

Cervical facial necrotizing fasciitis with mediastinic spread from odontogenic origin

Two case reports



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Cervical facial necrotizing fasciitis with mediastinitis spread from odontogenic origin. Two case reports

Necrotizing fasciitis is an uncommon soft tissue infection, usually caused by toxin-producing virulent bacteria especially in mediastinum. It is characterized by widespread fascial necrosis primarily caused by Streptococcus hemolyticus characterized by necrosis of skin, subcutaneous tissues, fasciae, and muscles. It usually occurs in adults and is most often localized to the abdominal wall, the extremities, the perineum, the pelvis, and the thoracic region. Localization to the head and neck area is rarely encountered. Descending necrotizing mediastinitis is a form of mediastinitis caused by odontogenic infection or deep cervical infections, which spreads to the mediastinum from the cervical fascial planes. Early diagnosis, prompt surgical drainage, monitoring of disease process, appropriate medical management in an intensive care unit and a multi-disciplinary approach can significantly reduce the mortality in this otherwise fatal condition.

KEY WORDS: Descending necrotizing mediastinitis, Necrotizing mediastinitis

Introduction

The descending necrotizing cervical mediastinitis are an acute polymicrobial infections originated from connective tissue more often of the oropharynx and neck. This is a very serious disease with severe complications that often lead to death with a reported incidence in the literature between 25% and 40% (Freeman Table Ia) ¹. Therapy should be quick and effective and provides not only great medical treatment but also surgical drainage, cervical and sometimes tracheotomy, and thoracotomy drains and \ or mediastinal drains ².

TABLE IA - Cumulative mortality of DNM in literature (Freeman)

	N° of Reports	N° of Patients	Survivors	Mortality (%)
Pearse (1938)	1	37	18	49
1970-79	8	13	9	31
1980-89	11	26	16	38
1990-98	27	57	43	25

TABLE IB

Perimandibular- submandibular space	21
Buccal space (4) and parotid –masseter space (2)	6
Pterygium-mandibular joint and masseter lodge	3
Submental lodge	4
Sublingual lodge	4
Retromaxillary lodge	2
Tonsillar and peritonsillar lodge	1
Lateral cervical Space (carotid-jugular 1) -->1 mediastinum	2
Temporal lodge	1
Orbital cavity (2) and sino-nasal cavity(6)	8
Parapharyngeal lodge	1
Brain (frontal lobe)	1
Total	54

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TABLE II

	Patient n°1	Patient n°2
Age	62	17
Sex	F	M
Cause	Odontogenic origin (Avulsion of molar)	Odontogenic origin Necrotic pulpitis of 37
Germes	Streptococco; Klebsiella; Stafilococco Aureus; Proteus; Pseudomonas Aeruginosa	Streptococco; Stafilococco; candida albicans; Serratia; Pseudo monas; Prevotella Oralis
Diffusion	Submandibular lodges; carotid and jugular lodge, anterior , posterior and superior mediastinum	Submandibular lodges, masticator space, parapharyngeal sapce, carotid and jugular lodge, anterior and posterior mediastinum
Surgical Treatment	Multiple cervical drainage, aggressive debridment, irrigation with hydrogen peroxide and iodinate antiseptics	Left transverse Cervicotomy and drainage
Farmacological Treatment	treatment: meropenem +piperacillin + metronidazole + amoxicillin + steroids then Diflucan	Imipenem + antifungal + steroids + daptomycin+ gastric protectors and amoxicillin levofloxacin
Course	Favorable hospital discharge in 14 days and clinical healing in 40 days.	Favorable hospital discharge in 12 days and clinical healing in 30 days.
Comorbidities	None	None

Material and Methods

In our department from 2003 to 2010 54 patients with a progression of odontogenic infection in spaces, cavities and parenchima two of them had a descending necrotizing cervical mediastinitis (Table Ib). The diagnosis was based on clinical manifestations of severe systemic and local efforts with dysphagia, odynophagia, dyspnea, pain, suffusion of soft tissue (in one of the patient), subcutaneous crepitus (gas). In both patients, the first young (aged 16) and the second older (aged 62) with no other systemic disease (no comorbidity) we had an odontogenic pathogenesis. The diagnosis was based primarily on instrumental OPT and CT scan.

