■ REVIEW ARTICLE

Disorders of the sternoclavicular joint

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The sternoclavicular joint is vulnerable to the same disease processes as other synovial joints, the most common of which are instability from injury, osteoarthritis, infection and rheumatoid disease. Patients may also present with other conditions, which are unique to the joint, or are manifestations of a systemic disease process. The surgeon should be aware of these possibilities when assessing a patient with a painful, swollen sternoclavicular joint.

The sternoclavicular joint is the forgotten articulation of the shoulder girdle. Over the last 20 years there has been a considerable expansion in the range of conditions affecting the glenohumeral and acromioclavicular joints which can be successfully treated by open or arthroscopic surgery. By contrast, most disorders of the sternoclavicular joint continue to be treated conservatively because of the poor results associated with surgical treatment and the potentially catastrophic complications with which such surgery is associated. Nevertheless, injuries to the joint, and infective, inflammatory and degenerative arthritides, are relatively common and may be the source of chronic disability if they are inadequately diagnosed and treated. In addition, a number of relatively unusual conditions such as the syndrome of synovitis, acne, pustulosis, hyperostosis and osteitis (SAPHO), condensing osteitis and Friedrich's disease[1] have a predilection for this joint, and are being recognised with increasing frequency. In this review, we summarise the clinical presentation and optimal treatment for those disorders which commonly affect the sternoclavicular joint.

Anatomy of the sternoclavicular joint

The sternoclavicular joint is a true diarthrodial joint, which forms the only synovial articulation between the upper limb and the axial skeleton. Movement at the joint can occur passively in three planes, and is usually produced by transmission of movements of the scapula on the chest wall. During abduction of the shoulder the sternoclavicular joint can elevate 35° in the coronal plane, and has a range of movement of 70° around neutral in the anteroposterior plane.[2] The expanded medial end of the clavicle articulates in a saddle joint with the superomedial manubrium.[3] The joint is shallow and its stability is therefore mainly dependent upon the integrity of four distinct ligaments, and the subclavius muscle. The latter has been shown to stabilise the joint, even after considerable rupture of the ligaments.[4] Of the four ligaments, a strong capsular ligament is formed by anterosuperior and posterior capsular thickenings, and its disruption can cause superior translation of the joint.[5,6] The ligament inserts at the medial clavicular epiphysis, which appears in late adolescence and usually ossifies by the age of 25 years.[2,7,8] As a consequence, many apparent injuries to the sternoclavicular joint in teenagers and young adults may actually be physeal separations. The other passive stabilisers of the joint are the intra-articular disc ligament and the extra-articular interclavicular and costoclavicular (rhomboid) ligaments. The strongest of these is the latter, which consists of anterior and posterior bands attached to the first rib.[9] These have been shown to limit rotation and to provide medial and lateral stability of the joint.[5]

The sternoclavicular joint lies in a subcutaneous position and swelling of the joint, or expansion of the adjacent bone, is therefore apparent at an early stage of disease. The thoracic inlet lies posteriorly, and contains the great vessels of the superior mediastinum, trachea, oesophagus, vagus and phrenic nerves. These structures are at risk during an acute posterior dislocation of the joint and fatalities have been reported from intrathoracic injury.[10] The proximity of these structures to the joint also places them at risk during surgery, and loose or unstable implants may migrate into the superior mediastinum to cause potentially fatal complications.[11]

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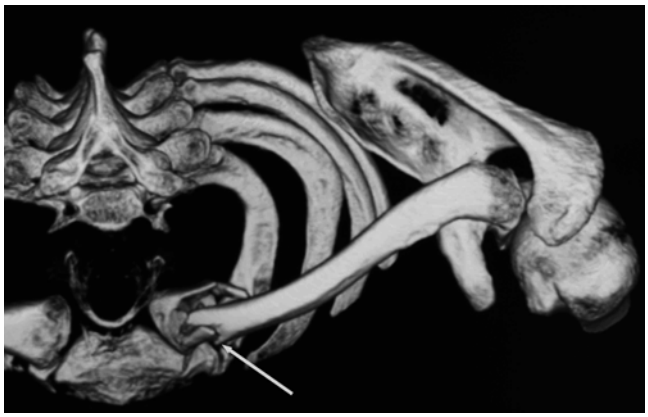

Fig. 1

A superior 3D CT reconstruction of a displaced and comminuted fracture of the medial end of the left clavicle (arrow) in a 40-year-old man.

Disorders of the sternoclavicular joint

The most common clinical presentation is pain and swelling in the area of the sternoclavicular joint, either after an injury to the shoulder, or insidiously, with no history of trauma. In both clinical situations, the localisation of the symptoms to the joint is usually clinically obvious, but the substantive diagnosis cannot be made without specialist imaging.

Injury to the sternoclavicular joint

Epidemiology

Dislocations of the sternoclavicular joint are less common than those of the glenohumeral and acromioclavicular articulations[12,13] and comprise 1% of all joint dislocations, and 3% of those in the upper limb.[2] They generally occur in active, young males as a consequence of the high-energy mechanism of injury.[14,15] Minor sprains of the joint and undisplaced medial physeal injuries are more common than dislocations, and many of these patients may not seek medical attention. Instability may be classified by direction (anterior or posterior), aetiology (traumatic or atraumatic), severity (sprain, subluxation or dislocation which are sometimes referred to as types I, II and III, respectively) and onset (acute or chronic).[16]

Pathophysiology

Dislocation of the sternoclavicular joint is uncommon because the energy produced by blunt trauma usually causes sprains, fractures or dislocations of the more peripherally-placed bones and joints of the shoulder girdle. A force of substantial magnitude and of particular vector is required to disrupt the strong ligamentous constraints of the joint. This commonly results from road-traffic accidents, sporting injuries and falls from a height.[14,15] Anterior and posterior dislocations are produced by indirect forces applied to the shoulder girdle from the anterolateral and posterolateral directions, respectively. This force vector compresses the humeral head against the glenoid, and is transmitted across the shoulder girdle to the sternoclavicular joint. A posterior dislocation may also occur from a direct force applied to the medial end of the clavicle, but the indirect mechanism of injury is more common than this direct mechanism.[17]

