Clavicle and Sternoclavicular Joint

Normal Findings

During Growth

The clavicle is a long bone that is preformed in connective tissue and contains a medullary canal. It starts to ossify before any other bone in the body. It is ossified from two primary centers, one medial and the other lateral, which appear in the fifth and sixth weeks of intrauterine life and fuse during fetal development. Enchondral (epiphyseal) ossification takes place at both the acromial and sternal ends of the bone. In the clavicle, then, we have the unusual situation of membranous diaphyseal and metaphyseal ossification coexisting with enchondral longitudinal growth in the same bone. Membranous ossification of the diaphysis contributes most to longitudinal growth during both the intrauterine and postnatal periods. Longitudinal growth at the acromial end of the clavicle is insignificant by comparison.

Sonographic measurements by Yarkoni et al. (1985) showed a linear correlation between the length of the fetal clavicle and gestational age. These studies indicate that, as a general rule of thumb, the gestational age of a fetus in weeks is approximately equal to the length of the clavicle in millimeters.

The secondary epiphyseal ossification center at the medial end of the clavicle can be useful for age determination, especially between late adolescence and the third decade of life.

In their retrospective CT study of 380 subjects under 30 years of age, Kreitner et al. (1998) discovered that the medial ossification center of the clavicle appears between 11 and 22 years of age. They observed partial fusion between 16 and 24 years of age (Fig. 3.74) and complete fusion no earlier than 20 years of age; fusion occurred in 100% of cases by age 27. On comparing their data with the previous literature, the authors found no ethnic differences in the onset of ossification, the duration of partial fusion, or the timing of complete fusion. Despite the relatively long time needed for bony maturation of the medial epiphysis, the authors claim that the ossification pattern of the clavicle can be used forensically to determine whether an individual is in his or her late teens or early twenties (e.g., for referral to a penal institution for juveniles or adults). The same criteria can also be used for the age determination of human torsos.

As in adults, the juvenile clavicle is anteriorly convex in its medial two-thirds and posteriorly convex in its lateral third. The individual segments of the clavicle can show varying radiographic densities, depending on the projection (Fig. 3.72).

The sternal end of the clavicle undergoes distinct morphological changes with aging:

- In the first decade of life, the sternal end is shaped somewhat like a mushroom with smooth or irregular contours (Fig. 3.73a).
- In the second decade of life, it becomes more cup-shaped and often has irregular borders (Fig. 3.73b).

In Adulthood

After about 25 years of age, the medial end of the clavicle is radiologically shaped somewhat like a pestle, often bearing a central notch or groove in its articular surface (Figs. 3.75b, 3.76).

In adults as in children, the three-dimensional curvature of the clavicle should be considered when the bone is evaluated. This is necessary to avoid the misinterpretation of deformities, especially in atypical projections (e.g., an elevated scapula in the AP projection, etc.).

An approximately 4-mm-wide band of soft-tissue density is normally seen parallel to the superior border of the clavicle. It represents an orthograde projection of the skin that overlies the clavicle.

Fig. 3.72a, b The pediatric clavicle.
a The increased density at the center of the clavicle in an infant is a projection-related effect.
b Normal curvature of the pediatric clavicle.

Fig. 3.75a, b The adult clavicle.
a The increased density at the center of the clavicle is a projection-related effect.
b The increased density at the center of the clavicle is a projection-related effect.
**Fig. 3.73a, b** Morphological changes in the medial clavicle during growth.

*a* The medial end of the clavicle is mushroom-shaped during the first decade of life (9-year-old boy).

*b* The medial end of the clavicle is cup-shaped in the second decade of life (12-year-old girl).

**Fig. 3.74a–d** Ossification of the medial clavicular epiphysis.

*a* Radiograph in a 21-year-old man shows partial ossification of the medial epiphyseal center of the clavicle.

*b–d* CT scans from a convicted criminal of unknown age. The scans were taken to determine whether the individual should be placed in a juvenile or adult penal facility. The partially fused medial clavicular epiphysis proves that the patient must be between 16 and 26 years of age. If the epiphysis were completely fused, he would have to be over 20 years of age.

**Fig. 3.75a, b** Anatomy of the clavicle.

*a* Superior aspect.

*b* Inferior aspect.

Note the pronounced S-shaped curvature of the clavicle and the configuration of the medial articular end.
On atypical projections, superimposed portions of the scapula can lead to strange or confusing findings (Fig. 3.77).

