Necrotizing Fasciitis initially Managed as Cellulitis: A Report of Two Cases

***Ifueko P. OSAGHAE, **Osamwonyi C. ERIAMIATOR**

*Department of *Oral/Maxillofacial Surgery, **Department of Family Dentistry, Dental Center of Central Hospital Benin, Edo State.*

Correspondence
Dr I.P Osaghae
Department of Oral/Maxillofacial Surgery
Dental Center of Central Hospital, Benin, Edo State.
Email: Ifueko02@yahoo.com; ifuekoosaghae@gmail.com

ABSTRACT

Objective: To report two cases of cervicofacial necrotizing fasciitis which were initially managed as rapidly spreading cellulitis.

Case Description: These two cases of cervicofacial necrotizing fasciitis presented in our clinic with features of rapidly spreading cellulitis of odontogenic origin. Incision and drainage was done and patients presented after 48 hours with necrotic tissue. Diagnosis of cervicofacial necrotizing cellulitis was made and aggressive debridement of necrotic tissue was carried out.

Conclusion: Diagnosis of necrotizing fasciitis can be difficult when one is unfamiliar with the condition which can cause a delay in treatment. This may be compounded by the presenting symptoms which are nonspecific and may mimic cellulitis. An index of suspicion must be retained in all cases of cellulitis.

Keywords: *Cellulitis, Necrotizing fasciitis, Presentation, Treatment.*

INTRODUCTION

Diagnosis of necrotizing fasciitis (NF) can be difficult when one is unfamiliar with the condition thereby delaying treatment. This may be compounded by the relative lack of clinical signs and symptoms during the early course of the infection (Sahdy, 2005). NF is an infection that spreads along the fascial planes causing subcutaneous tissue death with relative sparing of skin and underlying muscles, characterized by rapid progression, systemic toxicity, and even death (Green et al., 1996). Presenting symptoms of NF are nonspecific and may include mild cellulitis, edema, and occasionally crepitation (Green et al., 1996). This condition is regarded as a surgical emergency due to associated high rate of morbidity and mortality (Tung-Yiu et al., 2000; Obiechina et al., 2001; Tiu et al., 2005; Subhashraj et al., 2008).

Cervicofacial NF (CNF) is rarely-suspected in the early phase because the disease is uncommon, the presenting signs and symptoms are often benign, and there is frequently a dental infection or pharyngitis thought to explain the findings (Lee et al., 2010).

Citation: Osaghae IP, Eriamiator OC (2020). Necrotizing fasciitis initially managed as cellulitis: A report of two cases. Nig J Res Orofac Infect Oncol; 3(1):24-29.

At onset, NF can be difficult to differentiate from cellulitis and other superficial infections of the skin (Puvanendran et al., 2009). Studies have shown that only 15% to 34% of patients with NF have an accurate admitting diagnosis (Hefny et al., 2007). Patients are sometimes treated for simple cellulitis until they rapidly deteriorate (Choi, 2015). Misdiagnosis and delayed treatment can result in death from sepsis, mediastinitis, carotid artery erosion, jugular vein thrombophlebitis, or aspiration pneumonia (Lee et al., 2010).

Although mortality from the disease has been reported to have improved after the advent of antibiotics, it remains a common affliction in the developing and underdeveloped nations due to poverty and lack of awareness (Whitesides et al., 2000; Ndukwe et al., 2002). It is generally accepted that predisposing factors to NF include diabetes, alcoholism, tobacco abuse, immunosuppression, malnutrition, advanced age, peripheral vascular disease, renal failure, underlying malignancy, and obesity (Quereshy et al., 2009).

Precipitating events of NF include surgery, minor invasive procedures (e.g., joint aspiration and acupuncture), intravenous drug use, penetrating injuries (e.g., insect and animal bites), soft tissue infection, burns, and child birth (Puvanendran et al., 2009). Investigations include Computed Tomography (CT) which is the best way to detect the boundary and severity of NF. In contrast to abscess, NF shows an area of fluid accumulation without significant rim enhancement at the periphery (Tung-Yiu et al., 2000). The objective of present study was to describe two cases of cervicofacial necrotizing fasciitis which were initially managed as rapidly spreading cellulitis.

