity, this case highlights a rare complication of prosthetic joint infection related to routine dental care.

CASE PRESENTATION

A 65-year-old Caucasian woman with a medical history significant for hypertension, hyperlipidaemia and osteoarthritis status post-knee replacement in 2009, with postoperative course free of complications, had done well until 6 months prior to admission, when she began to experience mild right leg and knee pain for which she was diagnosed with iliotibial band syndrome; acupuncture treatment was initiated. She experienced sudden onset of pain, swelling and subjective chills roughly 10 h prior to presenting to the emergency room. Her review of systems was otherwise negative. However, the patient had voluntarily starting a vigorous dental flossing regimen, with bleeding from her gums as a result.

On physical examination she was afebrile with a normal heart rate. Right knee examination revealed mild effusion, erythaema and increased warmth to the touch. There was significant pain on passive and active range of motion. Mild swelling below the right knee was also observed. The rest of the systemic examination was normal.

In the emergency room, orthopaedic surgery was consulted and right knee aspiration performed.

INVESTIGATIONS

Complete blood count was normal. Sedimentation rate was elevated at 24 mm/h and C reactive protein was elevated at 5.7 mg/dL. Electrolytes were normal except for mildly low sodium of 134 mmol/L and minimally low chloride of 95 mmol/L. Right knee joint fluid analysis results were white cell count (WCC) 40 000/CU MM, RBC 6444/CU MM, 95% segs, 3% lymphocytes, 2% monocytes, pH 7.38, 1.028 specific gravity. Right knee X-ray showed right knee arthroplasty, well seated components and no fractures. Venous Doppler ultrasound of the right leg was negative for deep vein thrombosis. Aerobic surgical culture had identified growth of *S. gordonii*. Anaerobic surgical culture observed no growth. Synovial fluid Lyme was negative. Blood cultures times two were negative.

TREATMENT

The patient underwent right knee arthrotomy, irrigation and debridement of right total knee arthroplasty and exchange of polyethylene with retention of right knee prosthesis. Surgical cultures were obtained showing Gram-positive cocci resembling strep. Final cultures grew *S. gordonii* and the patient was initially treated with vancomycin and later switched to ceftriaxone when cultures were finalised. After finishing her 6 weeks of intravenous antibiotics, she was placed on Pen VK suppression.

OUTCOME AND FOLLOW-UP

The patient completed 6 weeks of intravenous antibiotics and was later transitioned to Pen VK suppression. She was doing well at follow-up 12 months after her initial infection.

DISCUSSION

Nearly one million total knee arthroplasties and total hip arthroplasties are performed in the USA each year, significantly improving quality of life. This number is estimated to reach more than 4 million annually by 2030.[1] Prosthetic joint infection is one of the most serious complications of prosthetic joint implantation. The rate of prosthetic joint infection ranges between 0.5% and 1.0% for hip replacements, 0.5% and 2% for knee replacements, and less than 1% for shoulder replacements.[2–5] The rate of infection is highest during the first 2 years following surgery. The major risk factor associated with infection is superficial surgical site infection. Additional risk factors include: malignancy, prior joint arthroplasty, advanced age, immunocompromised conditions, post non-articular infections, prior infection of the joint, prolonged duration of surgery, high body mass index, postoperative haematoma formation and diabetes mellitus.[4 6–8]

The diagnosis of prosthetic joint infection can be difficult and utilises many different diagnostic tests

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including radiological, serological and microbiological. Recommended work up includes thorough history taking and physical examination, testing for sedimentation rate and CRP, plain radiograph, diagnostic arthrocentesis with synovial fluid analysis including total cell count and differential leucocyte count, culture for aerobic and anaerobic organisms, and blood cultures.[9]

Prosthetic joint infections are categorised according to the timing of symptom onset after implantation. Early onset infections occur less than 3 months after surgery. They are usually acquired during implantation and often due to virulent organisms such as *S. aureus*, Gram-negative bacilli, anaerobic organisms or mixed infections. Delayed onset infections occur from 3 to 12 months after surgery and are also usually acquired during implantation. These infections are often due to less virulent pathogens such as Enterococci, coagulase-negative Staphylococci or Propionibacterium species.[10] Late-onset infections occur more than 12 months after surgery and typically result from haematogenous seeding. The majority of late onset prosthetic joint infections are related to *Staphylococcus aureus* and *Staphylococcus epidermidis* (57%) with viridans group streptococci accounting for close to 2%.[11]