Anterior dislocations are much more common than posterior dislocations,[14] and if the ligamentous structures fail to heal after closed reduction of an acute traumatic dislocation, the patient may develop recurrent symptomatic instability of the joint. This most commonly affects adolescent females, who usually have evidence of generalised ligamentous laxity.[18] The patient typically experiences subluxation on elevating the arm, which spontaneously reduces when the arm is lowered. The subluxation is often painless, and causes no restriction of daily activities.[19] Atraumatic subluxation and dislocation may also arise spontaneously or be the result of chronic palsy of trapezius.[20] This is usually a benign, self-limiting condition and surgical intervention is only recommended as a last resort.[15,18,20] Trapezius palsy may also be caused by dysfunction of the spinal accessory nerve secondary to radical dissection of the neck.[21,22]

Clinical presentation

Patients usually present with a history of trauma and subsequent pain and sternoclavicular deformity. The direction of an acute dislocation may be apparent on inspection, with prominence of the medial clavicle in anterior dislocation and a palpable defect adjacent to the sternum in posterior dislocation. Mediastinal compromise can occur in up to 25% of posterior dislocations.[15,23,24] Venous congestion in the neck or ipsilateral arm, hoarseness, cough, dysphagia or a feeling of choking suggest superior mediastinal obstruction from posterior displacement, and are indications for urgent reduction. The whole clavicle should be examined and distal tenderness should alert suspicion of a concomitant injury to the acromioclavicular joint ('floating clavicle') or clavicular fracture.[25,26]

If presentation is delayed, or the soft-tissue swelling is severe, the signs may be more subtle and specialist imaging may be required to confirm the direction and severity of the injury. In young adults (under 25 years of age) the distinction of a sternoclavicular dislocation from a Salter-Harris type-II physeal separation or medial clavicular fracture, often described as a 'pseudodislocation', may be difficult even with cross-sectional imaging.[15,27]

Investigation

Standard plain anteroposterior radiographs should be obtained in the assessment of an acute injury to the sternoclavicular joint. These may be supplemented by additional views, such as the oblique (Hobbs), lateral (Heinig) and 'serendipity' (40° cephalic tilt radiograph showing both sternoclavicular joints) views.[28] The interpretation of these is hindered by artefacts from neighbouring structures and their clinical use has been superceded by CT, with its ability to

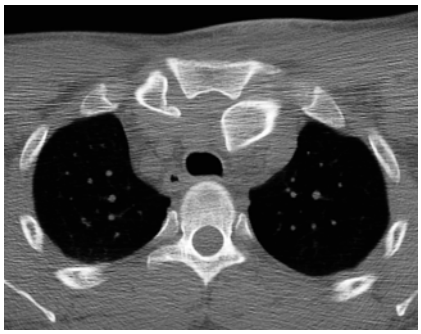

Fig. 2a

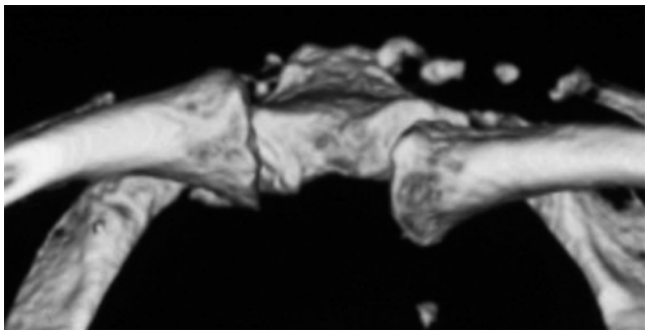

Fig. 2b

a) An axial CT scan of a posterior dislocation of the left sternoclavicular joint in a 30-year-old man. Note the proximity of the dislocated clavicle to the adjacent mediastinal structures. b) A superior 3D CT reconstruction of a posterior dislocation of the right sternoclavicular joint.

provide multiplanar and three-dimensional reconstructions (Fig. 1). These can distinguish between medial clavicular fractures, physeal separations and disruptions of the sternoclavicular joint.[29] Concomitant angiography should be performed if obstruction of the thoracic outlet or vascular injury is suspected. MRI may be useful in assessing the extent of associated soft-tissue damage after injury, but gross displacement of the bony structures may hinder interpretation.[30]

Treatment

Minor sprains and subluxations of the joint (grade-I and grade-II injury).[15] Minor sprains, joint subluxations and minimally-displaced medial physeal separations respond well to conservative measures such as oral analgesia, topical ice and a short period of immobilisation in a sling. The patient should be advised to avoid re-injury for three months, and contact sports or other high-risk activities should be prohibited, until there is clinical evidence of resolution.[20]

Dislocations (grade-III injury)[15]

Closed treatment. Acute (diagnosed within 48 hours of injury) anterior dislocations are usually unstable and should undergo attempted closed reduction under general anaesthesia.[19,20,24,31,32] Reduction of anterior dislocations is achieved by direct pressure over the medial portion of the clavicle of a supine patient with a solid pad placed between their shoulders. Patients should be counselled that there is a risk of recurrent instability, but that this rarely results in a functional deficit.[19,20,33]

Acute posterior dislocations (Fig. 2) have greater potential complications and should be reduced promptly. They may cause mediastinal compromise.[17] To reduce a posterior dislocation the arm is abducted and traction is applied while the shoulder is moved into an extended position. If this is ineffective, or if the dislocation occurred more than 48 hours previously, traction may be applied to the medial clavicle with sterile reduction forceps.[24] An alternative method is to position the arm in adduction at the patient's side and to apply traction at the same time as direct pressure to the front of the glenohumeral joint.[23,31] The stability of a successful closed reduction should be assessed under the same period of anaesthesia, since this will help to determine if subsequent re-displacement is likely to occur. The patient should always be admitted to hospital thereafter and monitored for signs of mediastinal obstruction. Acute posterior dislocations are often more stable once they have been reduced than anterior dislocations.[17] Patients should be treated by immobilisation in a sling and restriction of activity for six weeks after injury.

