Leprosy type-I reaction episode mimicking facial cellulitis-the importance of early diagnosis*

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Abstract: Leprosy is aneasily recognizable disease due to its dermato-neurological manifestations. It must be present in the physician's diagnostic repertoire, especially for those working in endemic areas. However, leprosy reaction is not always easily recognized by non-dermatologists, becoming one of the major problems in the management of patients with leprosy, as it presents clinical complications characterized by inflammatory process, accompanied by pain, malaise and sometimes the establishment or worsening of the patient's disabilities. We report the case of a patient with type-1 periorbital reaction admitted to the hospital, diagnosed and treated as facial cellulitis, whose late diagnosis may have contributed to the appearance or worsening of facial neuritis.
Keywords: Leprosy; Neuritis; Orbital cellulitis

INTRODUCTION

Leprosy is an infectious, chronic, and communicable disease caused by *Mycobacterium leprae*. It affects mainly the skin and peripheral nerves.[1]

Due to the ample clinical variations of the disease, the list of differential diagnoses is quite complex, often leading to delays in its identification and subsequent treatment.[1] Among the primary challenges, those are patients who initially present with type I or type II reactional episodes, thus requiring early diagnosis and institution of specific treatments in order to reduce the chance of developing physical disabilities and sequelae, which may negatively impact their social and professional lives.[2]

We present here the case of a patient who was primarily diagnosed with facial cellulitis and only later diagnosed with leprosy.

CASE REPORT

A sixty-seven-year-old male patient, married, born in Brejo Santo/CE, coming from Petrolina/PE, was admitted to the emergency room with a history of pruritus and redness in the left eye for the last 6 months. Also, 25 days prior to the admission,the patient developed periorbital edema and an erythematous plaque on the forehead and left periorbital region. He sought medical attention and was prescribed prednisone 60mg/day. At the time, he also reported numbness onthe left hand, associated with a lesion similar to the one on his face.The patient denied any systemic symptoms. Physical examination on admission showed an erythematous-infiltrated plaque reaching the left side of the forehead and edema on the periorbital region and nasal dorsum to the left (Figure 1). He was admitted with a diagnosis of periorbital cellulitis,

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prescribed treatment with Ampicillin + Sulbactam and laboratory tests were requested. Upon admission he presented 13.2g/dL hemoglobin; 39.4% hematocrit; 5,900 x10^9L leukocytes (67.9% neutrophils); 272,000 x10^9L platelets; 142mEq/L sodium; 5.2-mEq/Lpotassium; 19 mg/dL BUN; 1.0 mg/dL creatinine. The patient received antibiotic treatment for 10 days without clinical improvement, hence a CT scan was ordered and the diagnostic hypothesis was once more facial cellulitis, which prompted a proposal for antibiotic change (Figures 2 and 3). Nonetheless, a sensitivity test was performed on the plaque area and loss of thermal sensitivity was detected all over the lesion, as well as on the hand lesion, leading to the diagnosis of borderline tuberculoid leprosy (BTD) in reaction.

Bacilloscopy of the lesions was negative. Both lesions were biopsied and anatomopathological results revealed periadnexal and perineural granulomatous chronic dermatitis. With an established diagnosis of BTD in reaction, the patient was prescribed prednisone 60 mg/day and referred to a dermatologist, who started treatment with specific multibacillary-polychemotherapy (rifampicin, clofazimine and dapsone). Prednisone 60mg/day was maintained, with gradual tapering off after the reaction abated and in 15 days the patient showed improvement (Figure 4). Forty-five days after the treatment was started the patient presented marked recovery, although the presence of lagophthalmos on the left side was noticed, prompting physical therapy and close monitoring (Figure 5).

DISCUSSION

Reaction episodes are acute phenomena affecting skin and peripheral nerves. They may occur before, during or after the treatment and are the leading cause of nerve damage and disability secondary to leprosy.[3]

Reactions might be of type I, also known as reverse reaction or type II, namely erythema nodosum. Type I reactions present signs of acute inflammation such as pain, erythema, infiltration and edema of pre-existing lesions; also, the appearance of new lesions, papules and plaques (usually erythematous and adjacent to pre-existing lesions) may occur. Lesions may be single or multiple and are oftenulcerated. [3,4] Reactions occur in dimorphic leprosy forms (DL) such as borderline tuberculoid (BT), borderline (BB), and borderline lepromatous (BL). Type II reactions or

Figure 1: Erythematous-infiltrated plaque on the left region of the forehead, eyelids and nasal dorsum, at diagnosis

