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Case Report

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Rothia mucilaginosa, rarely isolated pathogen as an etiological factor of infection of soft tissues in young, healthy woman

Rothia mucilaginosa rzadko izolowany patogen jako czynnik etiologiczny zakażenia tkanek miękkich twarzy u młodej zdrowej kobiety

Authors' Contribution:
A Study Design
B Data Collection
C Statistical Analysis
D Data Interpretation
E Manuscript Preparation
F Literature Search
G Funds Collection

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Summary

This paper presents a rare case of facial soft tissue infection caused by the bacterial strain of *Rothia mucilaginosa*. Odontogenic background of infection and initial clinical presentation suggested the presence of typical bacterial flora and uncomplicated course of treatment. However, despite surgical intervention and broad-spectrum antibiotic therapy, the expected improvement of a clinical status was not achieved. Only detailed bacteriological examination allowed to establish a bacterial pathogen and start a targeted antibiotic therapy. The unusual clinical course was monitored by imaging CT examination and further surgical interventions. A significant improvement was obtained in the third week of hospitalization and further antibiotic therapy was continued by means of outpatient treatment. *Rothia mucilaginosa* infection together with dental intervention is a rare case, since most of the reports in the literature concern the patients with decreased immunity. In such patients, the most common areas of infection were: the peritoneum, lung tissue and meningeal spaces of the brain and the presence of a foreign body.

Key words: ***Rothia mucilaginosa*** • **dental infection** • **antibiotic therapy**

Streszczenie

W pracy przedstawiono rzadki przypadek infekcji tkanek miękkich twarzy szczepem bakteryjnym *Rothia mucilaginosa*. Zębopochodne tło zakażenia i początkowy obraz kliniczny sugerowały obecność typowej flory bakteryjnej i niepowikłany przebieg leczenia. Jednak mimo interwencji chirurgicznej i antybiotykoterapii o szerokim zakresie działania nie osiągnięto spodziewanej poprawy stanu klinicznego. Dopiero szczegółowe badania bakteriologiczne pozwoliły na ustalenie drobnoustroju chorobotwórczego i rozpoczęcie celowanej antybiotykoterapii. Nietypowy przebieg kliniczny kontrolowano badaniami obrazowymi TK i kolejnymi interwencjami chirurgicznymi. Znaczącą poprawę uzyskano w trzecim tygodniu hospitalizacji, a terapię antybiotykiem kontynuowano ambulatoryjnie. Zakażenie *Rothia mucilaginosa* w powiązaniu z interwencją

stomatologiczną jest rzadkim przypadkiem, tym bardziej że większość doniesień dotyczy chorych z obniżoną odpornością. U takich pacjentów najczęściej zakażone były otrzewna, tkanka płucna, przestrzenie oponowe mózgu, zakażenia z obecnością ciała obcego.

Słowa kluczowe: ***Rothia mucilaginosa*** • **zakażenia zębopochodne** • **antybiotykoterapia**

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Introduction

The odontogenic tissue infections around a mouth and face are dominated by microorganisms that are present in the oral cavity in dental plaque, gingival pockets, on the surface of the mucous membrane, etc. Most often, multiple strains of bacteria are observed in the initial phase of odontogenic infection. However, in the course of infection, while the conditions of tissue related to hypoxia change, so does the composition of dominant bacterial flora. The most common pathogens are: *Streptococcus milleri*, *Peptostreptococcus*, *Prevotella*, *Porphyromonas*, *Fusobacterium* [3,14] Tissue infections of face and oral cavity caused by bacteria *Rothia mucilaginosa* are rare, despite their presence in mouth and air passages [2,7,9,11,16,17]. The infections caused by this factor are often called opportunistic infections, concerning mainly people with impaired immunity, such as those undergoing immunosuppression [2,5,8,16]. They may also be accompanied by the presence of a foreign body [4,7]. Bacteria *Rothia mucilaginosa* belong to actinobacteria, also called actinomycetes. They are a slowly growing microorganisms, requiring 2–7 days of growth under aerobic conditions and 7–28 days under anaerobic conditions [10]. *Rothia mucilaginosa* do not grow on Chapman agar and Meuller-Hinton agar.

