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Case Report

Cervical Necrotising Fasciitis

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Abstract-

Necrotising fasciitis is a rare but rapidly progressive and potentially life threatening infection of the soft tissue, involving subcutaneous tissue and deep fascial layer. It may affect any part of the body, but the extremities, abdominal wall and perineum are most commonly affected .

Here we present a case of necrotising fasciitis of the neck and anterior chest wall which is a rare presentation . The risk factor of the disease was uncontrolled Diabetes mellitus. We could treat this patient successfully by early recognition, aggressive surgical debridement, intravenous antibiotics, fractional doses of insulin to control Diabetes and other supportive measures.

Key Words- Necrotising Fasciitis, Diabetes mellitus, surgical debridement.

Introduction

Necrotising fasciitis is a serious, devastating and mutilating soft tissue infection. It most commonly affects the extremities, abdominal wall and perineum, but it may affect any part of the body. This infection affecting the perineum was first reported by Baurienne in 1764. The term 'Necrotising fasciitis' was first coined by Wilson in 1952. It has various synonyms like Hospital gangrene (Brooks, 1966) (1), Meleney's gangrene (Meleney, 1924) (2) , Suppurative fasciitis .

Necrotising fasciitis is caused by aerobic and anaerobic microorganisms leading to massive tissue necrosis and septicemia. It is a rapidly progressive disease. Patients with Diabetes mellitus, immunocompression and malnutrition are at high risk. Early diagnosis, aggressive surgical debridement and appropriate intravenous antibiotic therapy can decrease the morbidity and mortality caused by the disease.

Case Report-

A 65 years old male, presented to our ENT Outpatient department, with a wound over anterior aspect of neck, fever and local pain since 5 to 6 days. Initially he had redness of skin of neck and severe pain over the same area, 8 to 10 days back. He took some treatment but had no relief. Skin of the neck turned black in next 2 days followed by pus discharge with persistent pain and intermittent fever. It was at this stage, he presented to us. He had no history of diabetes mellitus or hypertension. He was a chronic tobacco chewer.

Examination revealed, thin built, mildly febrile patient, with vitals maintained. There was an ulcerated wound over anterior aspect of neck extending from hyoid to suprasternal notch and anterior border of sternomastoid muscle on both sides. The floor was covered with dirty slough, with mild local tenderness. Skin of anterior chest wall was erythematous (Fig. 1). An urgent swab was sent for pus culture sensitivity and he was put on i.v. Metronidazole, Amikacin and 3rd generation Cephalosporin.

Laboratory tests revealed, marginally raised white cell count and raised fasting and postmeal blood sugar levels. His liver function and kidney function tests were normal. He was put on fractional doses of insulin. Culture sensitivity report revealed mixed flora and susceptibility of organisms to the antibiotics already started.

Surgical debridement of neck wound was done. After two days, the wound showed no signs of healing. Rather the skin of anterior chest wall turned dark with collection of pus underneath. Plain X-Ray soft tissue neck and chest revealed presence of air in the soft tissue. Looking at the nature of the disease, the diagnosis of necrotising fasciitis was made. Repeat surgical debridement was done where all the necrosed soft tissue and skin of neck and anterior chest wall was excised till there was fresh bleeding from the edges. Post operatively regular cleaning of the wound, antibiotic therapy, fractional doses of insulin and supportive treatment was continued. Drastic improvement in the condition of the patient was observed. Healthy granulation tissue observed and skin grafting was advised to the patient but patient refused .

After discharge, patient came for regular follow up. Complete wound healing was observed with some contracture of neck (Fig.2).

Discussion

Necrotising fasciitis is a severe, rapidly progressive, fatal infection of subcutaneous tissue and deep fascia. Absence of clear local boundaries explains the delay in recognizing the surgical nature of the infection.

The disease can be divided into 5 types [4] –

1. Type I or Polymicrobial necrotising fasciitis.
2. Type II or Group- A Streptococcal necrotising fasciitis.
3. Type III or Clostridial myonecrosis or gas gangrene.
4. Fournier's gangrene.
5. Lemierre syndrome.

Clinically there are two district forms of cervical necrotizing fasciitis, suppurative (characterized by purulent

Fig 1- showing neck wound with necrosis and erythema of anterior chest wall

Fig- 2 showing healed wound with contracture

fluid collection) & gaseous (characterized by gas formation)(3).

There are various risk factors in the development of the disease like diabetes mellitus, immunocompression, alcoholism, malnutrition and peripheral vascular disease. The causative organisms may be aerobes (10%), anaerobes (20%) or mixed flora (70%). Exact mechanism of development of necrotising fasciitis is not known. It is thought to be due to bacterial enzymes. Infection in superficial and deep fascial planes leads to vascular occlusion, ischemia and tissue necrosis. Superficial nerves are damaged causing localised anaesthesia . Signs of sepsis occur as the organisms and toxins are delivered into the blood stream(4).

Necrotising fasciitis is most common in the extremities, perineum & abdominal wall & predominantly occurs in elderly & immunocompromised patients. Few reports can be found in the literature involving the head & neck region & most of these follow an odontogenic or oropharyngeal infection while in cases involving the face or scalp regions, trauma has been the predisposing factor.

In the early stages of disease, the signs & syndromes of necrotizing fasciitis are said to be nonspecific with the skin presenting as red-hot smooth tense & tender without demarcation between involved and normal skin (5, 7). The key pathological process of uncontrolled proliferation of bacteria is angiothrombotic microbial invasion & liquifactive necrosis of the superficial fascia. As this process progresses, occlusion of perforating nutrient vessels to the skin causes progressive skin ischemia. This is the underlying event that is responsible for the cutaneous manifestation of necrotizing fasciitis as the disease evolves. Initially the horizontal phase predominates with rapid spread through the fascia with extensive undermining of the apparently normal looking skin. As the condition evolves, ischemic necrosis of the skin ensues with gangrene of the subcutaneous fat, dermis & epidermis, manifesting progressively as bullae formation, ulceration & skin necrosis.(8,9). Septic shock syndrome is a complication of the disease.

Pain, out of proportion of the external appearance and fascial necrosis are the diagnostic features of the disease. Laboratory tests like WBC count, blood sugar, serum creatinine, X-ray and CT scan may facilitate the diagnosis. Culture of the affected tissue is helpful in antibiotic therapy.

Once the diagnosis is made, immediate surgical debridement, intravenous antibiotics and supportive treatment like control of diabetes and improvement of nutrition is required. It is essential that all the areas of necrotic tissue be debrided (10). Surgical incisions should be deep and extended beyond the areas of necrosis until the viable tissue is reached. The wound should be kept open. Repeated debridement may be needed until the fresh tissue growth is observed. Reconstruction of the tissue defect is to be done

after the control of disease.

Hyperbaric oxygen therapy and intravenous immunoglobulin therapy are advocated at some centers as a part of aggressive treatment regime for necrotising fasciitis.

Mortality rate in necrotising fasciitis is as high as 25% and this rate is much higher in cases with sepsis and renal failure. These infections require an early diagnosis, aggressive surgical debridement and appropriate antibiotic therapy (11) that can successfully reduced the morbidity and mortality rate .

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