The microbiological examination gave us, in both cases, positivity for Streptococcus Viridans, Prevotella oralis, Pseudomonas aeruginosa and Staphylococcus aureus (Table II).

Report of cases

PATIENT N. 1

The first patient, a woman of 62 years after one week from extraction of two molars 37 and 47 (Fig. 1), had developed a submandibular abscess, for this reason she was admitted to our department .

Immediately she proceeded with an instrumental examination (CT scan) (Figg. 2-3) that showed a fluid collection that was interested from the submandibular space,

with outflow along the left margin of the SCM, involving superior mediastinum (Figg. 4-5). The general condition worsened rapidly with hyperpyrexia, tachycardia, dyspnea, dysphagia, chest and neck pain, and for this reason she was subjected immediately to surgical treatment with multiple cervical drainage (aggressive debridement) and irrigation with hydrogen peroxide and iodine-based antiseptics (Fig. 6).



Fig. 1: Intraoral view of 47 and 37 divorced.



Fig. 2: Rx postoperative Opt (after dental extraction).

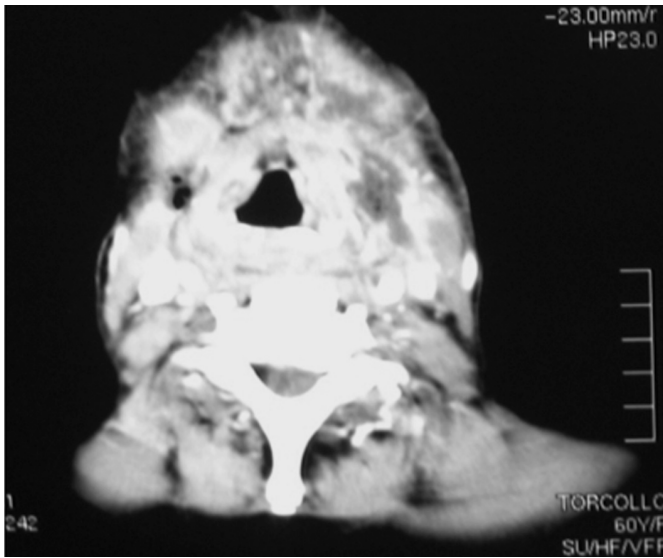


Fig. 3: The collection of fluid that develops below the parotid region; evident and minute gas collected in submandibular space.

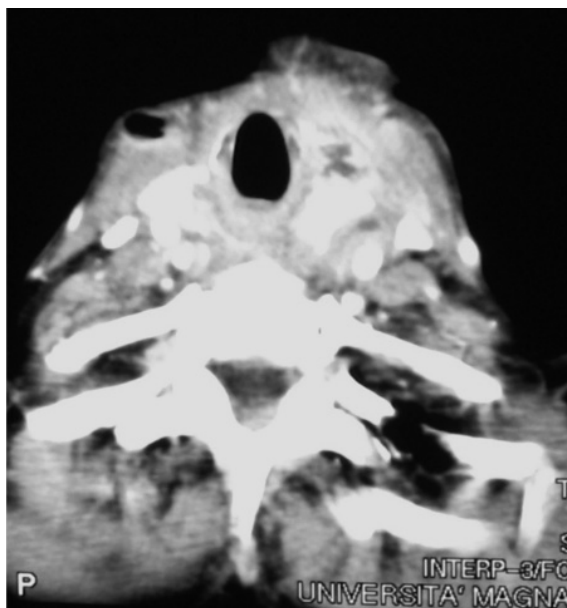


Fig. 4: The collection reaches the left mediastinum in proximity to the esophagus.