CASE DESCRIPTION

Case 1

A 85-year-old female patient presented to our clinic due to persistent toothache and swelling on the lower left jaw (L) of one week duration. She gave a two year history of toothache before presentation which has been intermittent due to frequent intake of Ampiclox and unidentified pain killers procured from a Patent Medicine shop in her village. Six months prior to presentation, she noticed a slight swelling on her left lower jaw and was given in addition to the previous medication, 5mg of prednisolone (she came with the sachet) which she took daily for 6 months until a week before presentation that swelling started increasing rapidly and toothache became persistent. No other medical condition was significant except the steroid use.

On examination, there was diffuse swelling of the submental and submandibular (L) areas which was tense and warm to touch. Intraorally, mouth opening was limited to about 1.8cm interincisal distance. Swelling of the floor of the mouth and raised tongue were observed. The teeth could not be examined. A diagnosis of cellulitis of odontogenic origin was made and admission process was started. The patient's initial vital signs were: blood pressure (BP) 110/70 mmHg, pulse rate (PR) 80 beats per minute, respiratory rate (RR) 20 cycles per minute, and temperature (T) 36.4°C. She was placed on intravenous infusion of 0.9% normal saline, blood was taken for further investigation and intravenous ceftriaxone 1.0gm; metronidazole 500mg and intramuscular paracetamol 600mg were administered. We promptly drained the abscess by incising the submental and submandibular (L) areas. About 10mls of pus was expressed. Drains were inserted and a dressing placed over the submandibular (L) and submental areas and the patient was sent to the ward to continue her medication, the ceftriaxone was given 12 hourly, metronidazole and paracetamol, 8 hourly.

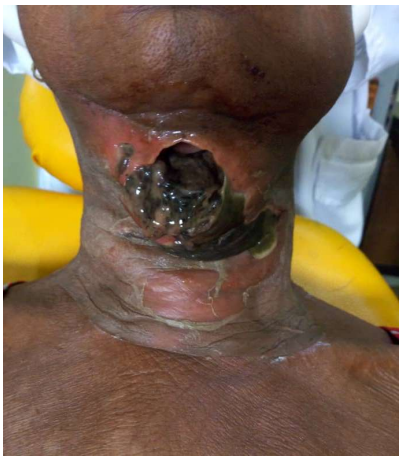

Figure 1a: 48 hours after I&D

Figure 1b: after debridement of non-viable tissue

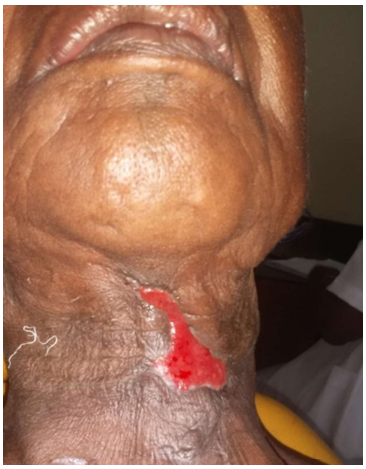

Figure 1c: Satisfactory granulation at 6 weeks

The next day, the results of her laboratory investigation revealed a Fasting blood sugar (FBS) of 105mg/dl (range in our environment: 80-120mg/dl), haematocrit (Hb): 9.7g/dl, Packed Cell Volume (PCV): 31%, (total white blood cell count) WBC: 10,5000mm^3 (differentials: Neutrophils- 80%; Lymphocytes- 12%; and others- 8%). Retroviral screening was non-reactive. Her mouth opening was observed to have improved and examination revealed deeply carious 36 and very mobile 42 which were extracted under local anaesthesia. Dressing was changed and patient was sent back to the ward to continue management, the 0.9% normal saline alternating with 4.3% dextrose saline.