On the Columbia base, with the addition of sheep blood, they grow as white colonies resembling the colonies of coagulase-negative staphylococci or micrococci [9] (Fig. 1). Morphologically, they are Gram-positive cocci arranged in pairs, clusters and tetrads [9,10,16]. *Rothia mucilaginosa* were first isolated from milk in 1900. They were called Micrococcus mucilaginosus, then classified as *Staphylococcus salivarius*, and then again reclassified as *Micrococcus mucilaginosus*. Problems with the classification resulted from the similarity of these bacteria to staphylococci, micrococci, streptococci and enterococci [9]. In 1982, based on biochemical characteristics they were classified into *Stomatococcus mucilaginosus*. In man they were first isolated in 1978 [4,8].

Case Study

A 20-year-old woman was admitted to the Clinical Department of Maxillofacial Surgery because of increasing swelling and pain in the left cheek. The medical history revealed that the occurrence of pain was preceded by a toothache (27). Due to the presence of chronic periapical changes and pain, the tooth was classified as not suitable for conservative treatment and was extracted the day before the patient was admitted to the hospital. After the tooth extraction, the patient did not apply the prescribed antibiotics. Due to the growing soft tissue swelling of the cheek and general discomfort, the patient was admitted to the hospital. The patient did not report any coexisting diseases and allergies. The only drugs she took regularly were contraceptives. The external oral examination revealed significant facial asymmetry caused by the inflammation of cheek soft tissue, accompanied by the swelling of the eyelids of the left eye. The eyeball was moving properly, the conjunctive remained without any signs of inflammation. The skin next to the change was pale pink, palpable hard, infiltrated, without any symptoms of fluctuation. Nose permeable, cranial nerve function preserved. Neck examination showed enlargement and tenderness of the submandibular lymph nodes of group II B and II C, while maintaining their mobility. The intraoral examination revealed the exaggeration of tissue and redness of the mucous membrane in the area of the upper molars on the left side, the lack of tooth 26 and the postextractive wound of alveolar 27. No sinus alveolar connection was found. Laboratory tests showed a fairly significant leukocytosis (17700/µl) and increased

Fig. 1.

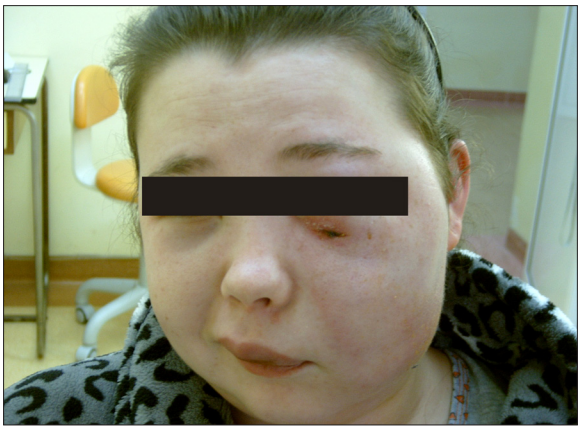

Fig. 2.

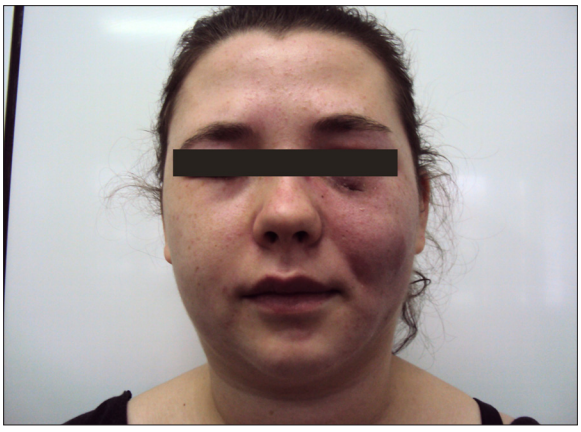

Fig. 3.

