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Fatal necrotizing fasciitis following a non-treated mandibular fracture: case report

Fasciíte necrotizante fatal devido a fratura mandibular não tratada: relato de caso

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ABSTRACT

Necrotizing fasciitis is a rare and severe infection characterized by extensive and quickly progressing necrosis of the subcutaneous tissue and muscle fascia associated with high mortality rates in the head and neck region. We present a case of fatal necrotizing fasciitis due to an untreated mandibular fracture. Eight days after the trauma, the patient was admitted to the hospital and died on the sixth day of hospitalization.

Indexing terms: Case report. Mandibular fractures. Necrotizing fasciitis

RESUMO

Fasciite necrosante é uma infecção rara e grave caracterizada por necrose extensa e de rápida progressão do tecido subcutâneo e fáscia muscular associada a altas taxas de mortalidade na região da cabeça e pescoço. Apresentamos um caso de fasciite necrosante fatal devido a uma fratura mandibular não tratada. Oito dias após o trauma, o paciente deu entrada no hospital e faleceu no sexto dia de internação.

Termos de indexação: *Relato de caso. Fraturas mandibulares. Fasciíte necrotizante.*

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INTRODUCTION

The necrotizing fasciitis (NF) is a rare, severe, devastating, and fulminating infection, characterized by necrosis of the fascia and adjacent soft tissue with quick progression and rapid expansion along the fascial planes [1].

When the NF occur in the area below the border of the mandible, above the clavicle, and anterior to the trapezius, it is called cervical necrotizing fasciitis (CNF) [2].

There are few reported cases of CNF developing after mandibular fracture [3, 4], causing this report crucial for professionals who treat maxillofacial trauma.

We present a case where the patient postponed the search for trauma assessment, resulting in fatal CNF due to an untreated mandibular fracture. This case report was prepared following the CARE Guidelines [5].

CASE REPORT

A 26-year-old black man was admitted to the emergency hospital with pain in the left hemiface, cervical and thoracic region. He reported a previous untreated mandibular fracture nine days ago. The patient was eupneic, flushed, conscious, oriented, afebrile, and had isochoric and photo reagent pupils. He denied deleterious habits and vices, other comorbidities, and drug allergies.

The facial examination exhibited bilateral submandibular hematoma, sialorrhea, halitosis, poor oral hygiene, trismus, dental malocclusion with mobility, and crackling in the left mandibular body. The Computed Tomography showed complete fracture with subcutaneous emphysema in the adjacent soft tissues.

The laboratory tests showed leukocytosis (11.5 mm3). After seven hours of hospitalization, the patient worsened with tachypnea and respiratory failure, with a saturation of 78%. Thus, the patient was referred to the intensive care unit, where orotracheal intubation was performed.

The next day, the patient was taken to the operating room for cervicectomy, debridement, and drainage of the cervical abscess. During the surgical procedure, the diagnosis of CNF was made due to extensive areas of necrosis of the subplatismatic muscle fascia and deep cervical planes (figure 1). Thus, debridement and necrosectomy of several muscular planes and deep fascia were performed.

Antibiotic therapy was made with gentamicin (80mg 8/8 hours) associated with the meropenem (1g 8/8 hours) and vancomycin (500mg 12/12 hours). Laboratory tests showed immature neutrophils at 21% and urea at 48mg/dL.

On the first postoperative day, the patient had sepsis. He was intubated, on mechanical ventilation, hemodynamically unstable, and receiving a noradrenaline vasoactive drug (80 ml/h).

On the second postoperative day, the laboratory tests demonstrated the values: leukocytes (10.5 mm3), immature neutrophils (13%), urea (109 mg / dL), creatinine (2.47 mg / mL) and CRP (448.45 mg / L). In the cervical region, a foul odor and the presence of a necrotic area were noted. The previous antibiotic therapy was suspended and replaced with the prescription of oxacillin (2 g 8/8 h), metronidazole (500 mg 8/8 h), and cefepime (2 g 8/8 h).

