Orthopedics and Rheumatology
Open Access Journal
ISSN: 2471-6804

Case Report
Volume 18 issue 4 - August 2021
DOI: 10.19080/OROAJ.2021.18.555998

Ortho & Rheum Open Access J


# Culture-Negative Peptostreptococcus Micros Knee Septic Arthritis in Rheumatoid Arthritis Diagnosed By 16s Ribosomal DNA PCR

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**Submission:** July 18, 2021; **Published:** August 05, 2021

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## Introduction

*Peptostreptococcus micros* or Parvimonas species an anaerobic, Gram-positive cocci that is a normal oral and gastrointestinal flora, is a very rare cause of septic arthritis. *Peptostreptococcus micros* knee septic arthritis has been reported in a patient soon after an intra-articular corticosteroid injection for osteoarthritis. We present a case of right knee joint septic arthritis due to *Peptostreptococcus micros* in a rheumatoid arthritis (RA) patient who was receiving treatment with oral prednisolone. Synovial and blood cultures as well as conventional microscopy were negative on several occasions, and the infection with *Peptostreptococcus micros*, was only diagnosed with bacterial PCR. Treatment with prednisolone can mask the systemic responses to infection and therefore a higher index of suspicion and perseverance in testing is important in this clinical setting.

## Case Report

A 62-year-old male Bengali RA patient was seen at a local district general hospital's emergency department with a 2-week history of progressively worsening right knee pain and swelling associated with a low-grade fever of 37ºC and a large right knee effusion. He was on Prednisolone 5mg per day. The knee was aspirated, and turbid synovial fluid (SF) was sent for microscopy, culture, and sensitivity (MC&S). Microscopy revealed pus cells without any organisms. In view of the patient's stable clinical condition, he was discharged from hospital on the same day without any antimicrobial therapy. Cultures were negative and the patient was not re-called.

He also had a history of type 2 diabetes mellitus, hypertension, ischaemic heart disease, osteoporosis and secondary osteoarthritis of the knees. In the past, after failing at least 6 disease-modifying anti-rheumatic drugs (DMARD), he had been started on Adalimumab 2 years previously combined with Leflunomide. Both drugs had been stopped 8 months prior to this episode following a pulmonary infection with Group C Streptococcus which was complicated by lung abscess. This was successfully treated.

He was seen in the rheumatology clinic 2 weeks later and by then his right knee effusion had worsened leading to pain and inability to weight bear. On examining the right knee, was hot, tender and swollen. There was also pitting oedema on his right leg up to his knee which raised the possibility of a concomitant deep vein thrombosis which was later excluded through an ultrasound Doppler scan. His RA had flared, and he had synovitis in many other peripheral joints.

He was admitted into hospital on the same day. He was alert and well with vital signs within normal range and a temperature of 37ºC. Initial blood tests showed a raised white blood cell count (WBC) of 15.1 10*9/L, neutrophil count of 12.2 10*9/L and a C-Reactive protein of 403mg/L. Turbid yellow SF was aspirated from the right knee. Microscopy revealed numerous pus cells (95% neutrophils and 5% lymphocytes) and no organisms. No growth was again obtained. The right knee x-ray showed a large knee effusion with advanced degenerative changes. A presumptive diagnosis of a septic arthritis was made and intravenous benzyl penicillin 1.2grams QDS and flucloxacillin 1gram QDS were started. He underwent an arthroscopic knee washout and biopsy, and samples were again sent for MC&S and Acid-Fast Bacilli cultures (AFB) as well as synovial histology.

By Day 3 of antibiotic therapy, his inflammatory markers had improved with the CRP falling to 324mg/L and by Day 5 it was 133mg/L. Arthroscopic washout samples grew no organisms and by day 7 of antibiotic therapy, it was felt that septic arthritis was unlikely, and antibiotics were stopped. At this point it was felt that his effusion was most likely due to his long-standing osteoarthritis of his right knee coupled with a flare of his RA since he had synovitis in several other joints.

