CLINICAL FEATURES OF ORO-MAXILLOFACIAL NECROTIZING FASCIITIS: A CASE REPORT

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ABSTRACT

Aim: To review current concepts and clinical features of the facial necrotizing fasciitis (NF), with the objective of optimizing the accuracy of early therapeutic intervention. **Materials and Methods:** This clinical study was conducted to analyze the specific clinical characteristics of descending necrotizing cervico-facial fasciitis within the Department of Oral and Maxillofacial Surgery of Emergency Institute. An important inclusion criterion was the identification of a correlation between the topographic localization,number of involved spaces, and t mechanism of infection spreading through the fascial compartments. **Results and Discussion**:The present case illustrates the convergence of clinical, imaging, and laboratory criteria characteristic of descending necrotizing cervico-facial fasciitis developing in a septic context. The correlation between rapid clinical progression, imaging-documented fascial involvement, and biological markers of systemic inflammation supports the mechanism of deep dissemination of the suppurative process along cervical anatomical planes. **Conclusions:** The review of current concepts regarding facial necrotizing fasciitis highlights that therapeutic success relies on the integration of clinico-biological algorithms. A multidisciplinary approach, iterative surgical intervention, and targeted antibiotic therapy are the fundamental pillars of modern management.

Key words: phlegmons, odontogenic infection, necrotising fasciitis, surgical debridement

INTRODUCTION

Necrotising fasciitis is characterized by its fulminating, devastating, rapidly-progressing, and generalized necrosis of the superficial fascial layer and the involved cutaneous tissue. Necrotising fasciitis occurs more commonly in patients with compromised immune systems, and more frequently in the abdominal wall, perineum, and extremities. Involvement of the head and neck structures, especially the scalp, is rare. In Brunworth's review, in most major medical centers, the frequency of dental infection resulting in craniocervical NF is approximately 1.2 cases per year.

Necrotizing fasciitis has a long history, being decribed for the fisrt time by Hippocrates in the 5th century [2]. The term literally means "decaying infection of the fascia". The estimated incidence in the western world is about 0.24–0.4/100 000 people per year and it is ranked among most difficult emergencies encountered by healthcare providers [3]. However, in some areas of the world, necrotising fasciitis is more common and more than one case for 100 000 people is reported [4].

Necrotising fasciitis has multiple causes, risk factors, anatomical locations, and pathogenic mechanisms, and results in widespread tissue destruction, which may extend from the epidermis to the deep musculature. Mortality is high and even the survivors have a long clinical course. An early clinical suspicion and diagnosis are vital to the outcome, but an

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accurate diagnosis is reached in only 15% to 34% of patients at the time of presentation [5].

The disease occurs when the right set of conditions are present; in approximately 80% of all the cases, these include a disruption of skin's integrity that allows bacterial infection, such as surgical wounds,animal bites, lacerations, scratches, burns, minor invasive procedures (joint aspiration, acupuncture), intramuscular injection, and folliculitis [6].

Necrotising fasciitis is observed particularly in cases with comorbidities and risk factors include diabetes, chronic diseases, immunosuppression ,malnutrition, advanced age, non-steroidal anti-inflammatory drugs (NSAID) use, morbid obesity, liver cirrhosis, intravenous drug misuse, alcoholism, peripheral vascular disease, chronic renal failure, immunological disorders, HIV–AIDS, paraplegia, underlying malignancy and varicella infection [7], [8].

In head and neck surgery, it is paramount to consider possible variations of the blood vessels, including variants of the external carotid artery branching patterns [9],[10] and those of the neighboring cranial nerves. Considering the high impact of facial disfigurations on peoples' life quality a particular attention should be paid in dissection of the facial nerve trunk and its extracranial branches, characterized by a wide range of anatomical variation [11],[12],[13].

MATHERIALS AND METHODS

This investigation was designed as a retrospective cohort study aimed to characterize the clinical features, disease evolution, and severity indicators of descending necrotizing cervicofacial fasciitis managed within the Department of Oral and Maxillofacial Surgery at the Emergency Medicine Institute. A epidemiological study was conducted, which included 1635 patients admitted to the OMF department during the period from January 1, 2022, to December 31, 2025, diagnosed according to ICD-10 codes L03.2 and M72.5, with cervicofacial inflammatory processes. In line with the eligibility criteria, a cohort of 765 patients diagnosed with phlegmons from which 24 were diagnosed with necrotizing fasciitis in the oro-maxillofacial region were selected.