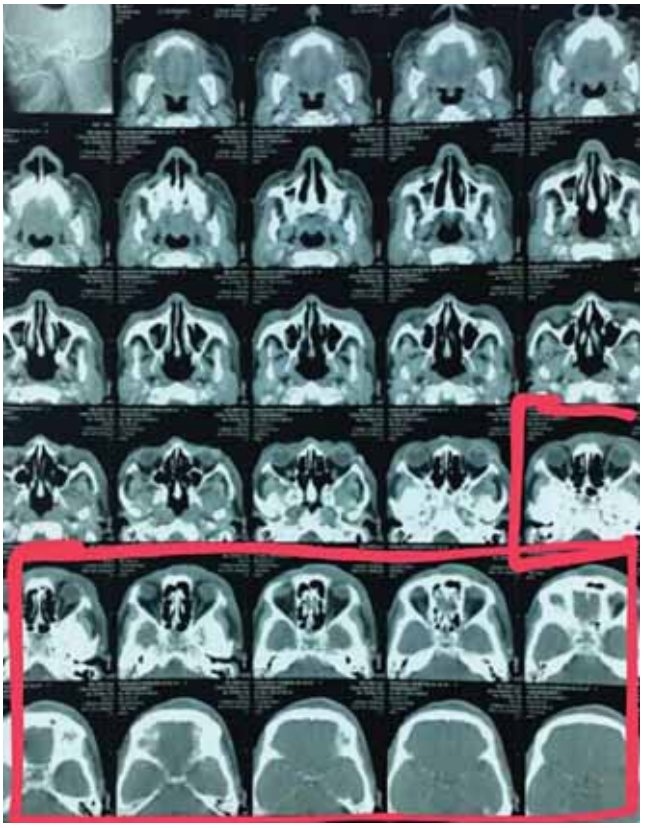

Figure 2: Facial CT scan: thickening with signs of enhancement by contrast on superficial plans/soft tissues of the left periorbital region, extending to the ipsilateral frontal region. Compatible with periorbital cellulite

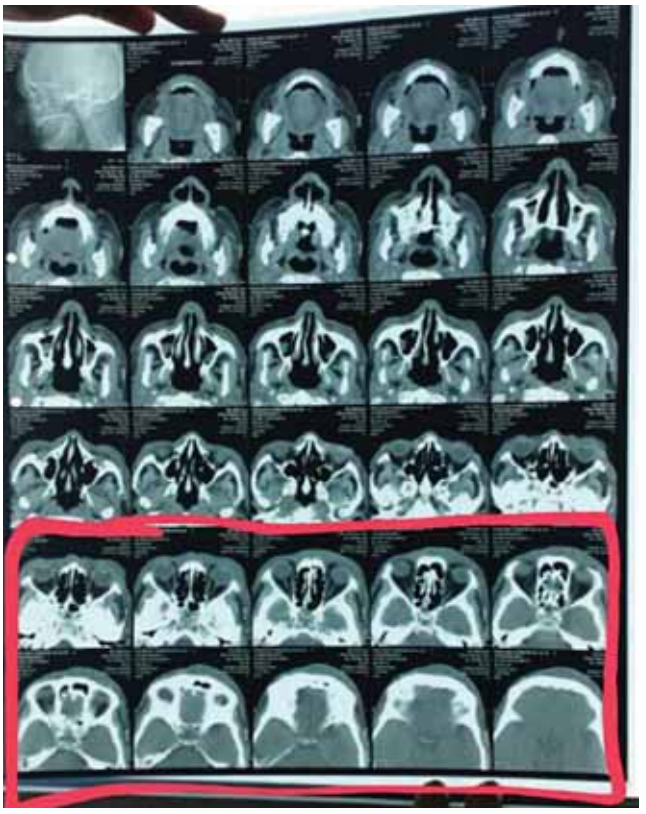

Figure 3: Thickening of theleftperiorbital region, compatible withperiorbitalcellulitis,is observed. No signs ofbone involvementorcollections

Figure 4: Partial regression of lesions after 15 days of treatment

erythema nodosum are associated with general symptoms such as fever, painful lymphadenopathy and malaise, manifesting as papules or painful, erythematous nodules. They occur in lepromatous (LL) and borderline lepromatous (BL) forms. [4]

In type 1 reaction, presented by the patient in this report, there are manifestations that can mimic erysipelas, cellulitis, drug eruptions, urticaria, psoriasis, sarcoidosis, lymphomas, sudden peripheral facial paralysis and even leprosy relapse itself.[5] The complete mechanisms involved in its genesis are yet unknown.It is accepted, however, that it results from an imbalance between pro-inflammatory cytokines (gamma interferon and interleukin-2, mainly) and anti-inflammatory cytokines (especially TGF beta and interleukin 10). [6]

In the clinical case in question, the patient was initially diagnosed and treated as facial cellulitis, because of the intense inflammatory process he presented, without taking into consideration the reported paresthesia or the lesion on the left hand, which was similar to the facial one. Due to the lack of improvement after the therapy was introduced, bacilloscopies of the lesion and conventional sites (earlobes and elbow) were later performed with negative results, as

Figure 5: Regression of lesions, although with the presence of lagophthalmos, 40 days after diagnosis

frequently occurs in BTD forms. Biopsies of the lesions revealed granulomatous dermatitis with leprosy in reaction as the primary diagnosis. The delayed diagnosis probably contributed to the neurological impairment. Even though Brazil is a country with high levels of endemicity, many typical cases are only diagnosed in polarized forms and after sequelae have already been established.[7]

In endemic areas, leprosy reaction is the first diagnosis to be suspected in cases like this (reactive form of BT) in which the initial lesion consists of an erythematous plaque on the innervation area of the facial nerve, resulting in neuritis (also common in reactional states) and subsequent lagophthalmos. Among leprosy patients, those with BT form usually develo-preactional states earlier and with greater neural involvement, which requires diagnosticand therapeutic agility in order to prevent disabilities such as those developed by ourpatient.[8] ❑

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