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Case Report *Volume 4 Issue 1*

Bilateral Facial Cellulitis - Case Management in a Tertiary Hospital

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Abstract

Cutaneous cellulitis is a frequent condition in daily clinical practice, but its frequency of involvement varies with the affected site, being more common in the lower limbs and uncommon in the face. We will report the case of a 46-years-old female patient admitted to a tertiary hospital in the city of Sao Paulo, SP, Brazil, with the diagnosis of bilateral facial cellulitis with good clinical evolution after treatment with intravenous antibiotic therapy.

Keywords: Facial Cellulite; Antibiotic Therapy; Adults; Immunosuppressed

Abbreviations: ASO: Serology using anti-streptomycin; ADB: Anti-Deoxy Ribonuclease; BHS: Beta Hemolytic Staphylococcus; SIRS: Systemic Inflammatory Response Syndrome.

Introduction

Cellulite is characterized by the infection that occurs in the dermis and subcutaneous tissue through the entry of pathogens in these places due to ruptures in the skin barrier [1]. Clinically, it presents with limited erythema, heat, pain and local edema [1,2] and there may be drainage of purulent secretion through the lesion.

The frequency of involvement of the face is 4-24% while in the lower limbs it is 57%, upper extremities 16% and other sites 3% [3,4]. The etiology of facial cellulitis can be odontogenic or non-odontogenic, based on the source of the infection [5]. Complications can be associated with the involvement of the orbital or periorbital regions in addition to the risk, although rare, of involvement of the cerebral venous sinus [3].

The main etiopathogenic agent is Streptococcus beta hemolytic (BHS) [3,6] but with other possible agents in specific clinical conditions. Staphylococcus aureus is usually related to purulent secretion drainage, requiring specific antimicrobial coverage, in addition to the evaluation of possible methicillin-resistant strains (MRSA) [1].

The present case report aims to illustrate the case of an immunosuppressed patient admitted to a tertiary service having a diagnosis of bilateral facial cellulitis as well as its evolution during hospitalization.

Case Description

Patient C.T.B.S, female, 46 years old, white, natural and from Sao Paulo, Brazil, admitted to the infectious diseases ward of Complexo Hospitalar Heliopolis, in Sao Paulo. She has been diagnosed with HIV 11 years ago, in irregular treatment with tenofovir, lamivudine, darunavir and ritonavir, has viral load of 35,602 copies/ml and CD4 of 251; presented pulmonary tuberculosis in 2010, without adequate treatment.

One month after hospitalization she presented itchy eyes being self-medicated, with no improvement of the symptoms. Five days before hospitalization, she developed persistent high fever and painful nodulation in the left occipital region,

with edema in the parietal, frontal, orbital regions and auricular pavilions, associated with local phlogistic signs and bilateral purulent ocular secretion drainage. She has no visual changes, headache or any focal symptoms. The patient was admitted to the infectious disease ward, diagnosed with periorbital cellulitis and vancomycin was introduced in the dosage 2g a day. Two days after admission, she presented edema, erythema, local heat, drainage of purulent secretion, in addition to sero-hematic crusts in the bilateral malar region (Figure 1), no new febrile episodes, reduced leukocytosis and improvement of the aspect of the lesion present on the admission. She was evaluated by the dermatologic team, and two incisional biopsies were performed with a 4mm punch in the right preauricular region and the hypothesis of facial cellulitis was suggested, since, at this moment, the edema and the phlogistic signs were concentrated in the malar region bilaterally (Figure 2). On this occasion, the patient had difficulty opening the eye due to major edema in the malar region, without disturb in eye movement or diplopia. Edema in the occipital region, still present, associated with fine local and retroauricular desquamation.

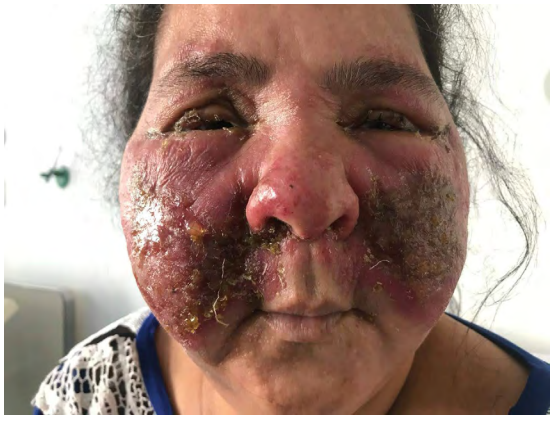

Figure 1: Two days after hospitalization.

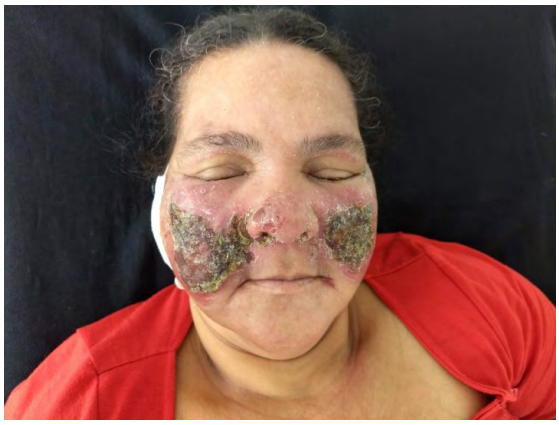

Figure 2: Five days after hospitalization and biopsy day.