A small defect is occasionally seen in the superior border of the middle third of the clavicle. It represents the foramen of a canal that transmits the medial fascicle of the supraclavicular nerve (Fig. 3.78). According to anatomists, this nerve canal is present in 2–6% of the population. Duplication of the canal was described by Pahl (1955) (Fig. 3.79).

Besides neural foramina, nutrient canals also occur in the clavicle and are most conspicuous at the junction of its middle and lateral thirds (Figs. 3.75b, 3.89a, b).

The site where the coracoclavicular ligament attaches to the clavicle may exhibit pits, cortical irregularities, or a normal conoid tubercle (called also the coracoid tubercle; Fig. 3.75).

The site where the costoclavicular ligament attaches to the inferior medial border of the clavicle (Fig. 3.81) normally bears a notch (Fig. 3.75b) that can be quite conspicuous in some individuals, especially those who subject the shoulder to heavy loads (Fig. 3.80a-b).

Fig. 3.76 The medial end of the clavicle is pestle-shaped or stamp-shaped after the middle of the third decade (27-year-old man).

Fig. 3.77 Apparent "exostosis" at the superior border of the clavicle: a projection-related artifact.

Fig. 3.78a, b Canal for the medial fascicle of the supraclavicular nerve (arrow).

Fig. 3.79 Duplication of the neural foramen for the supraclavicular nerve fascicle (observation by Pahl).

Fig. 3.80a-c Ligament attachments. 

a Relatively deep notch at the attachment the costoclavicular ligament (arrow).

b Appearance in an anatomic specimen.

c "Roughness" at the attachment of the sternohyoid and sternocleidomastoid muscles.
The differential diagnosis of this feature should always include a rarefying fibro-ostosis or fibro-ostitis (i.e., an enthesisopathy). The cortex also appears rough or spongy in the superomedial portion of the clavicle that gives attachment to the sternohyoid and sternocleidomastoid muscles (Fig. 3.80c).

The medial or sternal end of the clavicle cannot be considered without noting its importance as a component of the sterno-clavicular joint (Fig. 3.81). This joint is the only true articulation that exists between the trunk and the shoulder girdle. The sternal side bears an extremely shallow joint cavity that is only partially congruent with the slightly convex but sometimes concave sternal end of the clavicle. As a result, the joint is inherently unstable. An interposed articular disk compensates for the lack of stable congruence. It also serves as a shock absorber for forces that are transmitted through the clavicle to the sternum. The articular disk undergoes regressive changes as early as 20–30 years of age, and after age 50 these “normal” changes can be extremely pronounced without causing clinical symptoms. The possible clinical significance of the articular disk is discussed on p. 310.

The capsule of the sternoclavicular joint is relatively broad and is lined by a synovial membrane. The joint is stabilized chiefly by the anterior and posterior sternoclavicular ligament, which is basically a thickening of the fibrous capsule. Other known thickenings of the capsule anchor the medial end of the clavicle to the sternum, and the extra-articular costoclavicular ligament helps stabilize the joint by binding the medial clavicular metaphysis to the first rib.

Normal CT-measured values for the width of the sternoclavicular joint space are listed in Table 3.2.

An intra-articular vacuum phenomenon (Fig. 3.95) can be observed as early as 20 years of age but usually is not seen until age 40 (in approximately 8% of healthy subjects) (Hatfield et al. 1984).

Table 3.2 Normal CT dimensions of the sternal region in millimeters (soft-tissue window, level 30 HU, window 500 HU, after Hatfield et al.). The values are approximately 10% higher than measurements using a bone window

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Overall mean</th>
<th>Male mean¹</th>
<th>Female mean¹</th>
<th>Minimum</th>
<th>Maximum</th>
</tr>
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<tbody>
<tr>
<td>Sternal size:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• AP</td>
<td>354</td>
<td>21.0</td>
<td>21.8</td>
<td>19.8</td>
<td>5</td>
<td>38</td>
</tr>
<tr>
<td>• Transverse</td>
<td>354</td>
<td>59.0</td>
<td>62.0</td>
<td>55.1</td>
<td>8</td>
<td>120</td>
</tr>
<tr>
<td>• Cranio-caudal</td>
<td>354</td>
<td>159.0</td>
<td>166.0</td>
<td>149.0</td>
<td>40</td>
<td>230</td>
</tr>
<tr>
<td>Cortical thickness:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Manubrium sterni</td>
<td>311</td>
<td>9.5</td>
<td>9.7</td>
<td>9.1</td>
<td>4</td>
<td>21</td>
</tr>
<tr>
<td>• Body of sternum</td>
<td>306</td>
<td>6.1</td>
<td>6.6</td>
<td>5.6</td>
<td>2</td>
<td>13</td>
</tr>
<tr>
<td>• Sternoclavicular joint distance</td>
<td>335</td>
<td>6.4</td>
<td>6.9</td>
<td>5.8</td>
<td>1</td>
<td>14</td>
</tr>
</tbody>
</table>

¹ Average age of subjects: males 45.1 years, females 44.7 years. Youngest subject 9 days, oldest subject 94 years
Pathological Finding?