In the presented case the diagnosis of cervicofacial necrotizing fasciitis was established on the basis of combined clinical evaluation, radiological findings (contrast-enhanced computed tomography), and intraoperative assessment.

Data extraction were focused on demographic parameters (age, sex), associated systemic conditions, interval between symptom onset and hospital admission, anatomical extent of fascial involvement, number of affected cervical spaces, evidence of mediastinal spread, laboratory inflammatory markers at presentation (including leukocyte count, C-reactive protein, and procalcitonin), and the hospitalization period, requirement for intensive care support, number of surgical debridements, and final outcomes.

RESULTS AND DISCUSSIONS

Infections of the inferior second and third molars are the most frequent causes of odontogenic cervical necrotizing fasciitis [9]. Wong's study also indicated the lower molars as the most common regions [10], and Whitesides et al. [11] revealed up to 81% of patients with cervical necrotising fasciitis caused by infections of the second and third molars. These teeth deepen into the mylohyoid insertion over the lingual side of the mandible. If untreated, infection that originates from these teeth can easily extend to the submandibular space, with subsequent dissemination into surrounding spaces, including sublingual, submental, and parapharyngeal spaces. Ultimately reaches skull base with cephalad extension, or caudally into the mediastinum and thoracic cavity[12].

Out of 6.988 patients hospitalized in the Department of Oral and Maxillofacial Surgery, 765 cases (10.95%) presented diffuse inflammatory processes, highlighting the substantial clinical burden of cervicofacial

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infections in maxillofacial surgical practice.

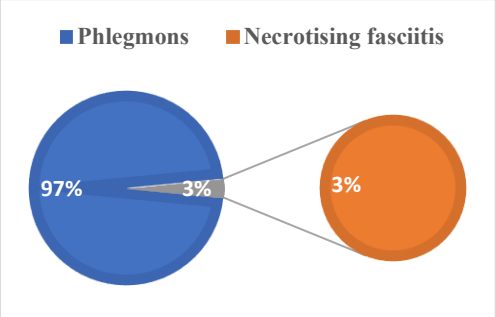

Figure 1. Distribution of necrotising fasciitis

The distribution presented in Fig. 1 demonstrates that phlegmonous infections constitute the predominant form of diffuse inflammatory pathology within the oral and maxillofacial region, whereas necrotizing fasciitis represents only a small fraction of cases-3%. From a pathophysiological perspective, the transition from localized inflammatory processes to necrotizing soft tissue infection reflects a complex interaction between microbial virulence, host immune response, and anatomical pathways of fascial dissemination.

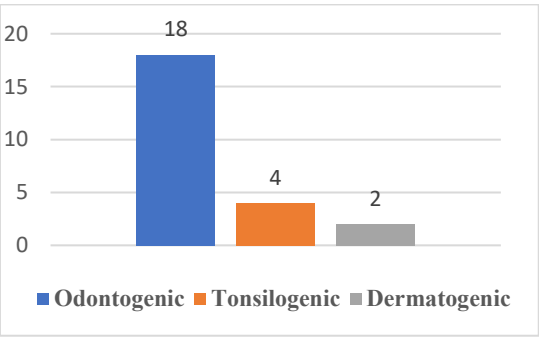

Figure 2. Etiology of necrotising fasciitis

As shown in Fig. 2, necrotizing fasciitis in the oral and maxillofacial region was predominantly of odontogenic origin-18 cases, whereas tonsillogenic and dermatogenic sources were observed far less frequently. This distribution underscores the pivotal role of dental infections as primary triggers of cervicofacial necrotizing soft tissue infections and highlights the propensity of odontogenic foci to propagate along cervical fascial planes, facilitating rapid disease progression and systemic involvement.

A 68-year-old male patient presented to Emergency Hospital with complaints of severe pain in the right hemifacial region, fatigue, apathy, pain on swallowing and mastication, limited mouth opening, and chills.

According to the patient, dental pain originating from tooth 45 had begun approximately six days prior to admission. He self-administered anti-inflammatory medication but did not seek dental or medical evaluation. Three days prior to admission, he noticed progressive limitation of mouth opening. Two days before presentation, odynophagia developed, followed by dysphonia one day prior to hospitalization.

On admission, the patient's general condition was deteriorated, with a toxic appearance and fever, accompanied by signs of systemic inflammatory response. He adopted an antalgic posture and presented with mild to moderate dyspnea, muffled voice ("hot potato voice"), and dysphagia.

