

Chronic Necrotizing Fasciitis / the Flesh Eating Bacterial Disease – An Unusual Sequel of Odontogenic Infections

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Abstract

Cervical necrotizing fasciitis is an unusual encounter in the general surgical practice, but is a life threatening condition requiring early recognition and adequate surgical treatment. Necrotizing fasciitis which holds deep fascia and subcutaneous fatty tissue including primarily surface fascia along with vein and nerve structure, is a fast progressing soft tissue infection of which mortality and morbidity is high. It is rarely seen in head and neck zone and usually develops after tooth and pharynx originated infections. This infection originating from a dental related source is rare. Early diagnosis, proper antibiotic therapy and surgical debridement form the factors affecting the success in cervical necrotizing fasciitis treatment.

Keywords: Cervical, Necrotizing fasciitis, Fournier Gangrene

Introduction

Cervical necrotizing fasciitis (CNF) is a rare infection of the fascial planes, which is less common in head

and neck, because of the rarity and higher vascularity in the region. It was first described in 1848. Wilson coined the term necrotizing fasciitis in 1952 and found no specific pathological bacteria related to the disease¹. Various terms are used for necrotizing fasciitis such as streptococcal gangrene, progressive synergistic bacterial gangrene, necrotizing erysipelas, suppurative fasciitis, acute dermal gangrene and fournier gangrene. It is characterized by fulminating, devastating and rapidly progressing condition. Most cases occur in the extremities, abdomen and perineum. It is a rare complication from dental infection that can lead to involvement of neck, mediastinum and chest wall. The reported cases usually are in people who are immunocompromised, but CNF also can occur in healthy people. CNF is polymicrobial. The bacteria involved are the same species as those that cause chronic dental infections or peri-apical infections of jaws. It has a classic manifestation of necrosis of subcutaneous fascia and may also cause necrosis of underlying muscle and overlying skin. The resulting pain is severe and out of proportion to the clinical findings. The early stages in CNF may resemble odontogenic cellulitis or an abscess. In early signs the skin usually is tonic and red. Hyperesthesia and anesthesia can be stated by touching. In the careful examination of the patient with full grown CNF, there is a rapidly growing panacula on which demarcation line can be observed². There occurs a tonic, purple skin zone encircling black necrotic zone in the middle and an expansive erythematic zone around it. Computed tomography may reveal gas bubbles in the tissues of neck, which are an early sign of CNF. Unless it is treated in early period, morbidity and mortality of CNF increases^{3,4}.

Materials and Methods

The present study was conducted on a patient presenting with infections of odontogenic origin in the outpatient Department of Oral and Maxillofacial Surgery (Figure 1)

A detailed history of the mode of onset and duration of infection was taken. General physical and detailed

extra-oral and intra-oral examination was carried out in the patient. Routine investigations of blood and complete urine examination was carried out. Bacteriological examination was carried out by collecting two pus samples aspiration from the abscess site with a disposable 16-gauge needle and syringe.

One of the collected samples was immediately transferred to pre-reduced thioglycollate broth prepared and sterilized in bijou bottle and then transported to clinical microbiology laboratory. The second sample was transported as such. The samples were processed for gram staining, bacterial culture and antimicrobial sensitivity.

Gram staining was carried out as under:

- A smear was prepared from the pus sample for gram staining on a clean grease free glass slide, air-dried and fixed in methanol.
- The smear was flooded with crystal violet for 1 minute.
- The slide was treated with gram's iodine for 1 minute.
- The slide was washed with acetone for few seconds and then with running tap water.
- The smear was counter stained with diluted carbol fuchsin for 30 seconds.
- Slide was washed with running tap water.
- The stained smear was dried and seen under oil immersion (100x) lens.