**Normal Variant or Anomaly?**

The **medial epiphysis of the clavicle**, like most epiphyses in the shoulder girdle region, is subject to pronounced morphological variations, ranging in appearance from small nuclei in fossa-like depressions (Fig. 3.82a) to disk-shaped structures. The persistence of these centers has also been observed (Fig. 3.82b).

In extreme cases the medial end of the clavicle may show a forklake or fish-mouth configuration, even if there is no persistent epiphyseal center (Ravelli 1955).

Another variant is the **costoclavicular joint**, which may occur in place of the normal ligamentous attachments between the clavicle and first rib (Redlund-Johnell 1986).

**Complete absence** of the clavicle is classified as a true dysplasia. A large defect in the acromial end of the clavicle, combined with malformations of the skull and hands, can occur as part of a cleidocranial dysplasia. **Congenital unilateral hypoplasia** of the clavicle with pseudarthrosis formation between the hypoplastic segments is another type of dysplasia that probably has an autosomal dominant mode of inheritance (March 1968, Höcht et al. 1979).

Another anomaly, the **lateral clavicle hook**, consists of a hooklike accentuation of the lateral curvature of the bone. This type of anomaly may be associated with Holt–Oram syndrome, osteodysplastia, or trisomy 18 (Igual and Giedion 1979). These conditions cannot be covered here in detail, but the principal anomalies are summarized in Tables 3.3 and 3.4 (after Reeder 1993).

A final possible normal variant is **duplication of the clavicle** (Golthamer 1957, Twigg 1981).

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Table 3.3 Etiological spectrum of thin or hypoplastic clavicle

<table>
<thead>
<tr>
<th>Common</th>
<th>Rare</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cleidocranial dysplasia</td>
<td>Brachial plexus paralysis</td>
</tr>
<tr>
<td>Holt–Oram syndrome</td>
<td>(unilateral) caused by</td>
</tr>
<tr>
<td>Osteodysplasia (Melnick–Needles</td>
<td>birth trauma</td>
</tr>
<tr>
<td>syndrome)</td>
<td></td>
</tr>
<tr>
<td>Pyknody sostosis</td>
<td>CHILD1 syndrome</td>
</tr>
<tr>
<td>Progeria (thin clavicle)</td>
<td>Cockayne syndrome</td>
</tr>
<tr>
<td></td>
<td>(thin clavicle)</td>
</tr>
<tr>
<td></td>
<td>Congenital clavicular</td>
</tr>
<tr>
<td></td>
<td>pseudarthrosis</td>
</tr>
<tr>
<td></td>
<td>Coffin–Siris syndrome</td>
</tr>
<tr>
<td></td>
<td>Goltz–Gorlin syndrome</td>
</tr>
<tr>
<td></td>
<td>(focal dermal hypoplasia)</td>
</tr>
<tr>
<td></td>
<td>Fucosidosis</td>
</tr>
<tr>
<td></td>
<td>Larsen syndrome</td>
</tr>
<tr>
<td></td>
<td>(thin clavicle)</td>
</tr>
<tr>
<td></td>
<td>Scapuloilic dysostosis</td>
</tr>
<tr>
<td></td>
<td>Spondyloepiphyseal dysplasia</td>
</tr>
<tr>
<td></td>
<td>(delayed ossification)</td>
</tr>
<tr>
<td></td>
<td>Trisomy 13 syndrome</td>
</tr>
<tr>
<td></td>
<td>(thin clavicle)</td>
</tr>
<tr>
<td></td>
<td>Trisomy 18 syndrome</td>
</tr>
<tr>
<td></td>
<td>(thin clavicle)</td>
</tr>
<tr>
<td></td>
<td>Turner syndrome</td>
</tr>
<tr>
<td></td>
<td>(thin lateral clavicle)</td>
</tr>
</tbody>
</table>

1 CHILD Congenital hemidysplasia, ichthyosiform nevi, limb defect

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Table 3.4 Hypoplastic stocky (“handlebar-like”) clavicle

<table>
<thead>
<tr>
<th>Common</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastrophic dysplasia</td>
</tr>
<tr>
<td>Holt–Oram syndrome</td>
</tr>
<tr>
<td>Thrombocytopenia-absent radius syndrome</td>
</tr>
<tr>
<td>Trisomy 18 syndrome</td>
</tr>
<tr>
<td>Normal variant</td>
</tr>
</tbody>
</table>
Fracture, Subluxation, or Dislocation?