On the third postoperative day, the patient had central and peripheral cyanosis with slow capillary filling time. He showed closed extremities and received noradrenaline and adrenaline. The cervical area, with muscles exposed, showed improvement without pus, necrosis, bleeding, and granulation tissue. The team suspended this antibiotic therapy and returned to the initial treatment with an increase in the intravenous dosage of gentamicin (500mg 8/8 h), meropenem (2g 8/8 h), and vancomycin (2g 8/8 h).

On the fourth day, the patient suffered a reversed cardiorespiratory arrest during the night, followed by tachycardia, fever, slowed peripheral perfusion, paleness, and a slightly distended abdomen. He remained in continuous moderate sedation, intubated on mechanical ventilation, and without bowel movements throughout the day. During the night, a new cardiorespiratory arrest occurred, leading to death (figure 2).

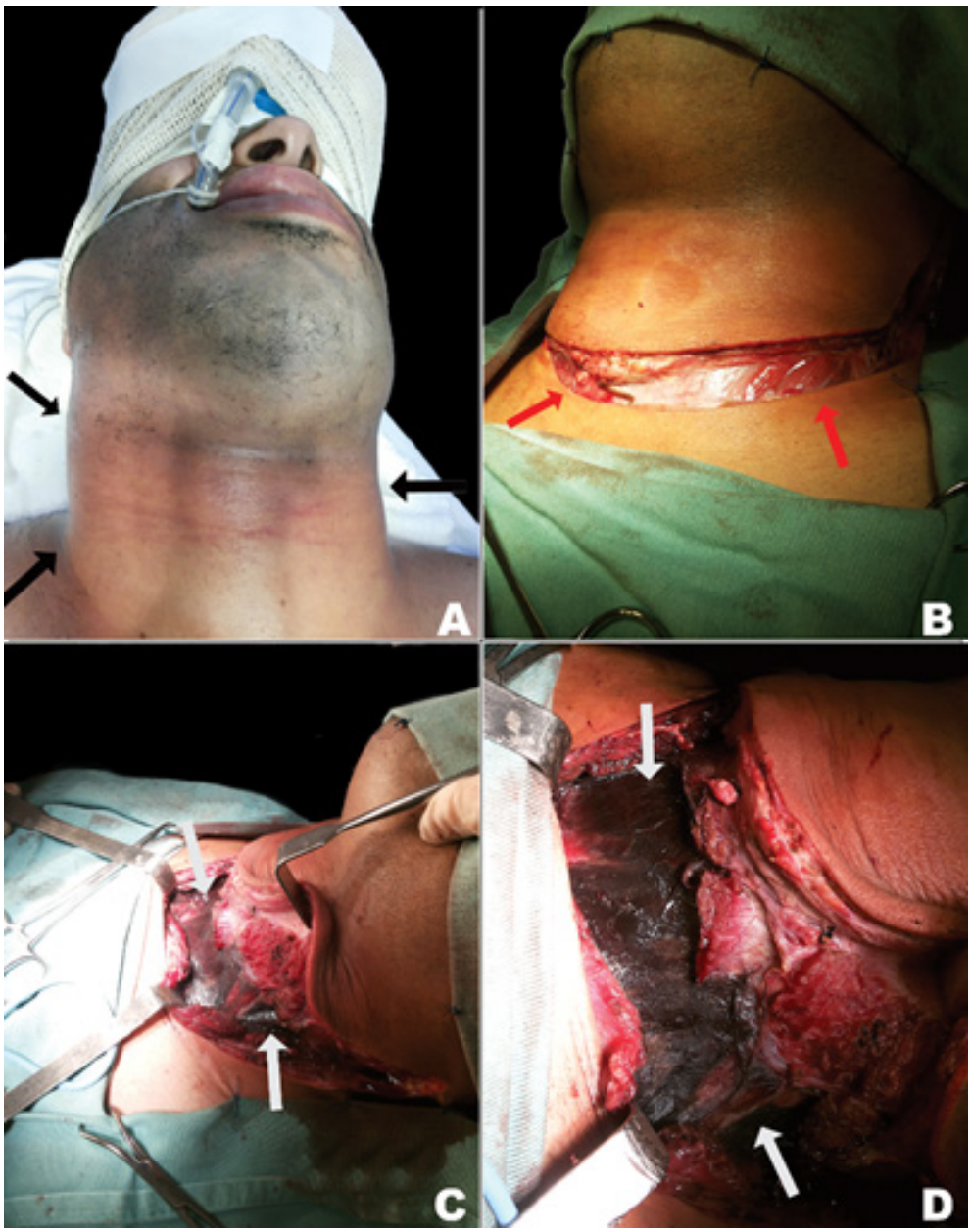

Figure 1. A) Edema observed in the cervical region (black arrows). B) Cervical incision made for access (red arrows). C and D) Exposure and a wide range of necrotic tissue (white arrows).

