ASCENDING NECROTISING FASCIITIS OF ODONTOGENIC ORIGIN: A CASE REPORT AND LITERATURE REVIEW

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ABSTRACT

Necrotising Fasciitis is a severe and sometimes fatal polymicrobial infection, which characteristically spreads along fascial planes to involve subcutaneous tissues, fascia, late stage skin, and possibly muscles. It is rare in head and neck region, with odontogenic infection being the commonest cause. Early diagnosis, prompt surgical intervention, aggressive antimicrobial therapy and intensive care are the key to successful management. A case of necrotising fasciitis with ascending infection into infratemporal and temporal spaces is reported.

INTRODUCTION

Necrotising Fasciitis (NF) is a rare life threatening condition characterized by rapid progressive necrosis of fascia, muscle, fat and a late stage skin with significant morbidity and potential mortality. Dental infections are the most common etiology for NF of face and neck followed by trauma, peritonsillar and pharyngeal abscesses, and osteoradionecrosis. Most of the NF cases of odontogenic origin reported in the literature are of descending infection into the neck. Four previous cases of ascending necrotising fasciitis of odontogenic origin have been reported in the literature. A case report of ascending necrotising fasciitis of odontogenic origin is reported.

CASE REPORT

A 34 years old male presented in the emergency department of King Fahad Hospital, Al-Medina Al-Munawwara, Saudi Arabia with 5 days history of progressively increasing right facial swelling. Previous medical history was insignificant. Patient had following vital signs: temperature 38.5°C, blood pressure 150/90, pulse 80/minute and respiratory rate 20/minute. Clinical examination showed massive right pan-facial swelling involving temporal, infraorbital, massesteric and buccal areas. Patient had limited mouth opening and fetor could be noted from the mouth. Intraorally there was swelling of parapharyngeal, ascending ramus and posterior buccal areas on the right side, with blue colored necrotic tissue in buccal and ascending ramus areas. Badly carious teeth # 17, 18 were present. Abnormal hematological values and vital signs at the time of admission and during the course of treatment are shown in table I. An urgent CT scan showed cellulitis of right infra temporal, massesteric, pterygomandibular, parapharyngeal and buccal spaces. Abscesses were present in right buccal, parapharyngeal and infra temporal spaces; with air bubbles present in pterygomandibular, temporal and infratemporal spaces (Figures 1 & 2). A diagnosis of ascending necrotising fasciitis secondary to carious teeth #17, 18 was made. Patient was admitted and empiric intravenous 600mg Clindamycin and 6 million unit crystalline Penicillin were commenced. Patient was operated within 6 hours of admission. Aggressive debridement of necrotic tissue in the buccal and ascending ramus areas was done until healthy bleeding tissue was encountered. Massesteric, pterygomandibular, submandibular, parapharyngeal, superficial temporal and deep temporal spaces...
TABLE 1: PATIENT'S LABORATORY AND CLINICAL MONITORING PARAMETERS
[ADMISSION (DAY 1) AND FOLLOW UP VALUES]

<table>
<thead>
<tr>
<th></th>
<th>Day 1</th>
<th>Day 4</th>
<th>Day 9</th>
<th>Day 13</th>
<th>Day 20</th>
</tr>
</thead>
<tbody>
<tr>
<td>White Cell Count (4-10 k/l/4l)</td>
<td>25.2</td>
<td>17.7</td>
<td>16.5</td>
<td>15.3</td>
<td>7.5</td>
</tr>
<tr>
<td>Platelets (150-300 k/l/4l)</td>
<td>240</td>
<td>360</td>
<td>411</td>
<td>717</td>
<td>656</td>
</tr>
<tr>
<td>C reactive protein (0.02-1.3 mg/dl)</td>
<td>25.6</td>
<td>12.8</td>
<td>3.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sodium (136-145 mmol/l)</td>
<td>135</td>
<td>133</td>
<td>130</td>
<td>132</td>
<td>136</td>
</tr>
<tr>
<td>Creatinie Kinase (21-232 U/l)</td>
<td>610</td>
<td>177</td>
<td>234</td>
<td>209</td>
<td></td>
</tr>
<tr>
<td>Albumin (35-50 g/l)</td>
<td>35</td>
<td>27</td>
<td>27</td>
<td>27</td>
<td></td>
</tr>
<tr>
<td>Temperature (°C)</td>
<td>38.5</td>
<td>37.8</td>
<td>37.8</td>
<td>37</td>
<td>37</td>
</tr>
</tbody>
</table>

were drained with minimal discharge through temporal, introroral and submandibular areas. Through and through corrugated drains were inserted in superficial temporal, deep temporal, pterygomandibular and parapharyngeal spaces. A corrugated drain was also applied in the masseteric area through submandibular wound. Teeth # 17 and 18 were extracted. Patient was transferred to intensive care unit and kept intubated for airway maintenance. Histopathological examination of the debrided tissue showed tissues composed of mucosa, sub-mucosa, minor salivary glands, fascia and

