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Major crush injury

Crushing injuries produce a wide range of pathological changes: minor and major but short lived ones are generally well managed, though failure to recognise the full extent of the damage may delay healing. Longer term compression of tissues, however, is less well understood.

Limb compression can injure through either ischaemia or muscle destruction,[1] and, though the end result may be similar, the initial treatment is different. A pressure of 40-60 mm Hg will start functional changes in nerves and muscles after 15 minutes[2] and can lead to ischaemic necrosis within four to eight hours.[3] Systemic manifestations of muscle damage and necrosis may follow prolonged pressure from a patient's own body weight when he or she is unconscious after a drug overdose[4] or while undergoing an operation.[5] The necrosis may also result from antishock garments.[6] Compartmental syndromes, with or without trauma, often go unrecognised because the appearance of the limb may be misleading. Pain, swelling, sensory change, weakness, and pain on passive stretching are present in varying combinations.[7] Any progressive neurovascular deficit should be determined by clinical examination supported by direct pressure measurement or electrical conductivity testing. These tests are essential, for the longer treatment is delayed the greater the chance of permanent damage. Computed tomography may help to show areas of myonecrosis.[5] Adequate decompression by fasciotomy or epimysiotomy, or both, is the treatment. The dead limb with imminent or established gangrene should be recognised and treated. Late arterial and nerve reconstruction may salvage length, but function is often severely impaired.[8]

The more severe crush injuries seen in patients trapped often for hours and often from natural disasters must have existed for years. They were mentioned during the first world war, but not until 1941 was the problem satisfactorily described.[9] The crush syndrome of acute renal failure, infection, ventilatory complications, neuropsychiatric disturbances, stress ulcers, and coagulopathies is now well recognised. For the past 10 years there have been on average two major disasters a year in Britain, all of them different and few of them producing the pattern of injuries associated with earthquakes or warfare.

Major crush injuries damaging more than one system, especially in the trunk or head, are often fatal for rescue is often delayed.[10] Injuries to the limbs are survivable, and published accounts deal almost exclusively with leg injuries. Since the Moorgate tube disaster in 1975, when two patients died of crush injuries,[11] only sporadic reports have appeared from Britain.[12]

Muscle severely damaged by entrapment releases its contents into the circulation on being freed. These patients, like those with severe compartment syndromes, have a rising blood urea nitrogen concentration; high serum concentrations of potassium, phosphate, and uric acid; a raised anion gap; a raised packed cell volume, thrombocytopenia, and often myoglobinuria.[4] Serum creatine kinase activities greater than five times normal are diagnostic of muscle damage in the absence of other injuries.[13] The high serum potassium concentration, especially if associated with a low serum sodium concentration, may cause cardiac arrhythmias and arrest soon after release.[14] Recognition of the severe crushing injury is essential, with shock being added to the previously mentioned signs. Peripheral pulses are frequently present initially, giving a false sense of security. Prehospital treatment should be aimed at preserving the sodium concentration during treatment for shock. Analgesics may help reduce the shock and will probably be needed for pain relief. Stewart and others have suggested that a tourniquet should be applied before freeing the limb and not released until arrival in the hospital, but the theory may be easier than the practice.[15]

Treatment has been aimed at preventing the sequelae, and forced alkaline diuresis with bicarbonate and mannitol will help protect the kidneys.[16,17] Sepsis is the greatest scourge, and closed injuries should be treated conservatively.[3] Limb elevation is not recommended.[7] Open wounds may need complete debridement of all muscle, and even in theatre damage is deceptive in its colour and back bleeding.[17] Mechanical or electrical stimulation, or both, may be required to tell if the muscle is alive; any dead muscle invites infection and secondary haemorrhage. If a patient with closed injuries deteriorates then extensive fasciotomy and debridement are needed.

Early amputation should be considered in patients with open wounds or in those deteriorating, but there is no single indication, although a ratio of creatine to creatinine greater than 1·10 is a pointer.[1] Any delay will accentuate the complications.[1] The morbidity from major crush injuries may still be high, but the mortality should be less than the 60-70% of 30 years ago.[18]

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Collagenous colitis: disease or diversion?