When patient was seen 48 hours after initial presentation and dressing was removed, a dusky red necrotic tissue was observed on her anterior neck under the chin (Figure 1a). An immediate diagnosis of cervicofacial necrotizing fasciitis was made. Aggressive surgical debridement was carried out and all non-viable tissues were excised (Figure 1b). This was repeated twice within 24 to 48hours with dressings placed after debridement. Patient insisted on discharge after a week and she was sent home with tablets cefuroxime 500mg 12 hourly and metronidazole 400mg 8 hourly for five days. Dressing was changed every two days till patient insisted on going back to the village. A satisfactory granulation tissue base (Figure 1c) was achieved at 6 weeks.

Case 2

A 60-year-old male patient was brought to the clinic by his neighbours with a two-week history of toothache and a week history of facial swelling. Patient gave a past history of intermittent toothache of about six years and he had massaged his face with warm moist compress intermittently for that duration. His past medical history revealed that he had suffered a cardiovascular disease for six years which resulted in the loss of mobility of his two legs and weak but useful hands. He lost his job and was abandoned by his spouse and children.

On examination, left and right submandibular plus submental diffuse, tense and tender swelling that were warm to touch with raised floor of the mouth was observed. Severely limited mouth opening prevented an intraoral examination.

A clinical diagnosis of Ludwig's angina was made and admission procedures were started. Patient was placed on intravenous fluids and blood was taken for laboratory examination. Intravenous ceftriaxone 1.0gm, metronidazole 500mg, gentamicin 80mg and intramuscular paracetamol 600mg were given, the incision and drainage of abscess was achieved through the left and right submandibular spaces plus the submental space. Rubber drains were inserted and dressing placed. Patient was sent to the ward to continue medication with twice daily facial dressing.

Laboratory investigation showed that his hematocrit was 9.6g/dl; Packed Cell Volume 27%; White Blood Cell Count 13,800mm^3 (differentials- Neutrophils: 87%, Lymphocytes: 9%, Others: 4%). To Hepatitis B antigen, he was negative, and was non-reactive to Retro Viral Screening. Fasting blood sugar was 65mg/dl (range: 80-120mg/dl). The 46 was observed to be grossly carious and extracted the next day as mouth opening had improved.

Figure 2a: 48 hours after I&D

Figure 2b: Granulation at 3 weeks (last visit)

On the third day of presentation, when dressing was removed, patient was observed to have necrotic bluish tissue extending from right to left submandibular areas and in front of the neck (Figure 2a). A diagnosis of Necrotizing Fasciitis was made and immediate surgical debridement was carried out with removal of necrotic tissues. A dressing was placed and patient was sent to the ward. Debridement was repeated for two days. Patient insisted on leaving as he could not afford to stay in the ward. He was discharged against medical advice. He came about a week after discharge as there was no one to bring him before then. A neighbour had been dressing the wound. He came for the last time, 2 weeks after the last visit (Figure 2b).

DISCUSSION

Early recognition of NF may be difficult, and the clinical differentiation of NF and cellulitis is also particularly difficult (Rouse et al., 1982; Barker et al., 1987; Rahmouni et al., 1994; Bisno & Stevens, 1996). CNF is rarely suspected in the early phase because the disease is uncommon, the presenting signs and symptoms are often benign, and there is frequently a dental infection or pharyngitis thought to explain the findings (Lee et al., 2010).

CNF typically mimics odontogenic infection at the early stage and the mode of presentation can be misleading. However, the presence of an unusual erythema on a dark skin with accompanying vesicle formation is a pointer to the diagnosis of CNF. Subcutaneous crepitations usually precede gangrene and sloughing of the fascia. Other features include rapid spread to the neck areas, severe pain and radiographic finding of subcutaneous gas (Obimakinde et al., 2012).

The two reported cases presented with the typical signs of a rapidly spreading cellulitis and no signs of vesicle formation which lead to our limiting the treatment to just incision and drainage with antibiotics cover.

Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are useful in cases where signs are equivocal or the diagnosis is in doubt (Rahmouni et al., 1994; Wykosi et al., 1997). Both radiological investigations have their roles in delineating the extent of the infection and showing soft tissue gas, but the critical condition of patients often precludes their use and may even delay treatment (Sahdy et al., 2005). CT and MRI facilities are not domiciled within our clinic and these two cases were critical, therefore sending these patients out for CT (which is more readily available than MRI) would have probably led to a fatality.