The patient's joint aspirate cultures grew *S. gordonii*, a member of the viridans group streptococci. The viridans group of streptococci is a heterogeneous group of organisms that are further subdivided into six major groups: *S. mutans*, *S. salivarius*, *S. anginosus*, *S. mitis*, *S. sanguinis* and *S. bovis*. The Strep *sanguinis* group includes *S. sanguinis*, *S. parasanguinis* and *S. gordonii*.[12] *S. gordonii* is an oral commensal bacteria and is not directly pathogenic; it is associated with dental plaque formation.[13] While not pathogenic in the oral cavity, *S. gordonii* initiates colonisation by creating surfaces for other colonisers to adhere to.[14] *S. gordonii*, however, has been implicated as a cause of endocarditis,[15] spontaneous bacterial peritonitis[16] and subcutaneous abscesses.[17] Cases of septic arthritis caused by *S. gordonii* are extremely rare with only two cases reported in the literature, one of which was a prosthetic joint infection.[18] Our case would be the second reported prosthetic joint infection; and the first to be reported in North America. Although it is difficult to establish direct causation, the only risk factor identified was the vigorous dental flossing regimen that the patient initiated prior to the onset of her symptoms. It is reasonable to speculate that the dental flossing regimen was responsible, given the timing of the infection, the organism identified, haematogenous seeding as the suspected mechanism and the known association between dental flossing/dental procedures producing transient bacteraemia.[19 20] This case illustrates a rare occurrence of a prosthetic joint infection related to dental care. Outside the realm of case reports, case–control studies did not identify an association between dental procedures and prosthetic joint infections.[21 22] The evidence is also lacking to support the use of antibiotic prophylaxis prior to dental procedures.[23]

Treatment of prosthetic joint infections involves surgery and antibiotic therapy. The specifics of treatment depend on the timing of the infection, the organism involved and other individual patient circumstances.[24] Patients diagnosed with a prosthetic joint infection who have a well-fixed prosthesis without a sinus tract, and who are within 30 days of prosthesis implantation or <3 weeks of onset of infectious symptoms, should be considered for a debridement and retention of prosthesis strategy.[9] Following surgery, our patient was treated with ceftriaxone intravenous for 6 weeks and was later transitioned to Pen VK. Several studies have shown that, when treated with debridement and retention of the prosthesis, prosthetic joint infections due to streptococci have a better outcome than do prosthetic joint infections due to other organisms.[25] These studies emphasise the critical role that specific microorganisms play in the success of debridement with prosthesis retention as a treatment modality. Tattevin *et al*[26] reported that a short interval (≤5 days) from onset of symptoms to debridement of the infected prosthetic joint significantly correlated with a successful outcome when the prosthesis was retained.

While on intravenous antibiotics, our patient developed profound neutropaenia, with WCC of 0.7×10^9/L and an ANC of 0.0×10^9/L, 6 weeks into the course of standard dose ceftriaxone. On discontinuation of ceftriaxone and initiation of Penicillin VK for chronic suppression, the neutropaenia resolved 12 days later. Neutropaenia is a recognised side effect of intravenous ceftriaxone, although the exact frequency is unknown.[27] This case illustrates the importance of routine laboratory surveillance when on prolonged antibiotic infusions.

Learning points

- *Streptococcus gordonii* is a rare cause of prosthetic joint infection and may be caused by dental flossing or other dental procedures known to cause transient bacteraemia.
- Prophylactic antibiotics prior to dental procedures are not routinely recommended due to extremely rare incidence of oral flora causing prosthetic joint infection and lack of evidence suggesting prophylactic antibiotics reduce risk.
- Debridement with retention of prosthesis is a viable treatment option with acute streptococcal prosthetic joint infections.
- Ceftriaxone in rare cases can cause neutropaenia.

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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