The use of external splints, figure-of-eight bandages and local pressure dressings are ineffective in preventing re-displacement of unstable reductions. Anterior dislocations which are either unstable or irreducible can often be accepted and treated by physiotherapy, modification of activity and strapping.[18] In young individuals, a substantial degree of remodelling can be anticipated during growth,[34,35] and the functional deficit with such skillful neglect is often minor.[15,33] Late thoracic outlet syndrome, exertional dyspnoea and chronic vascular insufficiency have been described in association with chronic posterior dislocations,[36,37] and degenerative change may occur in the incongruent chondral surfaces.

Operative treatment. Posterior dislocations which have failed to reduce after closed manipulation may cause cardiovascular compromise and therefore require urgent open reduction. This contrasts with anterior dislocations and instability in which surgery is only indicated when there is compromise of the skin, or in the rare cases in which conservative management has failed and there remains considerable pain, crepitus and symptoms of instability.[19,20]

Operative interventions for acute posterior dislocation include open reduction and soft-tissue stabilisation or resection of the medial clavicle. The proximity of the adjacent great vessels dictates that a cardiothoracic surgeon should always be available in theatre when open

surgical treatment of a posterior dislocation is attempted. A skin crease incision is made centred on the joint and forceful traction may need to be applied to the abducted shoulder to reduce the clavicle from its retrosternal position under direct vision. Application of reduction forceps to the medial clavicle may improve the direct traction and facilitate reduction.

The normal ligamentous stabilisers are usually unreconstructable after open reduction, but the reduction will usually be unstable unless some form of soft-tissue ligamentous reconstruction is attempted. These utilise grafts from various tissues including fascia lata, palmaris longus and the tendon of semitendinosus.[6,38] The last is typically inserted looped through drill holes in the medial clavicle and manubrium, and secured by sutures. The recent introduction of ultra-strong synthetic braided sutures, such as Orthocord (DePuy Mitek, Raynham, Massachussetts) and Fibrewire (Arthrex, Naples, Florida) may be useful in augmenting these soft-tissue reconstructions.

The results of stabilisation using ligament substitution have been mixed, with a high prevalence of soft-tissue complications and failure of the reconstruction resulting in a recurrence of the deformity. The best outcomes were shown with the use of a semitendinosus graft configured in a figure-of-eight arrangement through two pre-drilled holes in the clavicular head and manubrium.[6]

The use of Kirschner wires or Steinmann pins is contraindicated because of the complications which may be associated with their migration into vital structures. These include penetration of the heart, pulmonary artery, aortic arch, subclavian artery, lung and mediastinum.[11,39] The use of more stable implants such as wire sutures, suture anchors and custom-made plates (Balser; Peter Brehm, Chirurgie-Mechanik, Wiesendorf, Germany)[27,40] has been advocated, although these devices have a substantial risk of intrathoracic migration if breakage of the implant or loosening occurs. None of these techniques has been widely used or independently tested. Arthrodesis of the joint is also contraindicated because of the marked restriction in shoulder movement which it produces.

In the unusual situation in which an open reduction cannot be achieved or when there is no residual soft-tissue attachment to the medial clavicle after open reduction, an osteotomy may be performed with resection of approximately 1 cm to 2 cm of the medial clavicle.[41] The remaining medial clavicle should then be stabilised to the first rib by a ligamentous repair or augmentation, since studies have shown that the outcome of resection is poor without adjunctive soft-tissue stabilisation.[42,43]

The rarity of the floating clavicle injury means that evidence-based guidelines for its treatment are difficult to produce. In younger individuals, operative reduction and stabilisation of one or both ends of the clavicle are probably indicated, whereas in older, low-demand individuals, conservative treatment may produce satisfactory results.[44]

Non-traumatic disorders of the sternoclavicular joint

Clinical presentation

The sternoclavicular joint is a true synovial joint, and is therefore susceptible to the normal spectrum of inflammatory and degenerative arthritides, as well as a number of other disorders which are unique to the joint. Such disorders can present either acutely or insidiously with localised pain and swelling. A protocol for their investigation and treatment is shown in Figure 3. On examination, there may also be localised tenderness, warmth and bony enlargment. A full medical history should include enquiry about systemic illness, other arthritides and recent infection. Careful recording of the drug, social and family histories will clarify risk factors and aid the construction of a differential diagnosis (Table I).

Investigation

Conventional anteroposterior radiographs may show subchondral sclerosis, osteophytes, narrowing of the joint space, hyperostosis and calcification of the surrounding tissues, but may be difficult to interpret because of artefacts from the underlying superior mediastinal structures.[45] CT may detect disease processes which cause bony destructive or ossifiying lesions while MRI gives better resolution of inflammatory soft-tissue enlargement, and is also useful for detecting osteonecrosis of the medial clavicle (Friedrich's disease[1]). Isotope scanning may also be useful in assessing the degree of activity in the joint, and may help to detect remote foci of inflammation.

Laboratory blood tests are a useful adjunct to radiological investigations. General inflammatory markers (white cell count, erythrocyte sedimentation rate (ESR) and the level of C-reactive protein (CRP)), the level of rheumatoid factor and antinuclear antibodies and tissue antigen tests for HLA-B27 are useful initial screening tests. Inflammatory markers are all raised in septic arthritis, and the ESR may be disproportionately elevated in chronic inflammatory conditions such as hyperostosis.