![Fig 1: CT scan showing right sided panfacial cellulitis, buccal space abscess (A), fasciitis (B), myositis (C), air in the pterygomandibular space (D), and compromised airway (E).](image1)

![Fig 2: CT scan showing air along the temporalis muscle (A) and air bubbles in the infratemporal space (B).](image2)

muscles; all showing acute and chronic inflammation with extensive necrosis consistent with necrotising fasciitis. Antibiotic therapy adjustments were made based on culture and antibiotic sensitivity results (Table 2). Thorough irrigation of 1% hydrogen peroxide and baneocin (Biochemie, Austria) antibiotic solution (Bacitricin Zinc 250IU/UI, Neomycin 5000IU/UI per 5mg. 10mg. diluted in 200m1. of normal saline) was started, and was continued twice daily. Patient's facial and parapharyngeal edema started to regress from day 3. Patient was extubated on day 4. Pus discharge started from temporal and submandibular drainage sites from day 6. Patient's progress was monitored clinically and by hematological examination (Table 1); as well as, by CT scans on day 4, day 9 and day 16. Patient was given high protein diet and human albumin transfusion for hypoalbuminemia. Mouth opening

![Fig 2: CT scan showing air along the temporalis muscle (A) and air bubbles in the infratemporal space (B).](image2)

TABLE 2: MICROBIOLOGICAL CULTURE RESULTS AND ANTIBIOTICS USE

<table>
<thead>
<tr>
<th>Culture</th>
<th>Antibiotics used</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gram-negative aerobe rods</td>
<td>Clindamycin</td>
</tr>
<tr>
<td>Enterobacter spp</td>
<td>Crystalline Penicillin</td>
</tr>
<tr>
<td>Citrobacter freundi</td>
<td>Gentamycin</td>
</tr>
<tr>
<td>E colli</td>
<td>Imopenim</td>
</tr>
<tr>
<td>Gram-positive aerobe cocci</td>
<td>Flagyl</td>
</tr>
<tr>
<td>Staphlococcus Aureus</td>
<td>Vancomycin</td>
</tr>
<tr>
<td></td>
<td>Augmentin</td>
</tr>
</tbody>
</table>
exercises were started on day 13. Patient was pre-
scribed Aspirin 75 mg daily for thrombocytosis. Pus
discharge started to reduce on day 13 and stopped by
day 20. Drains were shortened and removed in stages.
Patient was continued on IV antibiotics till day 30,
when he was discharged from the hospital on two
weeks of oral Augmentin. Review at 6 weeks showed
limited mouth opening of 2.5 cm with good secondary
healing of debrided area and no sign of infection.

**DISCUSSION**

This disease was initially described in 1871 AD by
Jones. However, the term Necrotising Fasciitis' was
first used by Wilson in 1952. Most frequently involved
sites of Necrotising Fasciitis (NF) are groin, abdomen
and extremities. Head and neck involvement is un-
common; and can be divided into two groups of scalp
and eyelids, and face and neck. Spankus noted approxi-
ately a quarter of 300 cases had cranial involve-
ment. Most acute bacterial infections of maxillofacial
region resolve with minimal complication following
appropriate management. Eleven cases of cervical
necrotising fasciitis were reported from 422 admitted
infection patients in a 9 year period with a fatal
outcome in 2 patients. Significant morbidity and
mortality have been reported following cervical NF of
odontogenic origin with subsequent mediastinal in-
volvement. It has been postulated that the factors that
determine whether an odontogenic infection develops
into an acute soft tissue infection or into a necrotising
type of infection are related to either the host
immunological state and/or the synergistic effect of
associated infecting organisms. Group A beta
hemolytic streptococci and staphylococcus aureus,
alone or in synergism, are the initiating infecting
organisms in NF. Other aerobic and anaerobic
pathogens may be present, including bacteriodes,
clostridium, peptostreptococcus, enterobacteria,
conforms, proteus, pseudomonas, and klebsiela.

Systemic conditions such as uncontrolled diabetes
mellitus, renal disease, cardiovascular disease,
peripheral vascular disease, liver cirrhosis, obesity and
immune compromised status by either medical therapy
or HIV predispose individuals to NF at a much higher
rate than healthy persons.