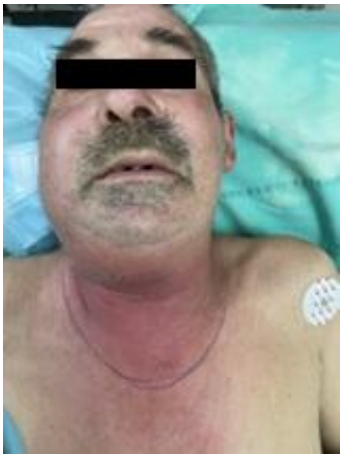

Figure 3. Exobuccal aspect

Local cervico-facial examination (fig.3) revealed marked facial asymmetry due to left-sided hemifacial edema extending from the left temporal region to the jugal and left submandibular areas, with further spread toward the anterior cervical region. The overlying skin was intensely hyperemic, glossy, and tense. There was obliteration of the mandibular angle

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contour and the submandibular groove. A mild left palpebral ptosis was observed, secondary to extensive edema. On palpation, a firm, "wooden" induration ("board-like" consistency) was noted. Fluctuation was absent, and local crepitus was detected. Mouth opening was limited.

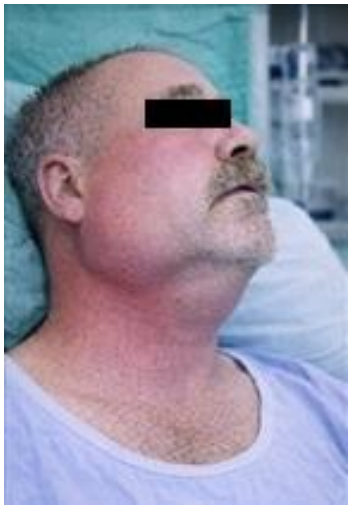

Figure 4. Lateral profile view

Intraorally, the buccal and labial mucosa were edematous, bearing dental impressions and covered with pseudomembranes. Saliva was viscous, and fetid halitosis was present. The oropharyngeal isthmus was narrowed on the right side. Severe trismus was documented, with a maximal interincisal opening of 1.0 cm, accompanied by grade III dysphagia and dysphonia. Tooth 45 presented coronal destruction and was tender to percussion. The patient's general condition was severe, with a body temperature of 38.5°C, blood pressure of 96/68 mmHg, and a heart rate of 130 bpm.

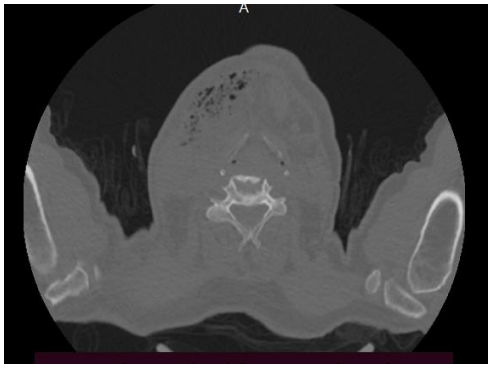

Figure 5. Imaging study- axial view

Multiplanar CT examination (sagittal, axial, and coronal reconstructions) demonstrated extensive inflammatory changes in the cervico-facial region, suggestive of a deep infectious process with fascial extension.

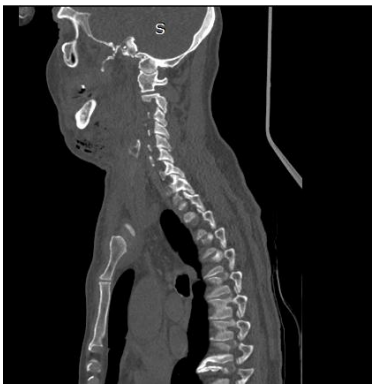

Figure 6. Imaging study-sagittal profile

Marked inflammatory involvement was observed in the submandibular, infratemporal, submental, and deep cervical fascial compartments (fig.6). Multiple hypodense areas associated with intratissue air collections (gas locules) were identified within the fascial planes. Inferior extension of the inflammatory process along the anterior visceral compartment was also noted.