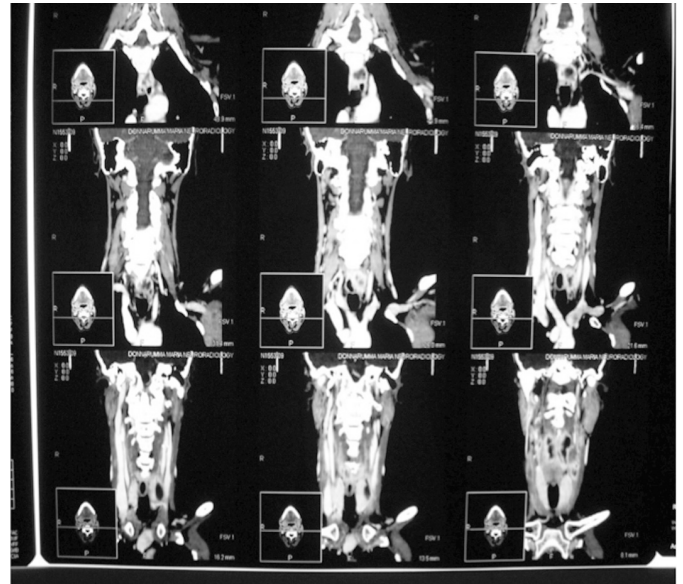


Fig. 5: Sagittal spinal TC cut where it is possible to detect the fluid collection that creeps back into the mediastinal lodge.



Fig. 6: Surgical drains.

Surgical treatment was associated with drug treatment with meropenem, piperacillin and metronidazole, and full-dose steroids. the patient's vital signs were monitored: blood count, liver and kidney function, blood cultures in febrile peaks, antibiogram, procalcitoninemy and PCR. Trachetomy was not practiced. The overall improvement was almost immediate, with the fall of leukocytosis and decrease of dysphagia, dyspnea and pain. After 12h she underwent again for a new thoracic and cervical CT, which showed a clear regression of cervical fluid collection and upper mediastinal disappearance net of the interest. On the sixth day the patient did not have fever with disappearance of dysphagia and a modest persistence of respiratory distress. Drug therapy was modified with the



Fig. 7: Satisfactory loco-regional conditions to the discharge.



Fig. 9: Perimandibular and laterocervical abscess.



Fig. 8: Clinical lateral view.



Fig. 10: OPT: apical granuloma of 37, 46, 47

addition of Diflucan (1 cp for day) and fluimucil (2g for 2 in 250 ml daily). In the fourteenth day was discharged in good general conditions (Figg. 7-8).

PATIENT N. 2

16 year old boy, was hospitalized for left perimandibular odontogenic abscesses (necrotic pulpitis with apical granuloma f 37) (Figg. 9-10), in precarious conditions: high fever, marked trismus and dysphagia. After a few hours from admission locoregional conditions tended to decline with the appearance of constrictive cervical pain, elevated leukocytosis (> 30 000 units of reference with a marked neutrophilia). The cervical and thoracic CT scan (Fig. 11) showed an abscess in the left submandibular space with outflow in carotid –jugular space.

The process reached the upper mediastinum. The patient underwent immediate surgery with cervical-tomic access (median and transverse). The lodge was opened and the submandibular carotid jugular space emptied of the abscess with a debridement of necrotic and colliquative tissue (Fig. 12). Was also extracted the dental element in necrotic pulpitis. Were not performed no further action like trachetomy of surgical drainage. After 24h a new CT was performed that showed a marked improvement of patient conditions with the disappearance of the abscess and process gases.

The clinical evaluation after the first 12 h of surgery were satisfactory, cervical thoracic pain disappeared, with improvement of mouth opening, reduced by the disappearance of dysphagia and dyspnea. In sharp decrease was leukocytosis, ESR and other inflammatory markers, sharp drop of temperature on first day and disappeared in the third.