Joint aspiration is best performed under ultrasonographic guidance to confirm that the needle has entered the joint and to prevent inadvertent damage to the mediastinal structures nearby. Joint fluid is analysed by light microscopy after Gram staining, and cultured for bacterial growth. A raised white cell count ($> 50\,000 \times 10^6$), or the presence of known pathogens on microscopy, is highly suspicious of infection, although if antibiotics have been administered before sampling then a lower white count may be significant. Crystal-induced monoarthritis may be diagnosed by microscopy and examination of the birefringent characteristics of the crystals.

Non-traumatic causes of sternoclavicular pain and swelling

Systemic arthritides

Osteoarthritis. This is the most frequent cause of pain and swelling of the sternoclavicular joint. The swelling may be asymptomatic, and moderate to severe degenerative joint

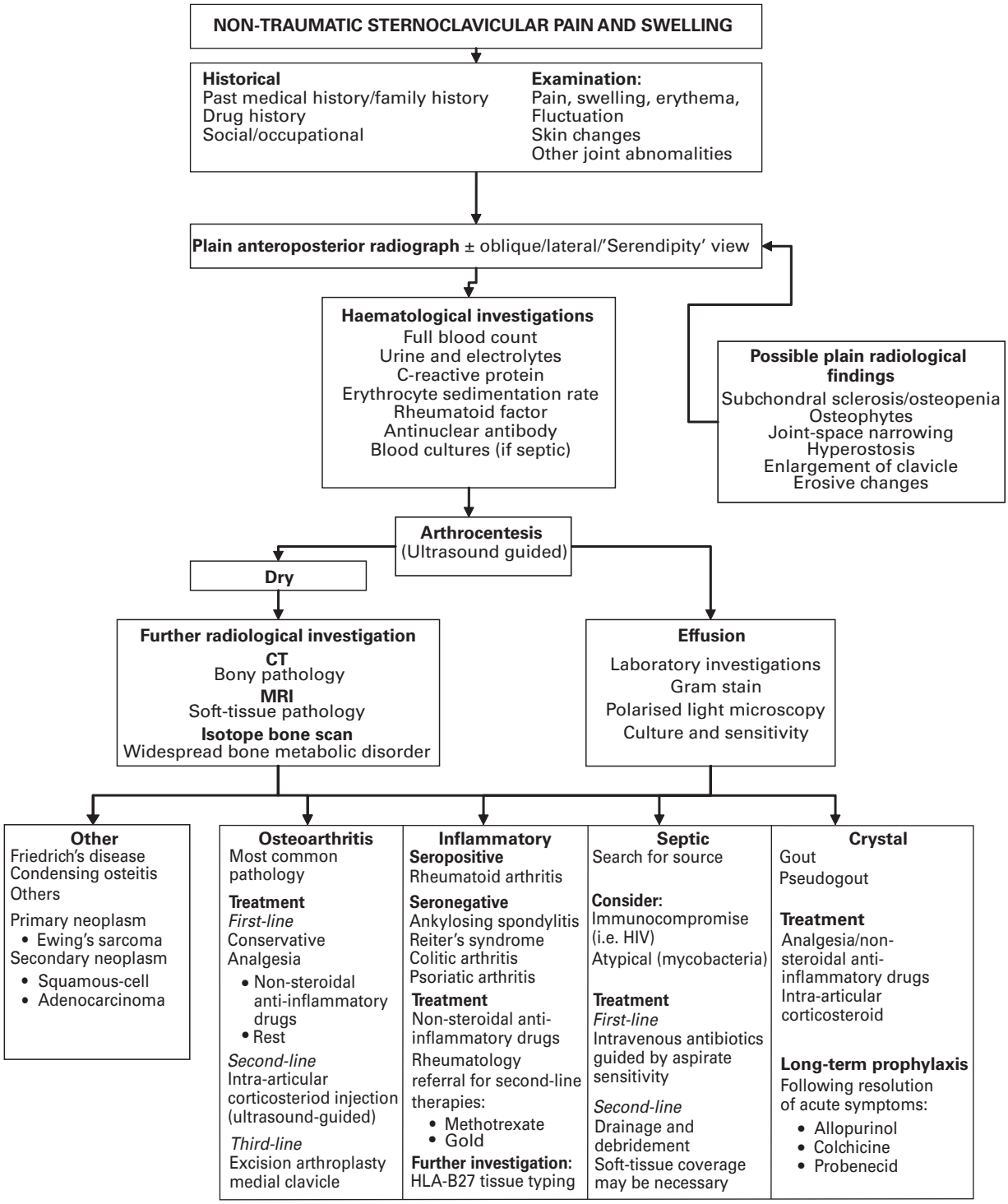

Fig. 3

Diagram giving the diagnostic protocol for the investigation of non-traumatic sternoclavicular pain and swelling.

changes have been found at post-mortem in over 50% of individuals aged over 60 years.[46] High-risk groups include post-menopausal women, patients with chronic sternoclavicular instability and manual labourers.

A symptomatic joint is characteristically painful on abduction or forward flexion of the shoulder beyond 90°. Examination may reveal osteophytic prominence over the medial clavicle and localised crepitus with shoulder move-

Table I. Diagnostic matrix for common non-traumatic conditions of the sternoclavicular joint