C-reactive protein (233.40 mg/l). The panthomographic and sinus X-ray images in the Water's projection showed no pathological changes, besides those of the postextractive alveolar. After the puncture of the tissue around 26–27 teeth, purulent content was obtained. The odontogenic submucous was recognized and the patient received intraoral incision of the abscess under local anesthesia. The procedure was performed on admission, in the antibiotic cover of amoxicillin with clavulanic acid at a dose of 1.2 g i.v. administered 30 minutes before surgery.

The abscess cavity was washed with disinfectant and drained, protecting the drain with a stitch. In the postoperative period, the intravenous empirical therapy was continued: amoxicillin with clavulanic acid at a dose of 1.2 g *i.v.* together with metranidazole at a dose of 3×0.5g *i.v.* In the next twenty four hours of hospitalization, despite the decrease of laboratory inflammation indicators (CRP 118.90 mg/l, WBC 17 400/ml), a significant extension of the inflammatory infiltration with involvement of the eyelids and closed palpebral fissure was observed (Fig. 2). Inflammatory infiltration also occurred in the submandibular area on the side of change. The lockjaw of IIo also ocured. The urgent angio-CT revealed a fluid reservoir on the front side of the maxillary sinus infiltration. The tissues were examined, without acquiring any inflammation content.

As the clinical effect of antibiotic therapy applied so far was not visible, antibiotics were discontinued, and the empirical antibiotics piperacilin with tazobactam started to be applied. In the subsequent days of hospitalization, the state gradually deteriorated. Infiltration of the check, lips and submandibular area became hard, skin became reddened and very tense. In addition, the inflammation started to spread to the temporal area. It was decided to execute the next revision of the inflamed tissue occupied during which swabs were taken for microbiological examination.

Based on the literature, 1 g of vancomycin applied every 12 hours with 1×900 mg of rifampicin orally were applied.

Therefore, it was decided to continue the earlier applied, empirical therapy of vancomycin and rifampicin. The clinical improvement was observed. Before that, the patient's medical condition had not improved, possibly because, as indicated in the literature, a strain of *Rothia mucilaginosa*, had been resistant to beta-lactams, received by the patient. The laboratory studies showed a significant improvement in inflammatory markers (CRP 10.20 mg/l, WBC normal. IgG, IgM levels were measured (result within normal limits), as well as compliment C3c and C4 (the result in the normal range). Malnutrition was excluded, HIV and ANA were marked as normal. Control CT was performed which showed absence of fluid within the tank face, but only a general inflammatory infiltration of soft tissues. The treatment of vancomycin with rifampicin was continued for 10 days, and showed a significant improvement of the local state and further normalization of laboratory test results. The patient was discharged from the hospital, heading for further treatment in the outpatient clinic. It was recommended to continue treatment with rifampicin at a dose of 1×900 mg for another 14 days (Fig. 3).

Microbiological problems

After the incision of the abscess, the material for microbiological tests was collected, in accordance with the applicable procedures (providing conditions for both the aerobic and anaerobic flora).

The microbiological tests revealed no microbiological cultures of microorganisms.

In next revision of the tissue swabs were taken for microbiological examination.

After 4 days, the information was received that the strain of *Rothia mucilaginosa* was cultured.

After the additional 48 hours of incubation on the Columbia agar with sheep blood, white colonies grew. Initially, after 24 hours of incubation, the colonies were very small, barely visible. However, the colonies did not grow on the Chapman agar for staphylococci. As early as in the suspension in salt, which was needed for identification, it turned out that the bacteria hardly hang in salt, in contrast to the staphylococci. The extra microscopic slide was prepared from the culture, stained using Gram method. The presence of Gram-positive cocci arranged in pairs was revealed.

The examination was conducted using identification test – GP card in automatic system VITEK 2 (bioMerieux). The

result achieved indicated with 99% of probability that the bred pathogen was in fact *Rothia mucilaginosa*, i.e. the certain result. In carrying out identification cards and setting the antibiogram card (AST P580), the organism was initially treated as coagulase-negative staphylococci.

The microorganism did not grow in antibiogram (card AST P580) in automatic system VITEK because it is slow-growing strain.