Certain diagnostic signs that have come to be associated with necrotizing fasciitis have been discovered to not always present. The findings of crepitus and soft tissue air on plain radiograph have been seen in only 37% to 57% of patients, respectively (Elliot et al., 1996).

Systemic toxicity is one of the distinguishing features of necrotizing fasciitis (Schutz et al., 2012). The White Blood Cell Count of case 2 was much higher than the accepted normal in our environment. This is not an unusual finding as cases of cellulitis present with such laboratory findings.

The diagnosis therefore remains a clinical one, and the information drawn from all the investigations should be used in conjunction (Sahdy et al., 2005).

The submandibular space has been recognized as the most commonly involved site. This may be due to the proximity of the apices of the mandibular molar teeth to the submandibular space which favours a direct spread of infection to this region from the offending teeth. The infection typically spreads downward to involve other areas in the neck (Obimakinde et al., 2012). The two patients in this report had involvement of their submandibular spaces in addition to the submental space in the initial presentation of cellulitis with the extension to the anterior neck in their late presentation as necrotizing fasciitis. In a case reported by Choi, the patient was initially diagnosed with odontogenic infection, but necrotic fascia confirmed fasciitis (Choi, 2015).

Most cases of necrotizing fasciitis are known to be polymicrobial and empirical broad-spectrum antibiotics administration is the recommended standard (Sahdy et al., 2005). Oral streptococcal species have been cultured from most NF but staphylococcal and anaerobic organisms are occasionally identified. NF appears to be caused by the same species of bacteria that cause chronic dental subapical infections, acute localized cellulitis, and tissue abscess (Quereshy et al., 2009). Although subsequent antibiotic management will be guided by sensitivities of culture sample taken intraoperatively, initial treatment should provide effective cover against Gram-positive cocci, facultative anaerobic Gram-negative rods, and anaerobes. A combination of penicillin, gentamicin, plus metronidazole was used in our clinic with good results and this is supported by other workers (Sahdy et al., 2005). However, gentamicin was not administered in the first case in this report because of her age and abuse of steroids. The integrity of her kidneys could not be

trusted (Sahdy et al., 2005). The 6 months use of steroids to control facial edema could have caused an immunocompromised state added to her advanced age of 85 years.

The precipitating factors of NF include immunosuppression, malnutrition, advanced age, peripheral vascular disease, renal failure, underlying malignancy, and obesity (Quereshy et al., 2009). The precipitating factor in the first case may be age and steroid therapy. The second case in this report had suffered from a cardiovascular disease which may be precipitating factor and not receiving proper care may also be a contributory factor.

Necrotizing fasciitis is regarded as a surgical emergency due to associated high rate of morbidity and mortality (Tiu et al., 2005; Tung-Yiu et al., 2000; Subhashraj et al., 2008; Obiechina et al., 2001). The initial goals of surgical debridement are to eradicate all necrotic tissue and drain loculated collections and fluid in the fascial planes (Choi, 2015). Surgical debridement can be done at the bedside (Sahdy et al., 2005). In the two cases presented, serial debridements were done at the bedside with successful results. Immediate reconstruction is not recommended after recovery from the infection. Such procedures can be considered once a stable wound edge is observed. Primary closure can be performed with various local flaps, skin graft, regional flaps and even free vascularized flaps (Richardson & Schmitz, 1997). No reconstruction was done in the two cases reported here.

CONCLUSION

Diagnosis of necrotizing fasciitis can be difficult when one is unfamiliar with the condition which can cause a delay in treatment. This may be compounded by the presenting symptoms which are nonspecific and may mimic cellulitis. An index of suspicion must be retained in all cases of cellulitis.

Financial support and sponsorship

This work received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Conflict of interest

The authors declare that they have no conflicts of interest.