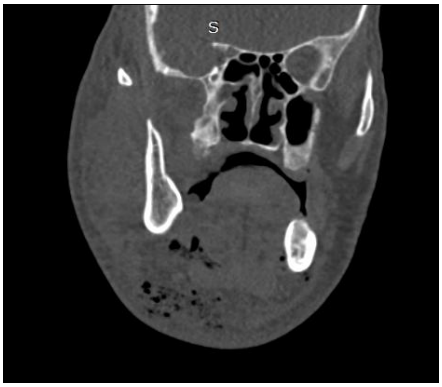

Figure 7. Imaging study coronal views

Laboratory investigations revealed high leukocytosis (24.5 × 10^9/L), elevated C-reactive protein (278.76 mg/L), procalcitonin level of 14.65 ng/mL, and erythrocyte sedimentation rate of 67 mm/hour, findings consistent with septic status.

Based on the clinical and paraclinical

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examinations, the diagnosis was established as: right-sided odontogenic hemifacial phlegmon involving the floor of the mouth and right lateral cervical region originating from tooth 45, complicated by necrotizing fasciitis and septic state.

A decision was made to initiate comprehensive management consisting of both surgical intervention and intensive medical therapy. Considering the clinical findings suggesting a difficult airway for endotracheal intubation, an open inferior tracheostomy was performed to ensure airway protection. The surgical protocol for wound management involved a horseshoe-shaped incision of approximately 10 cm (fig.4), encompassing the skin, subcutaneous tissue, adipose tissue, and fascial planes.

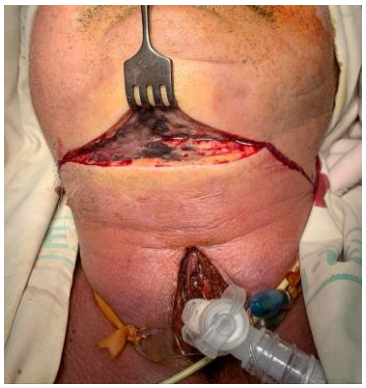

Figure 8. Intraoperative view

Intraoperatively, extensive fascial necrosis was identified (fig.8), characterized by gray-black devitalized tissue with loss of normal fascial luster, marked friability, and absence of bleeding upon incision. The necrosis extended along the fascial planes, involving both the superficial cervical fascia and the layers of the deep cervical fascia, findings suggestive of an aggressive infection with rapid dissemination. The fasciae exhibited a friable consistency and discoloration, indicative of thrombosis of the supplying microvasculature, associated with both liquefactive and coagulative fascial necrosis.

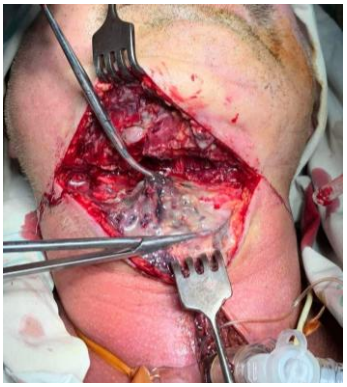

Figure 9. Necrotizing fasciitis

In Fig. 9, the fasciae appear edematous, friable, and profoundly structurally altered, with absence of normal fascial sheen and elasticity. Multiple microabscesses and deposits of necrotic tissue (necrotic slough) are visible within the fascial planes. Areas of liquefaction associated with purulent exudate and serohemorrhagic infiltration were present. Patchy zones of coagulative necrosis interspersed with inflammatory debris were observed, with rapid extension throughout all cervical fascial compartments.Pathological material was collected intraoperatively for microbiological analysis to determine the bacterial profile.

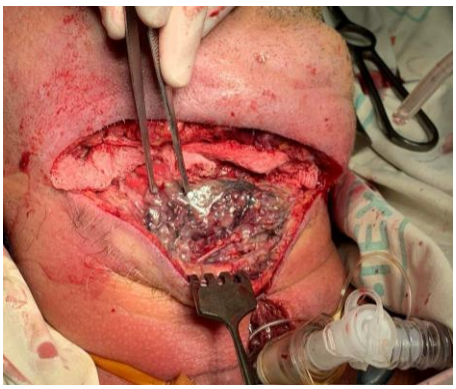

Figure 10. Excisional debridement

Fig.10 illustrates an advanced intraoperative stage of surgical debridement in the context of extensive cervicofacial necrotizing fasciitis.The operative field demonstrates loss of normal fascial architecture and structural integrity, associated with the presence of a thick layer of necrotic tissue exhibiting a glossy, liquefactive appearance. The

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necrotic fascia appears avascular, and patchy areas of coagulative necrosis coexist with liquefactive changes, suggesting a mixed mechanism of tissue destruction involving both ischemic and infectious components.