Then, additional real-MIC quantification using Etest (bioMerieux) was performed. In view of the fact that this organism does not grow on Mueller-Hinton substrate, the susceptibility assessment was performed on Columbia substrate with sheep blood. It turned out that the patient's strain was sensitive to rifampicin (MIC =0.006 mg/ml) and vancomycin (MIC = 1 mg/ml) in recomended Etest method (bioMerieux).

Discussion

The medical history and the initial state of the patient did not indicate the specific therapeutic difficulties. However, no improvement after surgical intervention and antibiotic therapy led to undertaking the detailed bacteriological studies. Initial difficulties with the identification of a pathogenic strain may have occurred due to previously carried out antibiotic therapy of a broad spectrum. The culture of such a rare pathogen was possible only in close cooperation of a clinician and microbiologist. The increase of CRP and leukocytosis, as well as no clinical improvement despite the antibiotic therapy led to prolonging the time of culture, thanks to which the organism could grow. Perhaps because of the slower growth, compared to other material cultured in microbiological laboratories, Rothia is a microorganism cultured so rarely. The bacteria resembled almost indistinguishably the coagulase-negative staphylococci. The identification of the microorganism was possible thanks to automatic bioMerieux VITEK 2 system, based on the analysis of several biochemical features.

The mention of *Rothia mucilaginosa* cultured from actinomycosis-like abscesses of cervico-facial area of an adult patient was found [10]. On the one hand, due to the slow growth of the microorganism, it was not possible to perform antibiogram (by test AST P580 automatic system VITEK 2 bioMerieux), and therefore, it was not known, to which antbiotics the organism was sensitive. On the other hand, antibiotic treatment had to be changed very quickly. Based on the literature [16], rifampicin at a dose of 900 mg/day and vancomycin 1 g every 12 hours were included in the further treatment. The condition of the patient improved. Perhaps the strain was resistant to piperacillin with tazobactam. The literature reports the cases where the failure was observed after the inclusion of amoxicillin with clavulanic acid [7,11]. Patient's strain was sensitive to rifampicin (MIC = 0.006 μg/ml) and vancomycin (MIC = 1 μg/ml). Not many reported cases of infection with the etiology of *Rothia mucilaginosa* have appeared in the literature. The following infections of this etiology have been described: endocarditis, bacteraemia, infections associated with vascular catheter, meningitis, brain-stem, peritonitis, bone and joint infections, osteomyelitis, spondylodiscitis [1,6,7,12,13]. Also described in the literature have been laryngological and oncology patients in whom pneumonia occurred on the etiology [2]. Another reported case concerns a case of infection in a patient with diabetes and hypertension [7]. *Rothia mucilaginosa* meningitis also caused meningitis in two children with leukemia after bone marrow transplantation [5]. Also described is a case of blood grow *Rothia muciulaginosa* in 3-year-old boy with Shwachman-Diamond syndrome. Boy was cured with rifampicin [16]. The pathogen also grew in 52-year-old man with granulomatous dermatitis who was subjected to chemotherapy for myeloid leukemia [8]. In 1988 the case of peritonitis of the *Rothia mucilaginosa* ethiology was recorded and described. The source of infection was a peritoneal catheter. This is again a case of a foreign body. The patient was cured with rifampicin and amoxicillin after 2 days of peritoneal fluid was clear [4]. All of the cases which are quoted in the literature describe patients with immune disorders or as an infection associated with the presence of a foreign body [4,7]. None of the cases described in the literature involved patients with no immune disorders. The question is why the infection occurred in our young patient. Treatment is a huge problem, especially when symptoms are present, but a microorganism responsible for infection cannot be cultured, as was in the described case. Perhaps, in the case of some "suspicious" pathogen, the time of culturing should be prolonged. Attention has been drawn to the need for antibiotic prophylaxis in the case of dental treatments, in order to prevent the development of infection. However, when choosing the antibiotics, it should be remembered that *Rothia mucilaginosa* may be resistant to penicilin [7,11]. There are still difficulties among microbiologists and clinicians in the diagnosis of infections of this type. The lack of reference laboratories, which should help in the diagnosis of the bacteria, is a problem, especially when we take into consideration that the infections occurred only in patients with impaired immunity. However, despite the diagnostic problems, the number of reports of infections caused by this bacteria is growing steadily.

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