On day 8 he was given an intravenous infusion of methylprednisolone 500mg and again on Day 10. After the second dose of methylprednisolone, the synovial biopsy's histological results came back which suggested changes consistent with a septic arthritis. It showed sections of fibroadipose tissue within the surface showing extensive fibrino-purulent inflammation. The subjacent tissue showed mixed acute and chronic inflammation with patchy necrosis and fibrin deposition.

In view of this result, the 3rd methylprednisolone infusion was not given, and he was restarted on intravenous antibiotics. This time intravenous flucloxacillin 1gram QDS and co-amoxiclav 1.2gram TDS were chosen instead. In view of the negative cultures on several occasions, a Polymerase Chain Reaction test (16S rDNA PCR) was performed on the SF sent during the arthroscopic washout (samples 3 & 4). Sample 4 was negative but amplification and sequencing of 16S rDNA, using broad range bacterial PCR primers, identified *Peptostreptococcus micros* from sample 3 (100% match over 1111 bases).

These results became available on Day 21. As the organism could not be cultured, antimicrobial sensitivity testing was not possible. Intravenous benzylpenicillin was restarted empirically, based on the expected sensitivity pattern for this organism, for another 2 weeks. This led to a marked improvement in his right knee swelling and the patient was able to weight bear with the help of intensive physiotherapy. His inflammatory profile continued to improve. He was discharged from the hospital on oral antibiotics to complete a total of 3 months of antibiotics treatment. At follow up in outpatient clinic up to 4 months, his ESR and CRP had normalised, and he had no joint effusion.

## Discussion

This case highlights the difficulties of diagnosing septic arthritis, caused by a relatively hard to culture organism of low pathogenicity, in an immunocompromised RA patient with several other co-morbidities. It has been well documented that the anti-TNF medications such as Adalimumab increase the risk of infections [1]. This case is unusual in that the patient had stopped Adalimumab 8 months before and 4 SF cultures taken at different times failed to grow any organism despite him having clinical features highly suggestive of septic arthritis.

To our knowledge this is the first time a case of Peptostreptococcus has been reported in a patient treated previously with anti-TNF biotherapy and on current oral prednisolone therapy. There are only 9 reported cases of *Peptostreptococcus* joint infection in the literature. Three of them had had joint replacement surgery {2 of these had RA; 1 had osteoarthritis} all of whom the infection occurred in the replaced joint [2-4]. The fourth case had neither joint replacement surgery nor a chronic autoimmune disease but had multiple myeloma [5]. None of the reported RA patients had previously taken anti-TNF biotherapy drugs.

*Peptostreptococcus* organisms are part of the normal flora of human mucocutaneous surfaces, including the mouth, intestinal tract, vagina, urethra, and skin. Due to its anaerobic growth requirements, it is a rather difficult organism to grow. All four samples from our patient failed to grow this bacterium in anaerobic culture. PCR techniques proved a useful adjunct in the diagnosis of this infection proving more sensitive than standard culture and are a more reliable method to detect this type of organism in this setting [6].

## Conclusion

This case, the first report of peptostreptococcal infection in a native joint in an RA patient previously with Humira, highlights the need to be alert to the possibility of unusual infections in this patient group. If there is clinical suspicion of infection in such a patient early input from the microbiology team should be sought, enabling the prompt application of appropriate diagnostic techniques, the newer molecular ones, such as 16s PCR. Our case also highlights the difficult of diagnosing septic arthritis in RA patients who present with septic arthritis and a concomitant flare of RA. A high index of suspicion should be borne in made in patients on steroids and/or anti-TNF biotherapy as in both situations, the systemic features of a septic infection will be significantly masked.

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**How to cite this article:** Chikanza IC. Culture-Negative Pepto streptococcus Micros Knee Septic Arthritis in Rheumatoid Arthritis Diagnosed By 16s Ribosomal DNA PCR. Ortho & Rheum Open Access J. 2021; 18(5): 555998. DOI: 10.19080/OROAJ.2021.18.555998

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