Unexplained chronic diarrhoea is common, and in 1976 a distinctive histological change of the large intestinal mucosa termed "collagenous colitis" was proposed by Lindstrom as a cause of chronic watery diarrhoea.[1] Ten years later more than 50 cases have been described.[1-15] The histological hallmark of collagenous colitis is a thick eosinophilic band in the superficial aspect of the colonic mucosa, immediately deep to the surface epithelium and sometimes extending around the uppermost part of the crypts. Examination by histochemistry,[2 3] electron microscopy,[3 14 16 17] and immunocytochemistry[9 18] has confirmed that the band is collagenous and has shown that there may be subtle changes in the adjacent epithelium, capillary endothelium, and pericryptal fibroblasts.[14 17] Subepithelial collagen in the colonic mucosa is not in itself pathological (its synthesis is a physiological function of the pericryptal fibroblast sheath related to epithelial cell turnover[19 20]), but it is usually inconspicuous on routine examination by light microscopy.[21] Most studies of its thickness in "control" samples show that the upper limit of normal can be taken as 7 μm,[3 5 9 11 22 23] whereas in collagenous colitis it usually exceeds 15 μm and in many cases reaches 60-70 μm.[1-15]

The cause of the excessive collagen deposition is a mystery. Although the term collagenous colitis trips easily off the tongue, the colitis is often unimpressive on histological examination. In most cases inflammatory cells, usually lymphocytes and plasma cells,[6] are only modestly increased in the mucosa, but sometimes eosinophils[7] or mast cells[9] are conspicuous. Nevertheless, the changes of collagenous colitis have occasionally followed a histologically proved acute, but non-specific, mucosal inflammation.[10 12] A thickened collagen plate is not a feature of ulcerative colitis, Crohn's disease, or any hitherto recognised variety of inflammatory bowel disease.[5 9 24]

Collagenous colitis occurs almost exclusively in women, and they may be almost any age (range 23 to 81 years).[1-18] Apart from the chronic watery diarrhoea, which may be present for weeks or many years, patients are remarkably well.[12] No firm association has been made with any other medical condition, although polyarthritis or rheumatoid disease has featured in some reports.[11 15 25 26] Routine haematological and biochemical measurements are usually normal, microbiological investigations are negative, barium studies are generally unremarkable, and endoscopic abnormalities of the large bowel mucosa are nearly always unimpressive, even if present. The term collagenous colitis was originally chosen by Lindstrom by analogy with collagenous sprue, a lesion of the small intestine in which villous atrophy and a subepithelial collagen band are associated with malabsorption,[27] but the two lesions are unrelated clinically and do not coexist. Collagenous colitis is confined to the colon and is usually diffuse,[7 13 23] albeit with some variation in the collagen thickness. Usually the change is apparent in a rectal biopsy, although not always.[22]

The course of the illness is variable: some patients have chronic persistent diarrhoea[14]; some have relapses and remissions[10-13]; and a few have apparent spontaneous resolution either with[7 8] or without[10 28] disappearance of the abnormal collagen band. Conventional medical treatment is usually ineffective,[12] although there are reports of improvement with metronidazole,[11 29] mepacrine,[4] prednisolone,[10 13] sulphasalazine,[23 26] and loperamide[11] and also with the homoeopathic remedy Natrum Mur.[12] But even those people with persistent symptoms suffer little detriment to their general health.[12]

As chronic watery diarrhoea is common and the histological changes of collagenous colitis seem to be rare the relation between them must be questioned critically. Does the thickened collagenous band cause diarrhoea by forming a barrier to water absorption, as suggested by Lindstrom,[1] or is it a consequence of the diarrhoea? Or do the collagenous band and diarrhoea coexist by chance? Two perfusion studies have supported Lindstrom's hypothesis by showing net secretion of sodium chloride and water into the colonic lumen,[18 30] but a third study gave conflicting results.[15] That the thickened collagen plate is a mere consequence of diarrhoea is unlikely because it is not found in other diarrhoeas of known cause.

Some aspects of collagenous colitis make us sceptical of its clinical importance; sometimes the band is seen without the diarrhoea,[22] and the band may persist after the symptoms have resolved.[10 28] Nor do the absence of a recognised aetiological agent, the widely variable clinical course, and the lack of a uniform response to many treatments support a specific disease process. Most published reports are of small numbers of cases with varied emphasis, and many contain conflicting findings. What is now needed is a coordinated effort to collect details of many carefully investigated patients. Meanwhile we conclude that while chronic watery diarrhoea and substantial thickening of the collagen plate undoubtedly coexist in some patients there is insufficient evidence to accept collagenous colitis as a disease entity. The onus remains on the protagonists of collagenous colitis to show that it is more than a histological change of questionable importance.

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