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First reported case of *Rothia dentocariosa* spondylodiscitis in an immunocompetent patient

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Introduction

Rothia dentocariosa is part of the normal human oropharynx microflora in 1.3%–29% of healthy individuals [1,2] and is commonly associated with dental caries and periodontal disease [1]. Invasive disease has been described essentially in immunocompromised hosts [3] and/or patients with a predisposing factor, such as cardiac valvular disease or Intravenous (IV)-drug users [4]. We present a unique case of a healthy patient with spondylodiscitis caused by this pathogen.

Case description

A 46-years old male, known for chronic epigastric pain, consulted a tropical and travel related infectious diseases outpatient clinic on day 7 of persistent fever. His symptoms started with febrile diarrhea followed by transfixing epigastric pain, two days after his arrival in the Middle-East for tourism. On

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ABSTRACT

Rothia dentocariosa is part of the normal human oropharyngeal microflora and is frequently associated with dental caries and periodontal disease. Invasive disease has been described essentially in immunocompromised hosts and/or patients with underlying conditions as predisposing factors. We present a case of an otherwise healthy 46-years old male with spondylodiscitis caused by this pathogen. Treatment with ceftriaxone and rifampin was successful. To our knowledge, this is the first *R. dentocariosa* spondylodiscitis reported in an immunocompetent patient, and the second one in the literature overall. © 2019 The Authors. Published by Elsevier Ltd. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

day 5 the diarrhea disappeared. However, night fevers of $38.5 \,^{\circ}$ C persisted, associated with severe diaphoresis, epigastric transfixing left paravertebral pain, and diffuse myalgia. The patient denied cardiopulmonary, urinary or cutaneous symptoms.

The patient is working in the financial sector. While travelling, he stayed in an upper-range hotel and did not eat raw food nor dairy products, had no contact with sick people or animals, and had no still water or mosquito bites exposure. The patient lived in a monogamous relationship and had no history of IV-drug use. One day before travelling and three days prior to the onset of his illness, he had dental scaling.

Clinically, the patient had Glasgow Coma Scale of 15, presented a low-grade fever (37.8 °C), his heart rate was 83/min, blood pressure 156/83 mmHg and was in no apparent distress with slightly painful epigastric palpation with left paravertebral pain radiation and moderate periodontal disease with gums recession without signs of acute gingivitis. He had no lymphadenopathy or organomegaly, cardiac and neurological status showed no abnormality. Spinal percussion and mobilization were not painful or limited.

The initial blood test on day 7 of fever showed normal leucocytes counts $(7.5 \times 10^9/L)$, with a mild lymphocytopenia $(0.95 \times 10^9/L)$, normal neutrophils counts $(5.93 \times 10^9/L)$ and an isolated mild relative monocytosis on day 9 (11.3 % of $6.7 \times 10^9/L)$. C-reactive protein was 37 mg/L and then fluctuated between 18 and 54 mg/L for more than one week. Liver enzymes, creatine

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Case report





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kinase, lipase and urine tests were normal. Two blood cultures and a broad-range PCR in the feces were negative for pathogens tested. Active cytomegalovirus, Epstein-Barr virus, Parvovirus B19, HIV, Human herpesviruses 6 and 7, Rubella and *Toxoplasma gondii* infections were excluded by negative serum PCRs and serology. The chest X-ray was normal. QuantiFERON[®], *Brucella* serology and Rose Bengal Tests, as bacterial broad-range blood PCR were negative.

A transthoracic echocardiogram was negative for signs of endocarditis by day 21. A thoracic-abdominal CT-scan on day 23, showed signs of vertebral endplate mirror erosions regarding the T10-T11 discus. The patient was then hospitalized for further workup.

On day 24, an aerobic bottle from a blood culture, done by day 17, became positive for Gram-positive cocci identified as *R. dentocariosa* by MALDI-TOF MS (Matrix-Assisted-Laser Desorption/Ionization - Time of Flight Mass Spectrometer) (MALDI-TOF MicroFlex LT Bruker Daltonik) with the highest score at 2.43.

