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MANUAL

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Topic: CLOSTRIDIAL AND NONCLOSTRIDIAL INFECTIONS IN SURGERY

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Gas Gangrene. Despite the widespread distribution of clostridial spores, gas gangrene is extremely rare in civilian practice (<0.1/100000 per annum). Contamination of wounds which must be common occurrence is, therefore, not usually followed by infection. The essential factor required for spore germination and production of the illness is reduced oxygen tension. This may result from severe contusion and laceration with necrotic tissue, devitalisation of a wound by compression, impaired blood supply, foreign bodies implanted in the depth of a punctured wound such as shrapnel or pieces of clothing (which may cause pressure necrosis and promote pyogenic infection) and soil which induces tissue necrosis by virtue of its high content of ionisable calcium salts and silicic acid. The oxygen tension of a wound may be further lowered by coexisting infection with pyogenic organisms. Gas gangrene has been reported following injection of adrenaline into the buttocks where the skin is often contaminated by clostridial spores from the patient's gastrointestinal tract.

Bacteriology. Gas gangrene is a mixed clostridial infection by saccharolytic (pathogenic) and proteolytic (saprophytic) organisms. The true pathogens are *Clostridium welchii* (*Cl. perfringens*), *Cl. oedematiens* and *Cl. septicum*. Type A, *Cl. welchii* is the most important human pathogen. It produces the α -toxin which is a lecithinase and breaks down the phospholipid constituents of red cells with the production of severe haemolysis. Other exotoxins produced by some strains of *Cl. welchii* include haemolysin (θ -toxin), collagenase (χ -toxin), hyaluronidase (μ -toxin) and deoxyribonuclease (γ -toxin). The exotoxins produce a cellulitis and progressive myonecrosis. They ferment the muscle carbohydrate with the production of lactic acid and gas (H_2, CO_2). The discharge from the wound is initially odourless. Spread of the necrosis occurs as a result of the exotoxin release and ischemia from pressure by gas and exudate within tight fascial muscle compartments. The affected area becomes tense, oedematous and crepitant. Initially, the dead muscle is odourless and brick-red in colour. Progressive putrefaction by the saprophytic clostridia (*Cl. sporogenes*, *Cl. histolyticum*) of the dead muscle mass completes the pathological process with the production of the characteristic foul, fishy odour and the greenish-black appearance of the affected limb. The profound toxæmia is due to the circulating exotoxins which may result in shock, haemolytic anemia, renal failure and jaundice. The organisms themselves do not invade the bloodstream except as an agonal event and this accounts for the foamy liver and gas bubbles found on other organs in some cases at necropsy.

Clinical features. The majority of gas gangrene infections are exogenous and result from contamination of large wounds as obtained in agricultural tractor injuries and battle casualties' appreciable number of cases in civilian practice are however, endogenous in origin and are due to contamination by bowel organism. In the western hemisphere, gas gangrene is most commonly encountered following amputation for peripheral vascular disease. Risk factors in this group include incontinence and diabetes. Other instances of endogenous gas gangrene include criminal abortions and infections following intestinal and biliary surgery. The incubation period between the initiating incident and the onset of the clostridial infection varies from 1 day to 4 weeks and its duration carries an inverse relationship to the severity of the illness and the

mortality. The clinical differentiation of clostridial cellulitis from the more severe clostridial myonecrosis has become increasingly inappropriate with earlier diagnosis and the frequent difficulties in distinguishing one from the other on clinical grounds alone. Furthermore, the demonstration of gas by the elicitation of crepitus or by radiography is not essential for the diagnosis as non-gas-forming clostridial infections of wounds are well documented in all the recent published series. Indeed the most acceptable and useful clinical classification is into gas-forming and non gas forming infections. The most important factor which determines whether the infection remains localised and non crepitant or becomes invasive with the production of severe toxæmia and gas formation is the presence of dead muscle. Thus the majority of cases following amputation for peripheral vascular disease fall in the gas-forming group whereas other operations tend to result in wound infection with little if any gas formation.