Conditions*	Demographics	Clinical/laboratory findings†	Radiological features
Systemic arthritides			
Osteoarthritis	Older patients (aged over 50 yrs)	Normal	Osteoarthritic changes
Rheumatoid arthritis	Women of any age	May have positive rheumatoid factor/antinuclear antibodies	Normal or erosive changes
Seronegative spondyloarthropathies	Younger men (aged < 40 yrs)	Positive HLA-B27	Normal or erosive changes
Crystal arthropathies	Older men (aged ≥ 40 yrs)	Joint fluid birefringence Elevated ESR in acute attack	Normal or secondary osteo-arthritic changes. Soft-tissue calcification
Infective conditions			
Septic arthritis/osteomyelitis	Any age	Systemic signs of sepsis (previously healthy patients) Elevated infection markers/ purulent aspirate	Effusion and soft-tissue swelling
Chronic recurrent multifocal osteomyelitis	Children and adolescents	ESR may be mildly elevated	Lytic bone destruction Association periosteal reaction Sclerotic changes later
Joint-specific conditions			
SAPHO syndrome	Middle-aged adults	Skin changes ESR/CRP may be mildly elevated	Erosive changes Peri-articular hyperostosis Ossification of ligamentous insertions 'Bulls-head' pattern on isotope bone scanning
Condensing osteitis	Younger women (aged < 40 yrs)	Normal	Expansion of the medial clavicle and medullary canal obliteration
Friedrich's disease[1] (osteonecrosis)	Any age	Normal	Osteonecrosis of the medial clavicle and cystic changes

* SAPHO, synovitis, acne, pustolosis, hyperostosis and osteitis

† ESR, erythrocyte sedimentation rate; CRP, C-reactive protein

ment. The changes are usually bilateral, but are often asymmetrical. CT shows the characteristic signs of osteoarthritis with narrowing of the joint space, subchondral cysts and sclerosis and the formation of osteophytes (Fig. 4) which is usually most prominent on the inferior aspect of the medial clavicle.[46] Sclerosis has a low-signal intensity on all MR sequences, but subchondral cysts produce foci of hyperintense signal on T_2-weighted images.[47]

The primary treatment is conservative and includes rest, modification of behaviour, analgesia and intra-articular injection of corticosteroids. Excision arthroplasty with resection of the medial clavicle may be effective in some patients who fail to respond to conservative measures for a period of six months.[48] This should be performed with preservation of the costoclavicular ligament and repair of the anterior capsule, or with a more formal soft-tissue stabilisation to the first rib if ligament preservation and capsular repair are not feasible.[41,48]

Rheumatoid arthritis. Formation of pannus, bony erosion and degeneration of the intra-articular disc may be present in the sternoclavicular joints of up to one-third of patients with rheumatoid arthritis.[49,50] The radiological changes may be minimal, but advanced disease shows bony erosions with destruction of the joint. Medical treatment of the systemic disease process may include the use of non-steroidal anti-inflammatory drugs, steroids and disease-modifying anti-rheumatic drugs. Second-line therapy should be supervised by a rheumatologist. Resection of the medial clavicle may be undertaken in the few patients who have severe symptoms which are refractory to medical treatment.

Seronegative spondyloarthropathies. Ankylosing spondylitis, Reiter's syndrome, colitic arthritis and especially psoriatic arthritis may involve the sternoclavicular joint (Fig. 5).[49,51,52] The joint is involved in 90% of patients with severe psoriatic arthropathy, and this is clinically significant in 50%,[53] whereas corresponding signs are present in only 4% of patients with ankylosing spondylitis.[54] In the presence of a seronegative polyarthropathy, detection of HLA-B27 is usually diagnostic, and these patients should be treated initially by non-steroidal anti-inflammatory drugs. Second-line drugs such as gold and methotrexate are used rarely for resistant cases and should be administered under the supervision of a rheumatologist.

Crystal-deposition arthropathy. Aspiration of the acutely-swollen sternoclavicular joint, and examination of the fluid under polarising light microscopy, may reveal crystals and give the diagnosis of either gout (negatively birefringent) or pseudogout (positively birefringent). Both these conditions are known to affect the sternoclavicular joint[55] and treatment by oral non-steroidal anti-inflammatory agents and local injection of corticosteroids is usually effective in the acute phase. Long-term medical prophylaxis can be under-

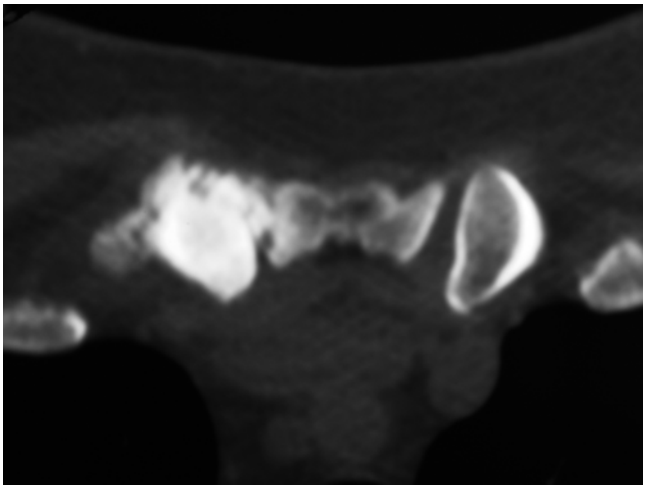

Fig. 4a

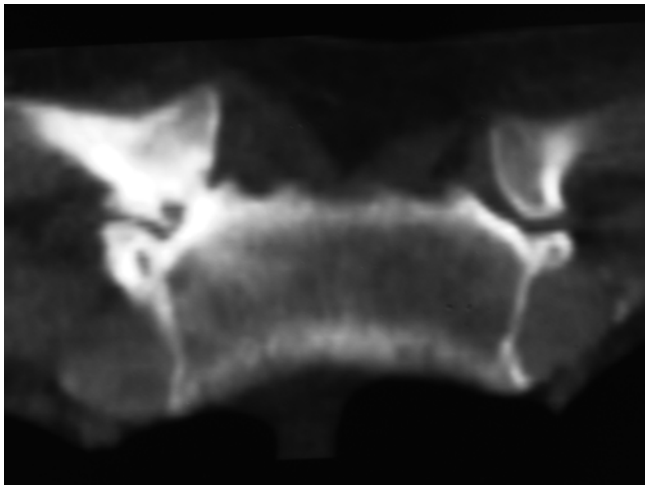

Fig. 4b

An a) axial and b) coronal CT scan of a 53-year-old man with a painless, hard swelling of the right joint showing subchondral sclerosis, pseudocysts prominent osteophytes and soft-tissue swelling of the joint.

