NECROTISING FASCIITIS – A RARE CAUSE OF NECK SWELLING IN A 2 YEAR OLD CHILD

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ABSTRACT: INTRODUCTION: Necrotising fasciitis is an unusual infection in the head and neck region characterized by rapid spread resulting in extensive subcutaneous necrosis and skin gangrene. Most patients who develop necrotising fasciitis have pre-existing conditions that render them susceptible to infection. Conditions that result in immunosuppression, such as advanced age, chronic renal failure, peripheral vascular disease, diabetes mellitus, and drug misuse seem to be risk factors. Prompt diagnosis and treatment is required for reducing the morbidity and fatality. **CASE REPORT:** We report an unusual case of this condition in a 2 year old girl, who presented with a diffuse swelling on the left side of the neck extending from the clavicle to reach mandible of 2 day duration. Here we discuss the treatment policy adopted and the outcome. **DISCUSSION:** Necrotising fasciitis is an uncommon but potentially fatal condition and can affect any part of the body. Necrotising fasciitis is very rarely seen in paediatric age group. A case of necrotizing fasciitis in a 2 year old is presented for its rarity and for its diagnostic dilemmas.
CONCLUSION: Necrotising fasciitis is a rare but life threatening condition that requires immediate action, but uncertainties still hamper prompt diagnosis and treatment
KEY WORDS: Necrotising fasciitis, Head and Neck, Paediatric

INTRODUCTION: Necrotising Fasciitis is a potentially fatal rapidly spreading soft tissue infection of polymicrobial origin. The condition frequently affects thorax, abdominal wall, extremities, perineum and groin. Its occurrence in head and neck is uncommon, majority of cases limited to involvement of neck, usually from infection of dental or pharyngeal origin. Successful management requires early diagnosis, prompt institution of broad spectrum antibiotics, aggressive surgical debridement to control infection. Here we describe an unusual case of necrotizing fasciitis in a 2 year old girl treated and cured successfully.

CASE REPORT: A 2 year old girl presented to the outpatient department with complaints of fever, swelling in neck and puffiness of face of 2 days duration .There was no relevant previous medical history. On examination, the child was febrile and less active. She was systemically normal. Oral cavity was normal and no dental caries was seen. A diffuse swelling was noted on the left side of the neck which started from just above the clavicle to reach above mandible. There was no local rise of temperature. The swelling was soft, pitting on pressure. The skin was erythematous. There was no subcutaneous emphysema palpable. There was no cervical lymphadenopathy.

Patient was admitted and evaluated. Lab investigations revealed hemoglobin of 8 gm%, total leucocyte count of 17500/cubic mm with a neutrophil count of 77%. ESR was 12. LFT was within normal limits. There was no hypoalbuminemia. An X-ray neck soft tissue lateral view showed oedematous soft tissue of neck with no compromise on the airway. USG Neck showed features of soft tissue edema with no obvious organized collection. Multiple aspirations were done from the neck swelling which gave minimal serous fluid and no frank pus. The aspirate was sent for culture and sensitivity. Child was given supportive measures and started on IV Antibiotics, Inj.Cefoperazone and Inj.Amikacin. Reports revealed grams stain showing polymorphonuclear leucocytes and gram positive cocci. Culture identified Staphylococcus aureus and sensitivity was studied. Blood culture revealed no growth. An MRI study was done on the 3 rd day of admission which showed subcutaneous spreading inflammatory lesion, soft tissue edema in submandibular region, left sublingual plane and left side of face. Antibiotics were continued. On day 5, the diffuse swelling started getting organized in the left submandibular region. On day 6, pus was seen to be localizing and fluctuation was elicited. The decision to do an incision and drainage was made. On incision, frank pus was pouring out; tissue necrosis was evident, reaching up to the periosteum of mandible. However, mandible was seen not to be involved. The collection was drained out entirely, necrosis extensively debrided and wound left with a drain. Drain was removed in 3 days. Daily dressings and local debridement was done for the wound. Complete healing of wound took place in 45 days. The child was discharged in good condition later.

DISCUSSION: One of the first reports of necrotizing infection was published at time of American civil war by Joseph Jones(1871) [1].The skin of affected parts were stated to be 'melting away'. Later cases with widespread superficial fascia necrosis with undermining of surrounding soft tissue were reported. Meleney called the entity 'streptococcal gangrene' as he found B hemolytic streptococcus to be the causative organism[2]. The term necrotizing fasciitis was first used by Wilson in 1952 because necrosis of fascia was the most characteristic finding[3]. Anaerobic organisms were studied to play an important role in the non clostridial gas producing infection[4]. It is known to occur most frequently in extremities, genitalia, chest wall[5]. When it occurs in head and neck region, it poses a real threat to patient's life.

