Necrotizing Fasciitis

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A rapidly progressing necrosis and oedema of the subcutaneous fat and fascia is sometimes encountered after trauma, insect bites or in the post-operative period. The process has been named necrotizing fasciitis and was first described by Meleney in 1924. It can affect any part of the body but is most commonly seen on the extremities (Defore et al., 1977; Guiliano et al., 1977; Meleney, 1929). The clinical features include tenderness, erythema, swelling depicting extensive cellulitis but showing no response to antibiotics, hot fomentation or raising the affected part (Grossman and Silen, 1962). There is no lymphadenitis and the progress is extremely rapid with the typical skin changes from red and purple to blue grey ill defined patches from 36 hours to 5 days after onset (Guiliano et al., 1977; White, 1953). Frank cutaneous gangrene develops by the 4th or 5th day (White, 1953). Sometimes bullae filled with clear thick pink and deep purple fluid develop on the affected area (Buchanan, 1970). Initially there is excruciating pain but later the regions get anaesthetized due to destruction of the cutaneous nerves. The underlying muscles are spared and can be seen as pink and viable structures beneath the gangrenous tissues (Guiliano et al., 1977). Systemic involvement is always present as extreme toxicity and prostration. Dehydration, electrolyte imbalance and hypoalbuminaemia occur secondary to the massive oedema. Hypocalcaemia (Beathard and Guckian, 1967; Rea and Wyrick, 1970), anaemia and hyperbilirubinaemia due to the haemolytic action of bacteria is a common feature. The infecting organism is mainly from the anaerobic group like Bacteroides. The other causative bacteria was found to be group A Streptococcus. Sometimes there is a combination of both. The etiology for the alarmingly rapid progress of the disease is yet to be determined. Also the reason for the involvement of the connective tissue with sparing of the muscles is not known. The haemolysins, fibrinolysins and hyaluronidase synthesized by the haemolytic streptococci could be a probable mechanism of the spread of the infection (Tehrani and Ledingham, 1977).

Mortality rates as high as 64 percent have been reported (Wilson and Haltalin, 1973). Death usually occurs due to respiratory failure, kidney failure, sepsis or multi-organ failure (Tehrani and Ledingham, 1977). An early diagnosis and surgical debridement helps in survival (Rea and Wyrick, 1970). A diabetic patient with necrotizing fasciitis faces a grave prognosis. Elderly patients with arteriosclerosis malnutrition or obesity have an equally poor chance of survival (Bahary et al., 1977). Lesions of the limbs fare better than those of the trunk and head, because 4they can be better handled (Buchanan, 1970).

Necrotizing fasciitis must be distinguished from erysipelas. The latter condition is accompanied with lymphangitis and Iymphad-enopathy and there is no skin necrosis (Hammar and Wanger, 1977). Cellulitis may be mistaken for this rapidly progressing lesion. Lymphatic involvement and response to appropriate antioiotic therapy differentiates the two. Meleny's ulcer is a spreading cutaneous gangrene which does not involve the deep fascia. It is caused by haemolytic streptococcus in combination with a haemolytic staphylococcus aureus (Grossman and Silen, 1962). The process is slow and it can be distinguished without much difficulty from necrotizing fasciitis.

Clostridial gas gangrene causes muscle necrosis with systemic toxicity and central nervous system changes. Diabetic gangrene is a slow process and is non-infectious.

Necrotizing fasciitis demands immediate surgical intervention. An incision at the level of the fascia and a p obe passed in the fascial plane confirms the condition if there is undermining of the skin (Bahary et al., 1977; Wilson and Haltalin, 1973).

The necrotic tissue should be totally removed through multiple longitudinal incisions in the fascia. The wound should be dressed with mesh gauze impregnated with antibiotics (Rea and Wyrick, 1970), or

hydrogen peroxide (Buchanan, 1970). The dressing should be changed three to four times daily and each time the wound should be probed for undermining of the skin (Rea and Wyrick, 1970). Parenteral antibiotic therapy should be started immediately. As clinical differentiation of the two groups of causative organisms is difficult, the drugs used should provide a coverage for anaerobes and aerobes. Clindamycin and chloramphenicol are effective against most causal agents. Ampicillin, Carbenicillin or cephalosporins with an aminoglycoside will be adequate for the Gram negative bacilli. Metronidazole and cefoxitin may be used for treating the anaerobes (Guiliano et al., 1977).

The hypoproteinaemia, dehydration and electrolyte imbalance should be duly corrected and closely monitored. The use of heparin by some workers (Hammar and Wanger, 1977) in the early stages has provided good results as have high dose steroids (Cotter and Morris, 1962). After the alleviation of toxicity and arrest of the necrotizing process, the open areas should be covered with split-thickness skin autografts.

It could thus be concluded that an early recognition and prompt surgical intervention in a case of necrotizing fasciitis could completely alter the prognosis.

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