The broad-spectrum antibiotic therapy with imipenem, high-dose daptomycin and antifungals associated with glucocorticoid therapy and gastric protectors was main-

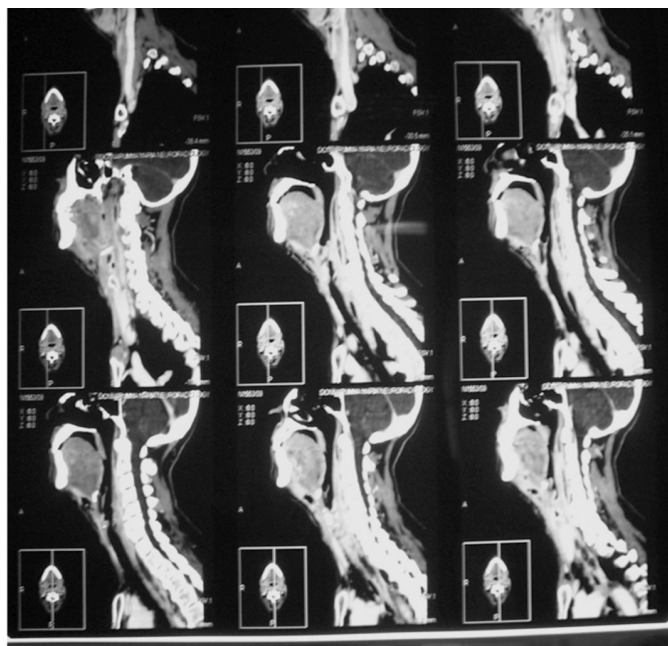


Fig. 11: Tc sagittal scans: abscess in the submandibular space with outflow of carotid jugular.



Fig. 12: Surgical drains.

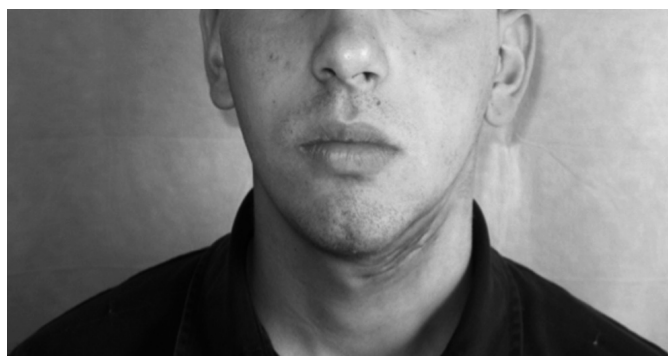


Fig. 13: Patient clinically cured at discharge.

tained until discharge occurred in the 12 th day of admission (Fig. 13) discharged with an additional antibiotic treatment for another 10 days with amoxicillin (1 g every 12h for os) and levofloxacin (500 mg every 24 hours).

There was a first clinical control after 15 days with a request of the parameters of inflammation.

The general and local conditions were more than satisfactory and the patient was discharged clinically cured.

Discussion and conclusions

The course of necrotizing fasciitis often has a rapid progression of an acute infectious process ³. Very often it can evolve so fast bringing the patient to death ⁴. From a meta-analysis Odontogenic causes affecting between 58,1% (Weatley et al.) and 11% of Own et al.⁵⁻⁶. These include the outbreak of departure are the focal processes of the last molars. The decline in mortality observed in recent years, though slight, is due to more targeted use of antibiotics and their greater power of action. The majority of DNM is supported by polymicrobial agents and between these anaerobes ,that having special affinity for the cellular lipid components, that quickly destroy muscle cells, erythrocytes and platelets, an increase of cascade of proteolytic enzymes as well as from the cellular lysis and also of bacterial cell.

The primary diagnostic procedure remains the ct scan (with variation of contrast) with a multidisciplinary approach in therapy between surgeon and infectiologist ⁷⁻⁸.