Patients are febrile and toxic and they present with rapidly progressing non fluctuant swelling of face, neck and chest. The subcutaneous tissue and superficial fascia may undergo purplish discoloration, starts sloughing and turn gangrenous and necroses. The skin is involved late in the course of infection when it becomes erythematous and edematous[7]. There is marked pain over swelling while skin is anaesthetic. Infection can spread to deeper layers of cervical fascia and can involve carotid sheath, cervical viscera, deep neck musculature, extending to mediastinum and anterior thoracic wall[6]. Bacteria release enzymes like collagenases and hyaluronidase which causes

spread of the focal infection. There is edema initially in the dermis which rapidly progresses to necrosis in epidermis, dermis and subcutaneous fat with dense polymorphonuclear infiltration. Blood vessels in subcutaneous plane undergo thrombosis and collagen gets fragmented[9]. The total leucocyte count is typically high with shift to left. Other investigations which can occur are anemia, hyperbilirubinemia, hypoalbuminemia, hyperglycemia and rarely hypocalcemia due to liquefactive necrosis of fat[8]. Imaging studies are not very helpful unless there is soft tissue gas is present. CT/MRI is better than plain films to pick up the findings[9]. It can progress to overwhelming sepsis and shock. Frank pus, lymphangitis and lymphadenopathy may occur. Histologically, intense acute inflammation with coagulative and/or liquefactive necrosis is seen. Granulocytes and plasma cells predominate. Muscle necrosis and vasculitis can occur with the thrombosis of small veins and arteries.

The aetiology of necrotising fasciitis is not fully understood, and in many cases no identifiable cause can be found. Causes of necrotizing fasciitis studied are dental infection, blunt trauma, scratch, bite, post surgery, tonsillar abscess, pharyngitis, irradiation with osteoradionecrosis, gunshot injury and are sometimes of unknown etiology[5]. Predisposing factors identified are immunosuppressed states like diabetes mellitus, atherosclerosis, malignancy and cirrhosis. Disease mostly is found to affect middle aged men. Various organisms have been reported to be responsible for necrotizing fascitis. It is thought to be resulting from synergistic bacterial infections, usually combination of streptococcus with other gram positive cocci, gram negative rod and anaerobic organisms. Initially it was thought B hemolytic streptococcus was the only pathogen, now it is shown to be a polymicrobial infection with significant facultative anaerobes. Most common organisms are group A Beta hemolytic streptococcus and Staphylococcus aureus. Other isolates are Group C, G and H streptococci, staphylococci, Hemophilus influenzae type B, bacteroides species and clostridia[9].

High index of suspicion, an early diagnosis, aggressive debridement and supportive care is required for adequate management of this condition. Debriding the necrotic fascia and subcutaneous tissue early in the course of disease can reduce skin gangrene and resultant loss. It is well understood that the necrosis extends beyond the limit of visible skin necrosis, so the soft tissue tissue needs to be extensively debrided. At surgery, the ease of dissection between the fascia and subcutaneous plane is characteristic. Necrotic fascia, thin brown fluid and patchy liquefactive necrosis is seen usually[9]. Repeated debridements are often necessary. Multiple drains are often required to drain and irrigate the wound adequately. Broad spectrum intravenous antibiotics, fluid resuscitation, electrolyte replacement are as important as the surgical approach. When debridement involves soft tissue defects, secondary reconstruction is required. Split or full thickness grafts, fasciocutaneous free flaps like scapula, lateral thigh and radial forearm flaps are the options[9].

CONCLUSION: Necrotising fasciitis is a potentially fatal infection characterized by rapid spread resulting in extensive subcutaneous necrosis and skin gangrene. It needs prompt diagnosis, rapid institution of broad spectrum antibiotics, intense supportive care and aggressive surgical drainage to prevent morbidity and mortality.

SUMMARY

- Necrotising fasciitis is an unusual infection in the head and neck region characterized by rapid spread resulting in extensive subcutaneous necrosis and skin gangrene.

- The aetiology of necrotising fasciitis is not fully understood, and in many cases no identifiable cause can be found.
- High index of suspicion, an early diagnosis. aggressive debridement and supportive care is is the key to a favourable outcome

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Figure 1: A diffuse swelling was noted on the left side of the neck

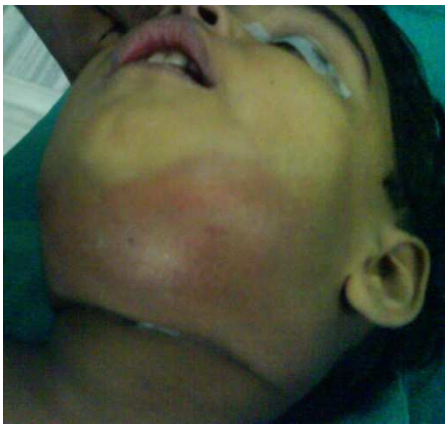

Figure 2: MRI showing subcutaneous spreading inflammatory lesion on left side of face

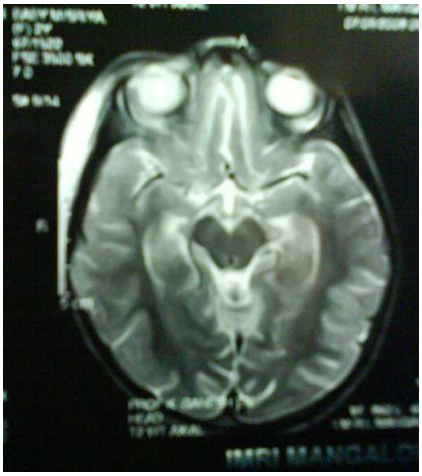

Figure 3: Frank pus pouring out through the submandibular incision.