Bacterial culture and antimicrobial sensitivity was done as under:

A portion of the collected sample in the bijou bottle was incubated on two culture plates of Brucella Agar base with 5% sheep blood. One of the culture plates was incubated at 37°C in the incubator under aerobic environment. Second culture plate was incubated in an anaerobic jar (Himedia) in which anaerobic conditions were created using chemicals supplied by Himedia (LE002B). This was incubated at 37°C for 48 hours. The bacteria isolated were identified. Antimicrobial sensitivity was tested by modified Kirby bauer's disc diffusion method. The organisms isolated on Brucella blood agar in pure culture were inoculated in a suitable broth medium (Peptone water in case of aerobes and thioglycollate broth in case of anaerobes) and incubated at 37°C for 4 hours. The density of the organisms in broth was adjusted to 10⁷ cfu/ml by matching the turbidity of the broth with 0.5 Mcfarland opacity standard tube. The broth was inoculated on Mueller Hinton agar plate by spreading it with sterile cotton swabs. It was allowed to dry. Antibiotic discs were applied on it using sterile forceps. The plates were then incubated overnight at 37°C. The zones of complete growth inhibition were measured. The interpretation of zone size into

sensitive, intermediate or resistant size into sensitive, intermediate or resistant was based on Kirby Bauer disc diffusion interpretation based on Kirby Bauer disc diffusion interpretation chart.

The diagnosis was made on the basis of history of the patient, clinical examination and investigations. Definitive management consisted of either only extraction of offending tooth or incision and drainage of abscess followed by tooth extraction as indicated. Extraoral or intraoral incision and drainage was done under local anaesthesia depending on the space involved. Haemostat was used to break the loculi of pus. Corrugated rubber drain was used to keep the pathway of drainage patent wherever required.

Empiric antimicrobial therapy was started, which consisted of intravenous Amoxicillin 1g + Clavulanic acid 0.2g, 12 hourly for severe infections. For serious anaerobic bacterial infections intravenous Metronidazole 7.5 – 15 mg / Kg was infused depending on the severity of the individual infection. After culture and sensitivity report was available, culture and sensitivity directed antimicrobial therapy was instituted.

Supportive therapy in the form of parenteral fluid, high protein diet and multivitamin was given. Removal of the offending tooth/teeth was carried out when signs and symptoms of toxicity were absent and sufficient mouth opening was there to carry out the tooth extraction. Data obtained was analyzed to study the microbiology of odontogenic infections and to evaluate the combined role of antimicrobial therapy and surgical intervention for successful management of odontogenic infections.

Results

The bacterial culture antibiotic sensitivity tests, staphylococcus aureus was discovered and growth of bacteroids species and peptostreptococcus seen after 48 hours of incubation at 37 degree Celsius. The final diagnosis came out to be right submandibular space abscess with cervical necrotizing fasciitis. After antibiotic sensitivity and culture report patient was continued Injection Augmentin I/V 1.2 gm BD and Infusion Metrogyl 100ml I/V for 7 days.

Slowly the granulation tissue developed after a month and showed negative bacterial cultures (Figure 2). Skin grafting was planned, but due to some circumstances, patient did not go for skin grafting procedure. Early post-operative functional results were very good with no limitations in movements, while cosmetic aspects were acceptable.

Discussion

Case Report

A 45 years old male patient, was referred to the Department of Oral and Maxillofacial Surgery with the chief complaint of pain and swelling with foul smell and pus discharge along with fever and inability to swallow solids since a week. Patient was well oriented to time place and person. As a general examination, paleness, tachycardia, shivering, lethargy, diaphoresis and appearance of illness could be seen. No other abnormalities could be identified except high WBC i.e. $14.2 \times 10^3/\text{dL}$ and urinary function was normal. Patient complained of pain which was severe in character and radiated towards neck, chest, abdomen and shoulders. Swelling could be seen in right side of submandibular and neck region and right and left side of chest. Necrotic skin could also be seen along with redness and erythema. The wound appeared necrotic with a dark base and foul, aerated, frothy discharge. Deep dermis both in the neck and thorax presented vascular thrombosis and did not bleed.

On palpation there was localized rise in temperature along with tenderness. Fluctuation was present in submandibular region & in middle of chest accompanied by crepitus and induration of margins. On intraoral examination, we could find drainage of pus. It also revealed poor oral hygiene status, trismus with mouth opening 0.5mm and hypersensitivity to hot and cold with respect to 45 and 46. Patient had undergone radiological investigations such as orthopantomogram which revealed carious 14,17,28 & the involved culprit teeth as 45 & 46. Right and left lateral view of mandible was taken to view the culprit teeth. A posterior-anterior view of chest and ultrasonography was advised which had normal findings. When pus examination was done, there was presence of gram positive cocci and gram negative bacilli. Patient was empirically put on following intravenous distribution such as Injection Maxicef 1 gm 8 hourly, Dalacin- C 600mg 8 hourly, Injection Augmentin I/V 1.2 gm 12 hourly and infusion Metrogyl 100 ml 8 hourly. Emergency surgical debridement was indicated with a high suspicion of infection with gas producing bacteria.