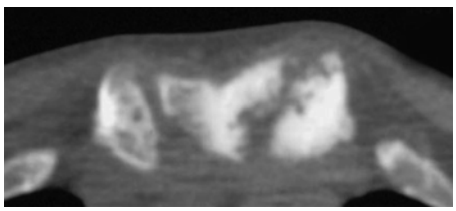

Fig. 5a

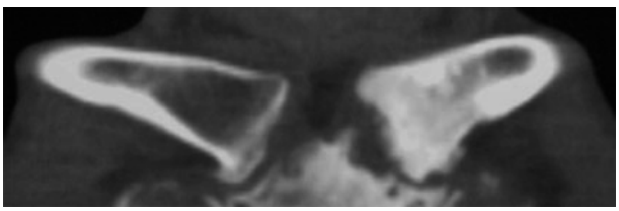

Fig. 5b

An a) axial and b) coronal CT scan of a 49-year-old woman with ankylosing spondylitis showing erosive changes and sclerosis of the left clavicle.

taken with the xanthine-oxidase inhibitor, allopurinol, or with second-line treatment such as colchicine.

Infective conditions

Septic arthritis and osteomyelitis of the medial clavicle. These should be considered as part of the differential diagnosis of a unilateral, painful, swelling of the joint or peri-articular region, particularly if the history is relatively acute. In a previously fit individual, there may be a history of a recent systemic infection elsewhere in the body,[56] and there are often associated systemic features of fever, chills and night sweats. These signs may be absent in elderly and immunocompromised individuals. Common predisposing conditions include HIV infection, steroid therapy, intravenous drug abuse, diabetes mellitus, alcoholism, renal dialysis and infected subclavian central lines.[49]

The common pathogens are *Staphylococcus aureus*,[57] coliforms,[58] *Streptococcus pyogenes* and *Neisseria gonorrhoeae*.[59] Infections from *Pseudomonas aeruginosa* and *Candida albicans* are commonly related to intravenous drug abuse.[60] In developing countries there have been reports of joint infection from *Mycobacterium tuberculosis*[61] and joint enlargment is commonly present in congenital syphilis and leprosy.

The laboratory markers of infection are grossly elevated in previously healthy patients, although these may be normal in debilitated or immunocompromised patients. All patients who present with an acute history of pain and swelling of the sternoclavicular joint should undergo an urgent ultrasound-guided aspiration of the joint, which should be examined by light microscopy before the commencement of antibiotics. If the presentation is subacute, CT may show erosions and widening of the sternoclavicular joint, periosteal reaction and, in chronic cases, sclerosis. Low-attenuation soft-tissue swelling which surrounds the joint may contain abscesses and extend into adjacent muscles or the anterior mediastinum.[62] MRI shows similar changes which have low-signal intensity on T_1-weighting and high-signal intensity on T_2-weighting, although sclerosis has low-signal intensity on all sequences. Peri-articular swelling is more prominent with

osteomyelitis of the medial clavicle and is associated with the formation of abscesses and either lytic, sclerotic or mixed changes.

If the joint aspirate is purulent, it should be aspirated to dryness and parenteral broad-spectrum antibiotics should be administered, guided by the initial Gram-stain findings. The antibiotic regimen is usually modified, based upon the culture and sensitivity results at 24 hours. Open wash-out and debridement of the joint should be performed if aspiration is incomplete, or if there is residual loculation. Osteomyelitis of the medial clavicle should also be treated initially by broad-spectrum antibiotic therapy, which may require to be continued for up to six months to achieve full eradication. Operative debridement and drainage are required if there is a failure to respond to antibiotic therapy within 24 hours of the initiation of treatment. Occasionally, in fulminant infection, when there is extensive abscess cavity formation and osteonecrosis, resection of the medial clavicle and any involved portions of adjacent ribs may be necessary. An adjuvant soft-tissue reconstructive procedure, such as advancement of pectoralis major,[63] is often later required to provide soft-tissue cover to the affected area.

Chronic recurrent multifocal osteomyelitis. This is a self-limiting condition which typically occurs in children and adolescents, and affects the metaphyses of long bones, the medial clavicle and the spine. There is an overlap between the features of chronic recurrent multifocal osteomyelitis and SAPHO syndrome. They are usually considered to be separate conditions although it has been suggested that chronic recurrent multifocal osteomyelitis is the childhood manifestation of SAPHO syndrome.[64] Patients present with localised pain and swelling, which is typically, but not invariably, symmetrical. Systemic manifestations are usually mild or absent. The ESR may be elevated, but other inflammatory markers are usually normal. Histological examination of biopsy specimens shows inflammatory changes, but cultures are sterile.

The lesions of the clavicle involve the medial end of the bone, do not cross the sternoclavicular joint, and initially appear to be aggressive, causing lytic destruction of cortical and cancellous bone and associated periosteal reaction. In the active phase, isotope bone scanning shows increased activity, while MRI indicates adjacent soft-tissue oedema, an abnormal signal in the clavicle, low-signal intensity on T_1-weighted images and hyperintense activity on T_2-weighted images.[65]

In the healing phase, MRI shows diffuse low-signal intensity, with scattered areas of residual high-signal intensity fat, on T_1- and T_2-weighted images, although short tao inversion recovery (STIR) sequences may show foci of hyperintense signal because of residual active disease.[65,66] After clinical resolution, there is usually sclerosis and expansion of the medial clavicle. Patients may suffer recurrent symptoms over many years at the original site or at additional sites.

