

Parvimonas Micra Bloodstream Infection in a Patient with Oral Mucositis

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Abstract

A *Parvimonas micra*, a normal constituent of oral flora, is an opportunistic pathogen more commonly associated with oral infections after dental procedures. Here we report a fatal case of sepsis in a patient suffering from oral mucositis secondary to radiochemotherapy for rhynopharynx carcinoma. This case highlights the importance of oral mucositis as an underestimated infectious risk factor.

Keywords: *Parvimonas micra*; Bloodstream infection; Cancer; Mucositis; Radiochemotherapy

Introduction

Mucositis is a disabling toxicity related to head and neck cancer (HNC) treatment [1]. As well as negatively affecting nutritional intake, quality of life, radiotherapy (RT) and/or chemotherapy (CT) delivery, it is associated with a higher risk of local and systemic infections which can be life-threatening [2]. Local infections are often sustained by *Candida* species and Herpes Simplex virus; systemic infections, are commonly due to *Candida* species, gram-negative bacteria and alpha-hemolytic streptococci [3].

Formerly known as *Peptostreptococcus micros* or *Micromonas micros*, the Gram-positive anaerobic cocci currently classified as *Parvimonas micra* (*P. micra*) are normal constituent of oral and gastrointestinal flora. While there is broad evidence to support the role of *P. micra* in periodontal and other oral infections, those outside the oral cavity have been underreported [4]. It is worthy to note that dental procedures are the main risk factor for *P. micra* infection; no case has been reported following radiochemotherapy (RT-CT) for HNC or secondary to oral mucositis [5].

We report a case of *P. micra* lung and, subsequently, bloodstream infection in a patient affected by rhynopharynx carcinoma treated with RT-CT.

Case Report

A 71-year-old Italian man presented with 2-day history of fever without any other acute complaint. His clinical history was significant for Parkinson disease from 7 years and long-standing hypertension. He had been diagnosed with high-grade, locally invasive rhynopharyngeal carcinoma in September 2016. Patient underwent RT-CT with intensity-modulated technique and concomitant weekly cisplatin infusion through peripherally inserted central venous catheter (PICC) (after first cycle he was shifted on carboplatin infusion for kidney toxicity). The total RT dose was 70.2 Gy with a daily fraction of 1.8 Gy; RT volumes were tumor, pathological nodes and elective drainage

nodes. Treatment was complicated with grade 2 oral mucositis and worsening of motor compensation.

Follow-up examinations revealed a partial response of the nasopharyngeal mass and the presence of a right iliac wing secondary lesion. Accordingly, patient was started with weekly infusion of carboplatin and cetuximab in May 2017. CT was soon complicated by grade 3 fatigue with consequent reduced mobility and grade 3 oral mucositis.

On examination, the patient was in poor clinical conditions but he denied any acute distress; vital parameters were as follows: temperature 37.9°C, blood pressure 120/70 mmHg, pulse rate 100 bpm, oxygen saturation 96% on room air. There was no spinal tenderness or other localizing sign; his heart sounds were dual and lungs were clear with good air entry on auscultation; abdominal exam revealed no abnormalities; there was no erythema or edema at the lower limbs.

Laboratory studies demonstrated a leukocyte count of $12.06 \times 10^3/\text{mL}$, a procalcitonin (PCT) of 0.31 ng/mL and a C-reactive protein (CRP) elevated at 183.4 mg/L; renal and hepatic tests were within normal; blood gas analysis documented a type 1 respiratory failure (PaO₂ 59 mmHg, PaCo₂ 33 mmHg). Chest-x-ray showed no abnormalities. Blood samples for culture were obtained from one peripheral venipuncture site and one PICC.

Assuming an atypical lung infection, the patient was started on cefepime 1 g b.i.d. and azithromycin 500 mg q.d. Three days into admission, with progressive worsening of respiratory failure, both blood samples grew *P. micra* and procalcitonin increased up to 12.07 ng/ml. After infectious disease specialist consultation, the patient was shifted on ampicillin/sulbactam and vancomycin.

Despite treatment, the patient died from progressive respiratory and multiorgan failure four days from admission.

Discussion

As this case shows, *P. micra* lung infection can be rapidly progressive and even fatal in at-risk patients. *P. micra* is an opportunistic pathogen which more commonly causes oral infections after dental procedures. Central nervous system, thorax, abdomen, genitourinary and soft tissues infections usually develop with

predisposing conditions such as immunodeficiency, diabetes, steroid treatment, and neoplasia. Multiple risk factors appear to be involved in our case: neoplasia, chemotherapy in progress, severe mucositis and consequent malnutrition, Parkinson disease and reduced mobility. It is likely that the interplay of these risk factors has been decisive in determining the negative outcome of therapies.

P. micra may cause different thoracic infections: pneumonia, lung abscess, pleuritic empyema, mediastinitis. In the case of pneumonia and lung abscess, the most common thoracic infections, clinical course is usually indolent but more aggressive presentation may occur as our case showed. It is important to recognize that chest-x-ray findings may be elusive and, as a matter of fact, we report a case in which thoracic imaging showed no abnormalities.

P. micra pneumonia should be treated with either clindamycin or a combination of penicillin and a β -lactamase inhibitor; it is recommended that such regimen lasts 2-4 weeks, depending response. In the case of a lung abscess, treatment should not withheld for up to 3 months or until the chest imaging clears, though treatment can be shortened with proper surgical drainage [6].

Even more important in our opinion, this case highlights the importance of oral mucositis as an underestimated infectious risk factor. While there is no direct cause-effect relationship between *P. micra* infection and cetuximab therapy, it should be appreciated that evidence suggests a higher infectious risk, particularly of pneumonia, in older HNC patients treated with cetuximab [7]. This drug is not immunosuppressant per se but in combination with platinum-based CT it can increase the frequency of mucositis and severe neutropenia.

To date, no evidence-based protocol has been developed for prevention and treatment of oral mucositis and this is particularly troublesome for HNC patients who face a substantial risk of severe mucositis. MASCC/ISO guidelines for oral mucositis recommend benzydamine mouthwash to prevent oral mucositis in patients with HNC receiving moderate dose radiation therapy (up to 50 Gy), without concomitant chemotherapy; the panel suggests oral care

protocols, low-level laser therapy and systemic zinc supplements to prevent oral mucositis in patients with HNC receiving moderate dose radiation therapy (up to 50 Gy), without concomitant chemotherapy. MASCC/ISO guidelines suggest only a symptomatic treatment for oral mucositis, with morphine or doxepine mouthwash. It is therefore evident that currently available evidence for preventing and treating oral mucositis in patients with HNC is insufficient. We advocate more studies be conducted on this field in the future.

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