Blood cultures, cerebrospinal fluid (due to two episodes of tonic-clonic seizure on the date of hospitalization), new serologies (hepatitis), rheumatological panel, viral loads for HIV and cytomegalovirus, CD4 and computed tomography of the skull were collected. There was no change in laboratory tests and in the image exam performed, and she has viral load of 314.15 copies/ml at that moment. The concentration of serum vancomycin was adjusted. There was a significant improvement in the lesion in the malar region and resolution of the edema previously presents (Figures 3 & 4). During hospitalization, the patient used vancomycin for 12 days, ceftriaxone for 5 days (introduced after the seizure event), intravenous acyclovir for 2 days and returned with the previous antiretroviral regimen.

Figure 3: Eight days after starting vancomycin treatment.

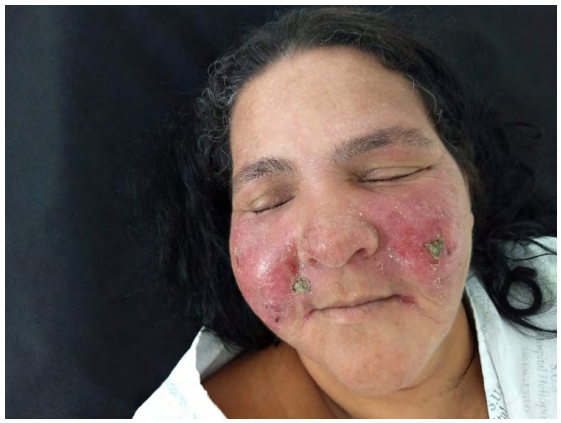

Figure 4: One day after finishing vancomycin treatment.

The report of the anatomopathological examination showed similar results in both samples: granulomatous folliculitis with suppurative foci and edema of the papillary dermis; the search for acid-resistant bacillus and fungi by the Ziehl-Nielsen, PAS and Grocott stains was negative in the samples.

Discussion

The clinical signs characteristic of cellulite, in general, are pain, heat, redness and edema with poorly defined limits with involvement of the dermis and subcutaneous tissue [1,2]. It is more frequent in the lower limbs, and facial involvement is uncommon [4]. Thus, when this site is affected, attention should be paid to the risk of complications such as thrombosis of the cerebral venous sinus, odontogenic or orbital infections, which may require surgical treatment [3]. Therefore, unlike other types of cellulite, when the face is affected, hospital treatment is recommended [3].

As for the etiopathogenesis, there is a predominance of beta-hemolytic Streptococcus, usually group A (Streptococcus pyogenes, in most cases), but with other possible groups (B, C and G) [7] in addition to Staphylococcus aureus, being commonly found on the skin surface as a colonizing pathogen [3,4]. Coverage for this agent is necessary when there is drainage of purulent secretion through the lesion [1,7,8]. Other pathogens may be related to facial cellulitis, such as Streptococcus pneumoniae, Haemophilus influenzae, Moraxella sp. and Pseudomonas aeruginosa, mainly in immunosuppressed people or with other associated infections such as sinusitis or orbital cellulitis, especially in children [1,2,7].

The diagnosis of cellulite is often made by clinical history and evaluation, especially in uncomplicated cases and without a risk factor [1]. Blood culture shows low levels of positivity, below 5% [8,9] as well as cultures performed both on skin biopsies and on lesion aspirates, with a variation, respectively, of 20% and 10% [10] being indicated in cases of neutropenic patients with cancer and undergoing chemotherapy, neutropenic patients with severe cell-mediated immunodeficiency and post-bite injury [3,1]. Culture of swabs collected on skin surfaces has low sensitivity and when obtained from chronic wounds or ulcers, they are generally polymicrobial and with multi-drug resistant pathogens that are not involved in the pathogenesis of cellulite [1,4]. Serology using anti-streptolysin (ASO) and anti-deoxyribonuclease (ADB) antibodies was more appropriate and more sensitive when compared to previous methods [2,4,9]. Therefore, some studies classify the etiology of cellulitis by beta hemolytic Staphylococcus (BHS) as confirmed (presence of positive serology (ASO and / or ADB) and / or growth of these bacteria in blood cultures or sterile tissue) or probable (growth of BHS in skin swab or satisfactory response to penicillin monotherapy) [4,6].

Factors such as: systemic involvement (fever, tremors and chills) [3]; criteria for systemic inflammatory response syndrome (SIRS), [7] and the presence of risk factors (immunosuppressed patients, children, extensive lesions) are necessary to guide the treatment of facial cellulitis.

In cases with absence of purulent drainage of the lesion and without systemic involvement, anti-streptococcal therapy (penicillins) can be used for a period of 5 to 10 days [7]. If purulent secretion drains from the lesions, the therapeutic coverage for Staphylococcus aureus (amoxicillin-clavulanate or cephalexin) should be expanded [1,7]. To evaluate the possibility of MRSA strains, especially in patients with risk factors (injecting drug users, children, previous infections by this agent, residents of long-term institutions, prisoners) [1,11] or in cases whose initial treatment did not respond after 24-48h2.6. In these cases, the coverage of antibiotic therapy for such pathogens (sulfamethoxazole-trimethoprim; vancomycin, clindamycin) should be expanded [1,7]. Empirical intravenous vancomycin therapy can be used in immunosuppressed patients or those with extensive skin lesions associated with signs of sepsis; patients with initial therapy failure or with SIRS criteria [2].

Facial cellulitis is an uncommon presentation in daily clinical practice, when bilateral is an even rarer entity. The diagnosis is usually clinical and the therapy is guided with the parameters of the patient's age, comorbidities involved, clinical presentation and risk factors for infection by methicillin-resistant Staphylococcus aureus. Faced with a patient with this region, the medical team needs to exclude orbital, periorbital, odontogenic and thrombotic involvement, in addition to complications such as cerebral venous sinus involvement. Thus, early diagnosis and treatment are essential for a favorable evolution of the condition.

Conflict of Interest: None in this Article.

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