Non gas forming Infections. The disease is mild and apart from pyrexia, there is minimal if any toxicity. The wound oedematous and erythematous and may develop a brownish discoloration, crepitus is absent, pain and tenderness are not severe. The mortality directly attributable to the clostridial infection is negligible.

Gas forming Infections. The condition declares itself by the onset of severe pain in the region of the wound and the rapid development of toxæmia, drowsiness, fever and tachycardia. The affected area becomes swollen, tense, oedematous and extremely tender. The discharge is initially odourless but subsequently becomes foul smelling. Gas is detected by the crepitus and by radiological examination. The overlying skin goes through a series of changes from inflammatory erythema to bullae formation and frank greenish-black gangrene. In severe cases jaundice, haemolysis and renal failure develop and often contribute to the death of the patient. The overall mortality is 40 % but the mortality directly due to overwhelming clostridial infection is 11-15%

Treatment. The treatment of gas gangrene consist of general resuscitative measures for shock and specific therapy: antibiotics, hyperbaric oxygen and surgical debridement. Antitoxin therapy has been abandoned because of its questionable value and high complication rate.

Antibiotic therapy. There is now both clinical and experimental evidence to indicate the value of antibiotic therapy in the prophylaxis against gas gangrene both in civilian practice, e.g. amputation for peripheral vascular disease and in battle casualty. The prophylactic antibiotic therapy must be administered adequate dosage and be continued until healing is complete. the benefit of antibiotic therapy is established clostridial infection remains doubtfullargely because of poor antibiotic penetration into ischemic tissue. Nevertheless, it still constitutes standard orthodox treatment in the potential lethal infections and may be effective in cases of mixed infections. Initially parenteral penicillin is administered large doses(1 - 2 megaunits 4 hourly) to all patients except those with penicillin sensitivity where erythromycin, lincomycin and tetracycline have been used. The antibiotic regime is subsequently altered to suit the bacteriology and sensitivity tests of the individual case.

Hyperbaric Oxygen Therapy. Despite the continued controversy, there is now sufficient retrospective information to indicate that hyperbaric oxygen therapy does benefit patient with pure clostridial infections and may result in rapid improvement in the clinical condition and in limb salvage. Hyperbaric oxygen therapy is started soon after the initial resuscitation and before surgical intervention. It consists of repeated treatment of 1/2hourse at a pressure of 250kPa.

Surgical Treatment. This is carried out after the first treatment in the hyperbaric chamber (if it is available locally). The aim of surgical treatment is adequate debridement with drainage and wide excision of necrotic tissue. Not infrequently this necessitates an amputation. Uterine infections are usually treated by evacuation and more rarely by hysterectomy. It is imperative that all devitalised tissue is removed even if this requires repeated excision. The surgeon should not be deterred by anatomical defects thus produced. Reconstructive surgery can be completed later if the patient survives.

Non-clostridial infective gangrene. Various clinical syndromes have been described as infective non-clostridial gangrene. The most common causative organisms are anaerobic streptococci but necrotizing infection with E.coli and bacteroids are well documented. The most common member of bacteroids species responsible for infective gangrene is Fusiformis which is often accompanied by Borrelia vincentii al though is doubtful if latter plays any part in the disease process. These gangrenous conditions usually arise on the background of debility and the causative e anaerobes often acts in association with Streptococcus pyogenes, staphylococci and coli form bacilli. However in some well-documented cases of infective cutaneous and subcutaneous gangrene, a causative organisms has not been isolated, despite repeated attempts at culture in some cases a precipitating factor, e.g. trauma, operation or viral infection, initiates the infection in others particularly in diabetic patients, the morbid process arises spontaneously. The syndromes that come into the broad category of non-clostridial infective gangrene include: cancrumoris and noma vulva Meleney's postoperative synergistic gangrene; Meleney's chronic undermining ulcer; anaerobic non-clostridial wound infection; Fournier's spontaneous scrotal gangrene; necrotising fasciitis; progressive dermal gangrene; puerperal gangrene.