Diagnosis of NF is frequently delayed, which can
result in significant morbidity and mortality. Rapid
spread of infection, cellulitis with disproportionate
pain, the presence of subcutaneous crepitations,
hyperaesthetic or anaesthetic skin and systemic toxic
manifestations may indicate possible NF. A fetor may
be present signaling presence of dead tissue.
Admission white blood cell counts of greater than 15x10⁹/⁠l
and serum Na of less than 135mmol/l are useful param-
eters that may help distinguish NF from non-NF
cellulitis when classic signs are not present.
Admission values of serum C-reactive protein and creatinine
kinase have been reported higher for gas NF patients
than patients admitted for cellulitis. CT scan helps in
early diagnosis and identifying extent of involved tis-
sues. Diffuse thickening and infiltration of cutis and
subcutis (cellulitis); diffuse enhancement and/or thick-
ening of the superficial and deep cervical fascia
(fasciitis); enhancement and thickening of the platysma,
sternoleidomastoid, strap muscles and other involved
muscles (myositis); and fluid collections in multiple
neck compartments have been reported as constant CT
features of cervical NF. Inconstant CT features
included gas collection, mediastinitis, and pleural or
pericardial effusions. It is easy to digitally explore
between skin and superficial deep fascial layer after a
small incision. Presence of foul smelling, watery "Dis-
h water" like pus along with necrotic subcutaneous and
fascial tissues also helps to confirm the diagnosis,
however, pus may not always be present.

Biopsy is essential for the diagnosis. Immediate diagnosis of NF
in this reported case was made based on the clinical
features of high grade fever, acute cellulitis, fetor,
intraroal necrotic tissue and oedema; laboratory features
of high white blood cell count (25.2 k/ml), high serum
creatinine kinase (610 u/l) and low serum sodium levels
(135mmol/ l); CT features of cellulitis, fasciitis,
myositis, abscess and air bubbles in the fascial spaces;
and was confirmed by histopathology.

Early diagnosis, aggressive surgical intervention
with drainage of pus and debridement of all necrotic
tissue, management of the underlying source of infec-
tion and predisposing condition and supportive therapy
(antimicrobials, rehydration, nutrition and rest) are the
basis of successful treatment of NF. Involved
carcinotic fascia is often more than the overlying involved
skin. Empiric broad spectrum antibiotic therapy
should be initiated because of polymicrobial nature of
the disease. Combination therapy of penicillin with
metronidazole or clindamycin; third generation cepha-
losporin with metronidazole or clindamycin; or a single
antibiotic coverage with broad spectrum beta-lactam drugs such as impenim would cover the microbial spectrum of NF. Antibiotic therapy should be continued on the basis of microbiological culture and antibiotic sensitivity test results. Airway should be assessed and secured with either endotracheal intubation or tracheostomy. Daily wound irrigation, inspection and repeated debridement of necrotic tissue should be done. More than one debridement and/or drainage may be needed. Wound management should include thorough twice daily irrigation with saline and 1% hydrogen peroxide. Antibiotic (Bacitracin) irrigation has also been recommended. In the present case aggressive debridement of the necrotic tissue was carried out. CT scan showed air bubbles in the deep temporal space with abscess cavities. Some authors recommend more aggressive debridement in the deeper planes, which would have caused greater morbidity. Systemic toxicity; local wound examination; serial laboratory examination of serum white cell count, sodium, C reactive protein, and creatinine kinase (Table I); and repeated CT scans were used to monitor patient’s progress. CT reassessment may show recurring deep fascial abscesses. When indicated, early surgical intervention should be done. All the laboratory parameters progressively improved (Table I). Serial CT scans also showed progressive control of the disease. There is a tendency for hyponatremia, hypoproteinemia and hypoalbuminemia in NF patients. Good nutritional support with high protein diet and monitoring is required. Use of hyperbaric oxygen has also been advocated to improve the morbidity and mortality.

The morbidity and mortality rate of NF are still high, which are directly related to the time lapse before the start of the appropriate treatment. The major complications of NF are mediastinitis, pericardial effusion, airway obstruction, rupture of major vessels, brain abscess, disseminated intravascular coagulation, sepsis, acute renal failure and respiratory failure. Death resulting from NF is most commonly attributed to respiratory failure, sepsis and multiple organ failure. In this case early recognition of NF, surgical intervention within 6 hours of admission, appropriate aggressive antibiotic therapy based on culture and sensitivity results, and thorough aggressive local antibiotic/antiseptic irrigation contributed to minimal morbidity only. The only complication that has occurred in our case was limitation of mouth opening, which may require coronoidectomy in future.

REFERENCES