Conditions which are relatively specific to the sternoclavicular joint

SAPHO syndrome. This syndrome is a spectrum of co-existent and chronic skin and osteoarticular conditions.[64,67-72] Many other terms have been used, including sternoclavicular hyperostosis and acne-associated spondyloarthropathy. The term 'pustule-arthro-osteitis' is also preferred by some authors[73] who feel that the acne-associated bone and joint changes differ from those seen in palmoplantar pustulosis, and that the two skin conditions should therefore be considered separately.

The theories of pathogenesis include low-grade infection and seronegative spondyloarthropathy or reactive arthropathy.[64,74] Inflammatory markers such as the level of CRP and the ESR may be mildly elevated, although the white blood count is usually normal or only slightly elevated, and bone biopsies are usually sterile. Histological examination initially shows inflammatory infiltrates, oedema and periostitis, which subsequently progresses to sclerosis and fibrosis. Aspiration may be required to exclude septic arthritis of the joint. Other diagnoses, including polymyalgia rheumatica, inflammatory muscle disease, and neck, shoulder, cardiac or pulmonary causes of diffuse neck, shoulder and anterior chest pain may need to be considered if the presentation is acute. In the more chronic relapsing form, the differential diagnosis should include chronic septic arthritis, Paget's disease of the clavicle, condensing osteitis, mediastinal fibrosis and Ewing's sarcoma.

Most patients diagnosed with SAPHO syndrome are middle-aged adults, although there is a slight female predominance. They present with localised pain, soft-tissue swelling and restricted sternoclavicular movement, which may be bilateral. The skin lesions are typically pustular and include severe forms of acne (fulminans or conglobata), palmoplantar pustulosis and psoriasis (Fig. 6). They are present in up to 60% of cases, and may precede or follow the osteo-articular manifestations, sometimes by years.

The osteo-articular symptoms may resolve after one or two attacks but more often become chronic and relapsing, or move to other sites. The sternocostoclavicular region (including the sternoclavicular joint, adjacent costosternal and costochondral junctions and the manubrium and manubriosternal joint) is involved in 60% to 90% of patients with SAPHO syndrome; two-thirds of these have bilateral involvement of the sternoclavicular joints. The spine, pelvis and long bones may also be affected, leading to localised symptoms in these areas.

The characteristic pathological musculoskeletal changes include synovitis, erosive or non-erosive arthritis, periarticular hyperostosis and ossification at the site of ligamentous insertions. Three radiological stages of osteoarticular involvement have been recognised.[71] In stage 1, the costoclavicular ligament is involved, with a soft-tissue mass and the formation of new bone on CT. In stage 2, there is arthropathy of the sternoclavicular joint and sclerosis of the adjacent clavicle, first rib, sternum and costal car-

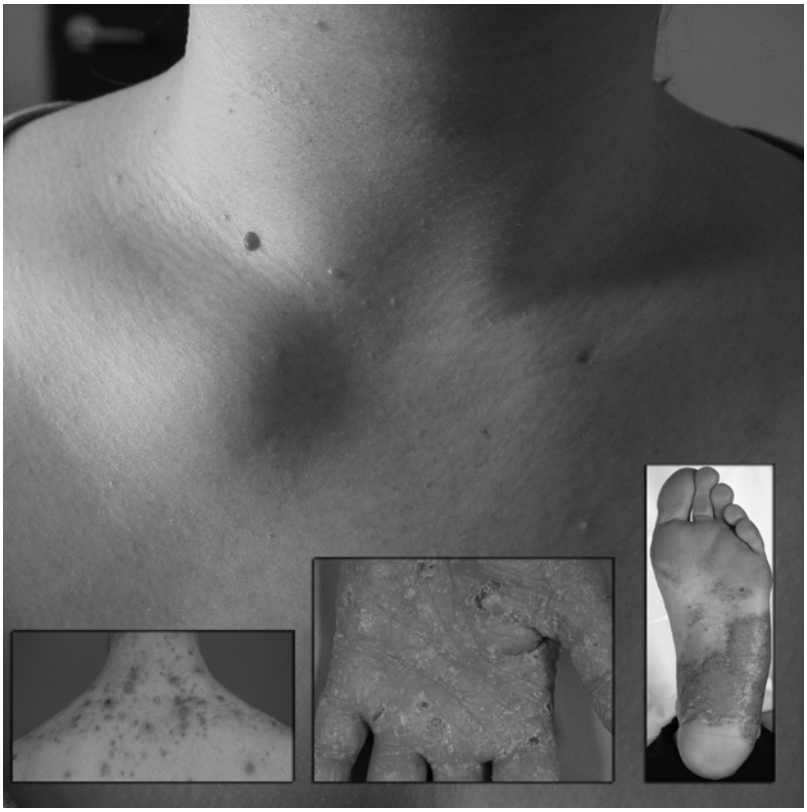

Fig. 6

Photographs showing the typical features of synovitis, acne, postulosis, hyperostosis and osteitis (SAPHO) syndrome with sternoclavicular swelling, acne fulminans and palmoplantar pustulosis (inset).

tilage. In stage 3, there is sclerosis, hyperostosis and hypertrophy of the clavicles, sternum and upper ribs and adjacent arthropathy, although occasionally a solitary bone is involved. CT clearly shows the bony changes and any associated soft-tissue mass. Water-weighted MRI sequences show bone and soft-tissue oedema but erosions and sclerosis are not well delineated. The 'bull's head' pattern of increased activity at the sternoclavicular joints and adjacent sternum on bone scintigraphy is said to be pathognomic of SAPHO (Fig. 7).[75]

Treatment is aimed at alleviating pain and inflammation, and there is evidence that the use of non-steroidal anti-inflammatory drugs and corticosteroids may be beneficial.[76] There are also some reports of successful treatment with calcitonin and pamidronate and immunosuppressive drugs such as sulphasalazine, methotrexate and cyclosporin A. The prognosis of this condition is considered to be favourable with a low rate of debility in the long term. Wide surgical resection of the ossified mass and the medial end of the clavicle and first rib has been used only for refractory cases, with mixed results.[77]