REFERENCES

Barker FG, Leppard BJ, Seal DV (1987). Streptococcal necrotizing fasciitis: comparison between histological and clinical features. J Clin Pathol; 40:335-341.

Bisno AL, Stevens DL (1996). Streptococcal infection of skin and soft tissues. N Engl J Med; 334:240-245.

Choi Moon-Gi (2015). Necrotizing fasciitis of the head and neck: a case report. J Korean Assoc Oral Maxillofac Surg; 41(2):90-96.

Elliott DC, Kufera JA, Myers RAM (1996). Necrotizing soft tissue infections: Risk factors for mortality and strategies for management. Ann Surg; 224:672-683.

Green RJ, Dafoe DC, Raffin TA (1996). Necrotizing fasciitis. Chest; 110:219-229.

Hefny AF, Eid HO, Al-Hussona M, Idris KM, Abu-Zidan FM (2007). Necrotizing fasciitis: a challenging diagnosis. Eur J Emerg Med; 14:50-52.

Lee JW. Immerman SB, Morris LG (2010). Techniques for early diagnosis and management of cervicofacial necrotising fasciitis. J Laryngol Otol; 124:759-764.

Ndukwe KC, Fatusi OA, Ugboko VI (2002). Craniocervical Necrotizing fasciitis in Ile Ife, Nigeria. Br J Oral Maxillofac Surg; 40:64-67.

Obiechina AE, Arotiba JT, Fasola AO (2001). Necrotizing fasciitis of odontogenic Origin in Ibadan Nigeria. Br J Oral Maxillofac Surg; 39:122-126.

Obimakinde OS, Okoje VN, Akinmoladun VI, Fasola AO, Arotiba JT (2012). Retrospective evaluation of necrotizing fasciitis in university college hospital, Ibadan. Niger J Clin Pract; 15:344-348

Puvanendran R, Huey JC, Pasupathy S (2009). Necrotizing fasciitis. Can Fam Physician; 55:981–987.

Quereshy FA, Baskin J, Barbu AM, Zechel MA (2009). Report of a case of cervicothoracic necrotizing fasciitis along with a current review of reported cases. J Oral Maxillofac Surg; 67:419-423

Rahmouni A, Chosidow O, Mathieu D, Gueorguieva E, Jazaerli N, Radier C, et al (1994). MR imaging in acute infectious cellulitis. Radiology; 192:493-496

Richardson D, Schmitz JP (1997). Chronic relapsing cervicofacial necrotizing fasciitis: case report. J Oral Maxillofac Surg; 55:403-408.

Rouse TM, Malangorin MA, Schulte WJ (1982). Necrotizing fasciitis: a preventable disaster. Surgery; 92:765-770.

Sahdy H, Matteucci P, Stanley PRW, Hart NB (2005). Necrotizing Fasciitis. BMJ; 330 (7495):830-833

Schütz P, Joshi RM, Ibrahim HHH (2012). Odontogenic necrotizing fasciitis of the neck and upper chest wall. J Oral Maxillofac Surg Med Pathol; 24:32-35.

Subhashraj K, Jayakumar N, Ravindran C (2008). Cervical necrotizing fasciitis: An unusual sequel of odontogenic infection. Med Oral Patol Oral Cir Bucal; 13:E788-91.

Tiu A, Martin R, Vanniasingham P, MacComick AD, Hill AG (2005). Necrotizing Fasciitis: Analysis of 48 cases in South Auckland, New Zealand. ANZ J Surg; 75:32-34.

Tung-Yiu W, Jehn-Shyun H, Ching-Hung C, Hung-An C (2000). Cervical necrotizing fasciitis of odontogenic origin: A report of 11 cases. J Oral Maxillofac Surg; 58:1347-1352.

Whitesides L, Cotto-Cumba C, Myers RA (2000). Cervical Necrotizing Fasciitis of odontogenic origin: A case report and review of 12 cases. J Oral Maxillofac Surg; 58:144-151; discussion 152.

Wykosi MG, Santora TA, Shah RM, Friedman AC (1997). Necrotising fasciitis: CT characteristics. Radiology; 203: 859-863.