The treatment involves the use of IV broad spectrum antibiotics, maintenance of airway, removing the focus infection (if possible) with cervical surgical and \ or mediastinal drainage. In severe cases, a thoracotomy may be necessary when there is a breaching of the casting infectious in the tracheal bifurcation ¹.

Careful clinical observation, continuous monitoring of biochemical parameters and hepatic and renal function, are necessary to support the surgeon or surgical team (maxillofacial - thoracic surgeon), for possible tracheostomy and mediastinal, cervical thoracic drainage. It is also obvious that an adequate response to surgical drainage is provided by a follow-up with CT scan, as well as the parameterization of inflammatory markers ⁹⁻¹¹.

The massive antibiotic therapy intravenously, in the first instance given broad-spectrum, is then modulated and customize in relation to bacteriology variety and to the antibiogram. In the first case was administered meropenem and piperacillin in a full dose, metronidazole in the first instance and then diflucan and amoxicillin ;in the second case daptomycin, imipenem, metronidazole in the first instance and then amoxicillin with levofloxacin.

We do not use tracheotomy (Ridder). For the treatment of primary outbreak was done in the first instance, in the second case, the drainage of the teeth in endodon-

tic necrosis and only later extraction of molars, in the first case from the multiwall surgical revision. In both cases it was not required mediastinal drainage (mediastinotomy-trans-cervical) but only cervical (cervicotomy).

The location was very obvious to the upper mediastinal CT only in the first of the two cases.

In conclusion, necrotizing fasciitis, being aggressive and often a fatal disease must be diagnosed very quickly and yet by a team with medical and surgical treatment and a multidisciplinary approach.

Yet there is no clear clinical evidence on the effectiveness of hyperbaric oxygen therapy, used as a support for complex surgical medical therapy. Finally, much attention must be given to the possible presence of other diseases (comorbidities), such as metabolic, cardiovascular and immunological disease factors that give us negatively affect to the outcome of cervical necrotizing fasciitis (DNI) and mediastinal (DNM).

Riassunto

La fasciti necrotizzante cervico mediastiniche sono infezioni acute polimicrobiche dei connetivi a partenza più spesso dell'orofaringe e dal collo. Si tratta di affezioni gravissime con complicanze severe

Che spesso conducono a morte con un incidenza riportata in letteratura tra il 25% e il 40%. Da una approfondita metanalisi le cause odontogene incidono tra il 58% e l'11%. Tra queste il focolaio di partenza sono i processi focali degli ultimi molari inferiori. Il calo della mortalità osservata negli ultimi anni, anche se lieve, è dovuta all'uso più mirato degli antibiotici e alla loro maggiore potenza d'azione. La maggioranza delle DNM è sostenuta da agenti polimicrobici e tra questi gli anaerobi che avendo particolare affinità per le componenti lipidiche cellulari, distruggono più rapidamente le cellule muscolari, gli eritrociti e le piastrine, con un incremento a cascata degli enzimi proteolitici provenienti oltre che dalla lisi cellulare anche da quella batterica stessa.

L'indagine diagnostica primaria rimane la CT a variazione di contrasto e la terapia si avvale di un'approccio multidisciplinare. Il trattamento prevede l'uso in EV di antibiotici a largo spettro, il mantenimento della pervietà delle vie aeree, la rimozione del focolaio infettivo (se possibile) con drenaggio chirurgico cervicali e/o mediastinico. Nei casi più gravi può rendersi necessaria una toracotomia allorché vi è un superamento, da parte della colata infettiva, della biforcazione tracheale¹⁶⁻³⁰.

L'attenta osservazione clinica, il monitoraggio continuo dei parametri bioumorali e delle funzionalità epato-renali, sono di necessario supporto al chirurgo o al team dei

chirurghi (maxillo-facciale – otorino – chirurgo toracico), per eventuali ulteriori drenaggi cervico-mediastinici. E altresì ovvio che una adeguata risposta al drenaggio chirurgico è data da un follow up CT, oltre che dalla parametrizzazione dei markers infiammatori.

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