The result of the microbiological culture exam was performed from the collection on the second day of hospitalization. The result showed the presence of Staphyloccocus hominis Subsp. hominis. This strain was having sensitivity to daptomycin, linezolid, rifampicin, synercid, tetracillin, and vancomycin. However, the test result was only available to the team after five days of collection.

DISCUSSION

This rare case showed the importance of early diagnoses and treatment for CNF. The patient's delay in seeking treatment for a sporting accident contributed to the negative outcome. Besides, diagnosing the early stages of CNF

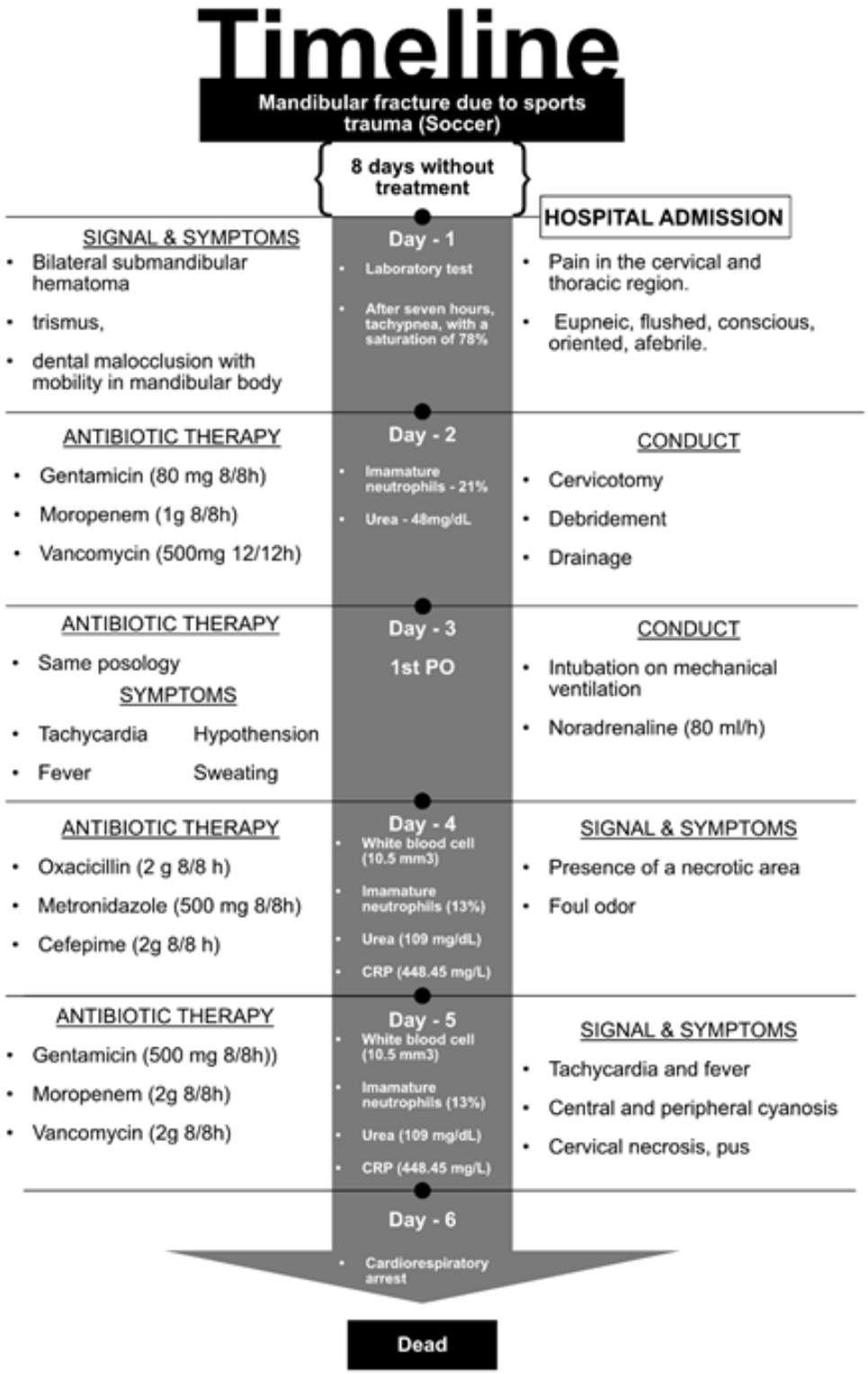

Figure 2. Timeline.

concerning other soft tissue infections of odontogenic origin is difficult and often leads to less aggressive treatment, resulting in increased morbidity and mortality [6]. With disease progression and nutrient and vascularization loss, the skin becomes dark, and necrosis sites become apparent. In our case, the initial edema was compatible with mandibular fracture associated with cellulitis without any dark coloration that could evidence FN diagnosis.

Upon admission, the patient was afebrile, which corroborates that 60% may not have a fever [7]. Other symptoms, such as erythema, pain, and edema, were presented in the case and related to the diagnosis. Also, bullae, crepitus, skin necrosis, hypotension are signs that may be present [7], but in the initial evaluation, they had not manifested, making it more challenging to diagnose the entity.

The laboratory risk indicator for necrotizing fasciitis (LRINEC) score can be valuable for early diagnosis [8]. The tool is based on six standard serum parameters: C-reactive protein (CRP), total white cell count, hemoglobin, serum sodium, creatinine, and glucose. Despite its controversial use in literature, a systematic review [9] demonstrated that the LRINEC score is a helpful adjunct in the clinical diagnosis of necrotizing fasciitis with a statistically positive correlation. In addition, a modification in the score was proposed with the addition of clinical parameters such as pain, fever, tachycardia, and comorbidities.

