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Frozen shoulder: adhesive capsulitis

Among the numerous causes of pain felt in the shoulder are referred symptoms arising from disorders of the neck, lungs, heart, diaphragm, and even median nerve compression at the wrist. Even when the shoulder itself is at fault the possible causes are many: examination must include the glenohumeral joint, the sternoclavicular and acromioclavicular joints, and scapular rotation on the chest wall to distinguish the various conditions. These include arthritis of synovial joints, inflammation of bursae, inflammation and sometimes calcific deposits in the supraspinatus tendon, other rotator cuff lesions, and bicipital tendonitis. Particularly disabling is the development of a painful stiff shoulder, also known as a frozen shoulder or adhesive capsulitis.

The pathological basis of frozen shoulder has been described by Neviasa,[1] who found at surgery a conspicuous absence of synovial fluid in the glenohumeral joint and a tight thickened capsule under tension applied to the humeral head, so limiting shoulder movements. He compared adhesions of the capsule with that of an adhesive plaster on the bare skin—hence the term "adhesive capsulitis." Manipulation of the shoulder tore the adhesions apart and made free movement of the humerus possible. Histological examination of the capsule showed only fibrotic and reparative inflammatory changes.

Typically, the symptoms include increasing and severe pain in and around the shoulder with progressive loss of both active and passive glenohumeral movements. In particular, external rotation of the shoulder becomes extremely restricted, with lesser loss of abduction and internal rotation.[2] The pain may be severe and may persist throughout the night and lead to considerable distress. Radiographs are usually normal but may show minor osteoporotic or degenerative changes of doubtful importance. Degenerative changes in the neck are common.[3] Arthrography[4] shows shrinking of the joint capsule with reduced capacity to accommodate contrast medium.

Why frozen shoulder should occur remains uncertain, but some factors are known. The peak incidence[5 6] is between 50 and 70 years and is slightly higher in women than men. Frozen shoulder may develop spontaneously but is a particular risk for patients with a hemiplegia or other conditions in which the arm may be immobilised, such as after thoracic surgery, myocardial infarction, and cervical herpes zoster. A prospective study[2] of patients admitted to the Atkinson Morley Hospital with cerebrovascular disorders, most of whom underwent neurosurgery, found that one-quarter developed frozen shoulders. The risk correlated with impairment of consciousness, the development of hemiparesis, the duration of postoperative intravenous infusions, increasing age, and depression. Other factors that have been blamed include recurrent trauma to the shoulder, perhaps associated with manual work, thyroid disease, ischaemic heart disease,[6] repeated ingestion of phenobarbitone[7] and isoniazid,[8] and diabetes.[9]

The known vulnerability of the microvascular supply of the supraspinatus tendon[10] and a finding of inflammatory reactions to small areas of infarcted tendon[11] led to immunological studies in patients with frozen shoulders. These showed lowered concentrations of immunoglobulins, reduced transformation of lymphocytes by phytohaemagglutinin,[12] and an increased incidence of white cell type HLA-B27.[13] Again, the interpretation of the findings is problematical.

Many doctors looking after patients with frozen shoulder believe they are psychologically abnormal—the so-called periarthritic personality,[14] with a passive apathetic attitude, muscular tenseness, and a low pain threshold. This profile has not, however, been confirmed in studies of personality inventories,[2 6 15] and raised scores for depression, anxiety, and hysteria that have been recorded may well reflect the effects of a persistent unpleasant disorder.

Fortunately frozen shoulder is usually self-limiting. Symptoms develop over about six months and may last a year or two; they then gradually disappear, though there may be some long-lasting pain and restriction of motion. Against this background the various treatments in common use remain far from proved. Mobilising physiotherapy is usually combined with intra-articular injection of local steroids, perhaps also with manipulation under an anaesthetic.[5 16 17] Radiotherapy,[18] sympathetic ganglion block,[19] and oral steroids have been suggested, but there is no justification for their use.

Prevention has an important part to play. In many cases a frozen shoulder may be traced to a combination of immobility and trauma. In particular, the joint is easily damaged in patients who have had a stroke. Common sense indicates that extreme care should be taken to protect the shoulder against undue stress when moving hemiplegic patients and in seating them in chairs. Careful active and passive exercises, not going beyond the normal ranges of motion, should be performed to maintain movements of the shoulders. Adhesive capsulitis

should be preventable in many patients at particular risk of this unpleasant problem.

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Indian childhood cirrhosis

A unique liver disease, which has resisted all attempts at understanding, affects young children in the Indian subcontinent and Malaysia. It is said to be extremely rare among expatriate children; information about the number of cases encountered in Britain (or in other parts of the world where Indians have settled) would be valuable. The disease is not confined to any one religious group or social class (a preponderance of cases from the upper classes is due to their better access to medical care), and in a third of cases it affects more than one member of the family.

In most of the children[1] non-specific symptoms, abdominal distension, and enlargement of the liver are succeeded in a few years by death from decompensated cirrhosis, causing either liver failure, ascites, or bleeding from portal hypertension. In a quarter the illness is more acute and resembles a continuing hepatitis, with death from liver failure in a few months. The liver damage is striking and unusual. The characteristic features are appreciable necrosis of liver cells with little sign of regeneration, extensive deposition of hyaline (especially in liver cells), patchy and "aggressive" fibrosis throughout the parenchyma, very little fat, and a scanty inflammatory cell response. The resulting damage has been called "micro-micronodular" cirrhosis.

The familial incidence suggests a genetic susceptibility to one or more environmental hazards, but no definite cause has been recognised. Malnutrition is clearly not a candidate, though some dietary substance or deficiency peculiar to a particular culture might still be responsible. Cell-mediated immunity is depressed in some patients and on the basis of raised concentrations of alpha-fetoprotein in nearly half their cases, Nayak *et al*[2] made the ingenious suggestion that persistence of fetal hepatocytes made the children vulnerable to an environmental agent. One of the hepatitis viruses is an obvious choice. The frequency of hepatitis B surface antigen, however, though high, is not remarkable for these populations, and no sign of hepatitis B has been found in biopsy or necropsy specimens of liver tissue.[3] Ingestion of a hepatotoxin such as aflatoxin seems to be disproved by the histological appearances, but perhaps ayurvedic remedies should not be entirely ruled out, since drugs like griseofulvin and colchicine cause hyaline deposits in the liver of animals.

Recent interest has centred on the finding of large amounts of copper and copper-binding protein in the liver.[4–6] This feature, with the hyaline deposits, is reminiscent of Wilson's disease. Whether these appearances are primarily metabolic or the consequence of the disease—as in prolonged cholestasis, for example—is not yet clear, though their distribution in liver cells favours a causative role. But careful study[7] of siblings of affected children has shown only minor "non-specific" changes in liver biopsy material and no excess of copper, and prolonged follow-up of 200 siblings has not produced a single case of Indian childhood cirrhosis. Obviously the next step is a controlled trial of penicillamine. Nevertheless, while we naturally hope that children will benefit, use of the drug may not throw any new light on this curious disease.

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