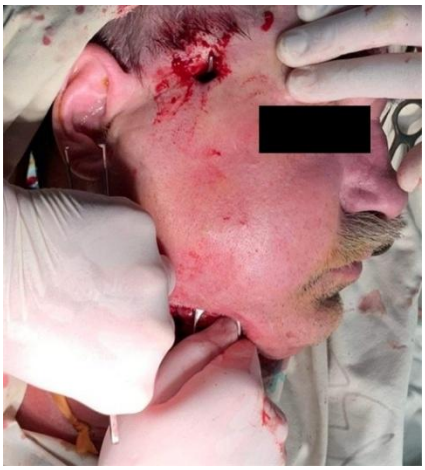

Figure 11. Drainage of the infratemporal space

Blunt dissection was performed to access the lateropharyngeal, pterygomandibular, and bilateral submandibular spaces, as well as the base of the tongue (fig.11). Grayish, fetid, serosanguinous pus was obtained and collected for microbiological analysis.

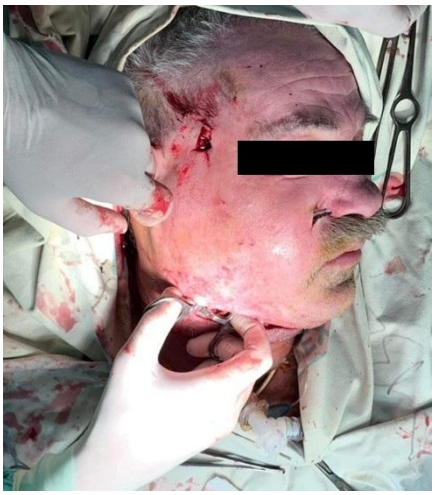

Figure 12. Drainage of the buccal space

Manual exploration revealed cavities extending toward the buccal, submasseteric and infratemporal spaces (fig.12). Consequently, radial skin incisions of approximately 4.0 cm each were performed. Approximately 10 ml of fetid, serosanguinous purulent discharge was evacuated, along with gas bubbles suggestive of anaerobic metabolism. Microbiological examination of the pathological material collected from the wound demonstrated the presence of *Streptococcus group G* in a significant concentration (10^5 CFU/mL), suggesting its major etiological role in the septic process. Antimicrobial susceptibility testing revealed a favorable susceptibility profile, with the isolated strain being sensitive to all antibiotics tested and no evidence of acquired resistance mechanisms.

Complications: Bilateral polysegmental pneumonia. In the context of a severe clinical presentation and suspicion of polymicrobial odontogenic infection, broad-spectrum intravenous antibiotic therapy was initiated, consisting of ceftriaxone, metronidazole, and gentamicin. This combination is justified according to the principles recommended by the IDSA guidelines for severe skin and soft tissue infections, which advocate the early initiation of broad-spectrum antimicrobial therapy in suspected necrotizing fasciitis, pending definitive culture and susceptibility results, in conjunction with aggressive surgical debridement [13].

The patient received intensive care treatment in the intensive care unit for 24 days, with daily surgical wound irrigation and debridement procedures. Following improvement in the general condition, he was transferred to the specialized department for continuation of treatment.

In order to avoid prolonged surgical procedures and massive blood loss, planned and staged debridement of the affected regions should be performed following the initial drainage of the suppurative process. Frequent surgical drainage and repeated debridement represent key elements in achieving favorable outcomes [14]. The present case illustrates the convergence of clinical, imaging, and paraclinical criteria characteristic of descending necrotizing cervico-facial fasciitis in a septic

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context. The correlation between rapid clinical progression, imaging-documented fascial involvement, and biological markers of systemic inflammation supports the mechanism of deep dissemination of the infectious process along cervical anatomical planes. The septic evolution confirms that descending fascial spread does not represent merely a local phenomenon, but rather reflects a systemic process with major hemodynamic and metabolic impact.Conclusions

1. The review of current concepts regarding facial necrotizing fasciitis highlights that therapeutic success relies on the integration of clinico-biological algorithms.
2. In odontogenic cases, extension into cervical compartments and the presence of sepsis necessitate early aggressive surgical management, combined with broad-spectrum empirical antibiotic therapy and intensive care monitoring.
3. Source control through repeated debridement and dynamic reassessment remains the therapeutic gold standard.
4. This case confirms that cervico-facial necrotizing fasciitis is not merely an extensive local infection, but a rapidly evolving systemic pathology in which dissemination along fascial planes represents the expression of microbial aggression associated with microvascular dysfunction and a severe systemic inflammatory response.

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