A spinal Magnetic Resonance Imaging (MRI) on day 28 confirmed a T10-T11 spondylodiscitis with vertebral endplates mirror erosions and signal abnormality of the adjacent vertebral bodies showing T1 hyposignal, STIR hypersignal and T1 contrast enhancement (Fig. 1). On day 29, discus and vertebra biopsy were performed; the direct Gram stain was positive for Gram-positive cocci, identified as R. dentocariosa by MALDI-TOF. A subsequent aerobic bottle blood culture became positive for Gram-labile rods, also identified as R. dentocariosa by MALDI-TOF MS. A transesophageal echocardiogram revealed no signs suggestive of endocarditis. Minimum inhibitory concentrations (MIC) were determined according to EUCAST guidelines [5] for the following antibiotics: penicillin 0.004 susceptible (S), ceftriaxone 0.023 (S), gentamicin 8 resistant (R), ciprofloxacin >32 (R), clindamycin 1 (R), trimethoprim-sulfamethoxazole (TMP-SMX) 0.23 (S), rifampin 0.023 (S), and vancomycin 1.5 (S; units in μ g/ml).

Ceftriaxone was started on day 30 (2 g IV qd) for 34 days, followed by a switch to oral TMP-SMX (800/160 mg tid) for 14 days. Rifampin (450 mg orally bid) was added for the entire 48 days of treatment.

After treatment initiation, the patient did not present any recurrence of fever, and inflammatory markers normalized. Due to persistent spinal pain, we repeated an MRI six weeks after antibiotics cessation, which showed low residual inflammation.

Discussion and conclusion

R. dentocariosa is a facultative anaerobic, pleomorphic Grampositive bacterium with varying morphology including coccoid, diphtheroid, and filamentous forms. Its identification can be challenging for clinical laboratories [6]. In our case, *R. dentocariosa* pleomorphism manifested as Gram-positive cocci and Gram-labile rods; definitive identification was obtained through MALDI-TOF.

Our patient presented with T10-T11 spondylodiscitis, likely a result from the minor periodontal intervention. In a review of *R. dentocariosa* endocarditis, periodontal disease was found in 60 % of patients, 75 % of whom were male [4]. In the general population, infections others than caries that involve *R. dentocariosa* are very rare. Systemic infections associated with *R. dentocariosa* have been mostly bacteremia [3]. High complication rates in up to 35 % cases have been described for endocarditis, including mycotic aneurysms, brain abscesses, intracerebral and subarachnoid hemorrhages [4]. In September 2015, Chowdhary *et al.* counted only 24 published endocarditis cases in the literature [7]. Bone and joint infections have been very rarely reported with only one case of vertebral osteomyelitis secondary to endocarditis [8] and one case of native joint septic arthritis [9], both described in immunocompromised patients.

In the present case, *R. dentocariosa* was initially considered dubious as etiological agent in absence of previously reported bone



Fig. 1. Spinal MRI T1w images after gadolinium contrast injection, in the sagittal plane through the thoracic vertebrae show inflamed T10 and T11 vertebral bodies.

and joint infections in immunocompetent patients for this bacterial species. However, due to the clinical deterioration, antibiotics were started before *R. dentocariosa* was identified in discus and bone biopsies. There is limited data available on *R. dentocariosa* antimicrobial susceptibilities. The most frequently used antimicrobial therapy is a combination of penicillin and gentamicin [3,4]. In our case, *R. dentocariosa* was gentamicin-resistant. Publications report a high sensibility to penicillin, cephalosporin and rifampin [4,10], and cephalosporin-rifampin has been suggested as first-choice combination [10]. Accordingly, our patient received a combination of two active antibiotics based on published endocarditis were found (two minor Duke Criteria) and no immune suppression was identified based on patient and family's history, full blood counts, and HIV virus detection.

In conclusion, we find that *Rothia dentocariosa*, part of the normal human oropharyngeal microflora, has an invasive potential even in immunocompetent patients. *R. dentocariosa* can cause a serious systemic infection that clinicians should be aware of, and treat accordingly.

Competing interests

None.

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Consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request

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