Surgical strategy consisted in aggressive debridement of as much as possible of necrotic fascial structures and resection of all affected skin down to viable tissue, muscular tissue was preserved.

Under strict aseptic conditions, using local anesthesia, an incision was given in the submandibular region and another incision was given in the anterior chest wall which was a vertical incision. Blunt dissection was done with hemostat and pus was drained. 2 pus samples were collected

i.e. one from the submandibular region and another from the cuticular region. These were sent to the microbiology laboratory for antibiotic and culture sensitivity.

Thorough irrigation was done with hydrogen peroxide and betadine using 8 no. ryle's tube which was inserted into the anterior chest wall. This was done twice daily. After 12 days, two incisions were given- one was near the hyoid bone and another below the clavicle. Both were transverse incisions. Further irrigations were done. Soft gauges impregnated with betadine were placed in all spaces that have been developed and the whole wound was covered in betadine soaked dresses. After one and a half month, a clavicular incision was given (followed by vertical and transverse incisions) and healing was seen with respect to that.

General Discussion

Necrotizing fasciitis is an insidiously advancing soft tissue infection characterized by widespread fascial necrosis. Organisms spread from the subcutaneous tissue along the superficial and deep fascial planes, presumably facilitated by bacterial enzymes and toxins. This deep infection causes vascular occlusion, ischemia and tissue necrosis. Superficial nerves are damaged producing the characteristic localized anesthesia. Septicemia ensues with systemic toxicity. Cervical NF is usually seen in men⁵. In most cases aetiology is polymicrobial (type I necrotizing fasciitis) and this form may initially be mistaken for wound cellulitis more so when toxicity is not obvious⁶. Type II is a streptococcal infection also called "flesh eating infection" and Type III is a gas gangrene with myonecrosis. The case presented in this paper is a typical Type I infection with spread on fascial planes.

Dental pathology is a most common source of it and parapharyngeal and peritonsillar infections follow this. The cause of odontogenic originated infection is mostly molar teeth in mandible. These teeth apices lying under the zone where mylohyoid muscle sticks to mandible provide infections resulted from these teeth, to reach the submandibular zone. Subsequently infection can move to head basis above and thorax sinus mediastinum below⁷. The risk factors causing CNF are diabetes mellitus, malnutrition, obesity, peripheral vascular illness, severe liver illness and AIDS.

For investigations, culture sensitivity tests should be done to identify the causative organism. Use of frozen section biopsy of suspected necrotizing fasciitis is a valuable technique for rapid diagnosis of this disorder⁵.

The key in management is early recognition and early debridement. It is essential to remove all the

non-viable tissue including fascia. Repeated debridement is required to prevent progress of lesion to viable tissues. In surgical treatment, drainage and excision of all necrotic tissues are necessary. In odontogenic infections, antibiotic regimen should have a broad enough spectrum to include infection's possible causes in addition to rare pathogens⁸. A combination of penicillin, clindamycin and an aminoglycoside, or of penicillin, chloramphenicol and an amonoflycoside should be effective until culture reports indicate specific regime⁹. Topical sulfadiazine can be applied to clear off the bacteria. Non-surgical management of deep cervical fascial infection emerges as a novel alternative but requires a close supervision and drainage of all abscesses. Some investigators have reported that hyperbaric oxygen treatment has a favorable effect on diseased tissue⁹. As there is no experience and no clear cut indications for such a conservative treatment, we do not advocate against such management except for well documented cases with limited extension.

Conclusion

CNF is considered one of the potential complications of long standing odontogenic infections in patients with immune compromised status particularly in lower dentition. Rapid recognition and rapid intervention are keys to successful treatment. Mortality is directly proportional to the time to intervention¹⁰. The management of necrotizing fasciitis affecting cervical spaces continues to be a challenge and results depend a great deal on surgical skills and courage to insure an early aggressive debridement. Clinical examination, correct empiric antibiotic selection and appropriate surgical intervention are the cornerstones of proper management of deep cervical infections. CNF patients have to undergo a long rehabilitation period before they can return to an active, functional lifestyle.

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