Condensing osteitis. This occurs almost exclusively in women aged between 20 and 60 years,[47] and is characterised by aseptic enlargement and osteosclerosis of the medial end of the clavicle and obliteration of its medullary cavity with little or no surrounding soft-tissue reaction. Trauma and chronic stress have been suggested as causes, and a resemblance to osteitis pubis and osteitis condensans ilii has been noted.[47,78] The patient presents with pain in the sternoclavicular joint and swelling which are exacerbated by abduction of the shoulder.[47] Conventional radiography shows sclerosis and possible expansion of the inferior aspect of the medial end of the clavicle,[79,80] which may also have an inferior spur.[47] CT delineates the sclerosis, expansion, medullary obliteration (Fig. 8) and inferior location better, whereas focally-increased activity is usually present on isotope bone scanning. MRI shows low-signal intensity on T_1- and low or intermediate signal on T_2-weighted images, although patchy areas of bright signal on T_2-weighting may be present. Bone and periosseous enhancement may be seen on post-contrast MRI.[47,81,82] The location of abnormal findings in the clavicle, the lack of involvement of the sternoclavicular joint and the normal appearance of the sternoclavicular joint, adjacent ribs and costal cartilages on all imaging investigations are critical to the diagnosis of condensing osteitis and

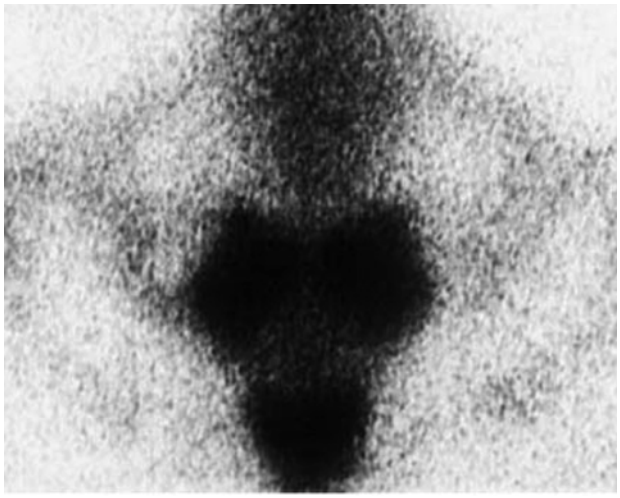

Fig. 7

Delayed-phase image of a radionucleide scan showing the characteristic 'bull's head' pattern of increased activity at the sternoclavicular joints in synovitis, together with acne, pustulosis, hyperostosis and osteitis (SAPHO) syndrome.

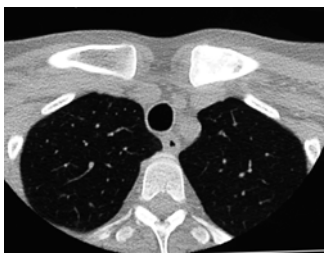

Fig. 8a

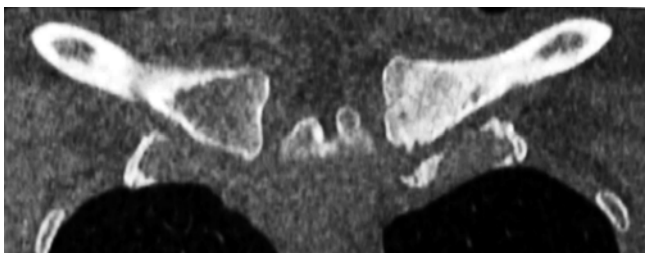

Fig. 8b

An a) axial and b) coronal CT scan of condensing osteitis of the left clavicle in a 46-year-old woman.

distinguish this condition from the osteo-articular manifestations of SAPHO syndrome. However, distinction from a primary or metastatic bone tumour may be difficult without a biopsy.

The condition is usually self-limiting and the local pain usually responds well to non-steroidal anti-inflammatory medication. Excision arthroplasty of the medial clavicle has been described in the rare cases in which pain is refractory to conservative measures.[79,80,83]

Friedrich's disease. Osteonecrosis of the medial head of the clavicle occurring without predisposing causes (Friedrich's disease) such as infection or trauma, is rare and usually unilateral.[1] Laboratory tests for inflammatory and rheumatological markers are normal. MRI may demonstrate areas of necrosis in the metaphysis, which show areas of cystic degeneration typical of osteonecrosis on biopsy. The treatment is symptomatic, with restriction of activity and administration of oral non-steroidal anti-inflammatory medication for relief from pain. The symptoms are usually self-limiting and complete resolution with remodelling of the medial clavicle typically occurs over a period of 12 to 18 months.[1]

Other conditions

The medial clavicle is a relatively common site for Paget's disease, and shows the typical clinical features of the condition.[84] Other benign lesions, including ganglion cysts[85] and synovial osteochondromatosis[86] have been reported. Ewing's sarcoma and other primary neoplasms may arise in this area,[87] while secondary squamous-cell carcinoma, adenocarcinoma and lymphoma have also been reported.[88]

Osteochondritis of the second to fourth costochondral junctions, together with hypertrophy and calcification of the costal cartilages (Tietze's syndrome[89]) presents with unilateral discomfort and swelling over the anterior chest wall, and the sternoclavicular joint may rarely also be

involved. The diagnosis is largely clinical based on the history and examination, and radiological and laboratory tests are usually normal.

Other uncommon conditions include neuropathic arthropathy, secondary to syringomyelia, which may cause destruction of the sternoclavicular joint,[90] haemophilic pseudotumours and erosive changes of the medial clavicle from either hyperparathyroidism or polymyalgia rheumatica.[91,92] Deposits of amyloid (β_2 microglobulin) may be found in the sternoclavicular joints of patients with the systemic disease.

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