The CNF is extremely rare due to the robust blood supply of the cervical area. A large percentage (97.1%) of patients have a past medical history. The association with diabetes mellitus (18.18%), alcoholism (9,09%), underlying malignancy (3%), corticosteroid therapy (1.5%), and HIV (1%) was found in a systematic review and analysis of 1235 reported cases from the literature [2]. Although CNF is more common in obese individuals and the immunocompromised, they can affect healthy individuals without risk factors [10,11].

CNF can be caused by odontogenic infection (47%), pharyngolaryngeal (28%), tonsillar (6%), traumatic, iatrogenic, or post-operative (4%) salivary gland (2%), skin (1%) between others [2]. There are few cases reported in the literature whose cause is a mandibular fracture [3,4]. Of these, Chin et al. (1995)[4] had a fatal outcome, and however, unlike our case, the patient was severely immunocompromised elderly. Nevertheless, the treatment was also delayed.

Among the microorganisms identified in the CNF, staphylococcus was found in second place, corresponding to 18.09% of patients. The most common was streptococcus in 61.22% of cases [2]. In this case report, staphylococcus hominis was isolated.

The overall mortality is considered high, ranging from 13.36% [12] to 18% [10]. Wang et al [12] proposed a three-stage classification of FN, with the signs and symptoms to the initial stage corresponds to only cutaneous symptoms (tenderness, erythema, and swelling). In the intermediate stage, the patient presents blister or bullae formation (serous fluid), and the last stage (late) presents crackling, skin anesthesia, and necrosis with dusky discoloration [12]. The evolution to the third and last stage (sepsis) was rapid.

CONCLUSION

The mandibular fracture can be an etiology of CNF who has a high mortality. The definitive diagnosis must be rapid and the treatment aggressive. The patient's delay in seeking treatment contributed to the fatal negative outcome.

Collaborators

FERREIRA MS participated in the data collection and writing the manuscript.; ARAUJO CFSN, DIB JE, DIB VBE, DIB MBE responsible for the clinical conduct and data collection; SILVA MAG supervised the writing and final analysis of the manuscript. All authors reviewed the text and approved its final version.

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