Fractures?

Clavicular fractures can occur as a result of birth trauma (Enzler 1950, Köster 1957). Initial callus becomes visible on radiographs in just 8–9 days. Clavicular fractures are not uncommon in children, especially before age 10, and comprise perhaps 50% of all injuries in the shoulder girdle region. The middle of the clavicle is a site of predilection for fractures. Often these pediatric fractures are very difficult to detect radiographically, especially since the majority are of the greenstick type.

As explained on p. 288, a superior tear in the periosteal sleeve of the lateral clavicle can allow the end of the clavicle to displace upward, leaving the cartilaginous portions in the acromioclavicular joint. The ligaments and capsule of the acromioclavicular joint are not disrupted. Basically this injury involves an epiphyseal separation corresponding to a Salter–Harris type I epiphyseal injury. A sternoclavicular epiphyseal separation (epiphysiolysis) can occur at the medial end of the clavicle (Lemire and Rosmann 1984).

In adults, 80% of all clavicular fractures involve the middle third of the clavicle. Fifteen percent involve the lateral third and may or may not be associated with a tear of the coracoclavicular ligament. The fracture line may extend into the acromioclavicular joint.

Only about 5% of clavicular fractures involve the sternal end, where they are very difficult to detect radiographically. Clavicular fractures mainly require differentiation from unusually prominent nutrient canals, nerve canals, and simple projection-related effects (Fig. 3.83).

Stress fractures of the clavicle most commonly occur after a radical neck dissection.

Subluxations and Dislocations

Traumatic dislocations of the sternoclavicular joint account for approximately 1% of all dislocations (Nettles and Linscheid 1968). The joint typically dislocates anteriorly and superiorly. Posterior dislocations are very rare (Fig. 3.84). They may be associated with severe clinical complications caused by pressure from the dislocated clavicle on the major vessels, trachea, esophagus, etc. (Cope and Riddervold 1988). Sternoclavicular dislocations are difficult to document with plain films, and we feel that a CT examination should be performed routinely whenever this injury is suspected.

Anterior and superior subluxation of the medial end of the clavicle due to regressive changes is a difficult diagnostic problem. Most patients are women 50 years of age or older who suddenly notice a “lump” over the medial end of the clavicle. The subluxation may indeed develop suddenly, or it may actually be present for some time before the patient becomes aware of it.

The clinical hallmark of these cases is a marked prominence of the clavicular head (Fig. 3.85a), which often feels somewhat springy to the touch. It may or may not be painful. Conventional radiographs are usually unrewarding due to the difficulty of obtaining a clear projection. A bone scan may show slightly increased tracer uptake in the region of the manubrium-clavicular joint. The most conclusive study is CT, which can document the anterior and superior displacement of the clavicular head (Fig. 3.85b–d). In many cases CT will also show definite regressive changes at the ends of the joint with subchondral sclerosis, small subchondral cysts, and marginal osteophytes. Etiologically, we feel that a painless subluxation is based on an age-related regressive process similar to the marginal osteophytes that develop along the acromioclavicular joint (see p. 290). The underlying cause may well be a constitutional laxity of the capsule and ligaments. We often confront this problem in cases that have been referred to us for consultation. The situation is different in patients who present clinically with pain and swelling. This implies a true pathological process in the sense of an active osteoarthritis with subluxation, similar to the changes that can affect joints elsewhere in the body.

Aumann and Brüning (1980) shed new light on this problem with their operative findings in six patients. They noted significant morphological disk changes (flattening and thinning, fragmentation, swelling) with otherwise normal-appearing bone and cartilage in the sternoclavicular joint. Clinically, the authors described transient joint swelling, severe pain, and crepitus during joint movements. In some cases the pain radiated throughout the shoulder girdle and to the back of the neck. The authors attributed the symptoms to the severe disk changes and believed that edema of the capsule and ligaments was responsible for the swelling that is seen in cases of degenerative subluxation.

Fig. 3.83 Mach effect produced by the superimposed clavicle and acromion.

Fig. 3.84 Posterior dislocation of the clavicle at the sternoclavicular joint. The arrow indicates the detached medial epiphysis (from Cope and Riddervold 1988).
Generally, regressive subluxation with prominence of the clavicular head as well as the disk abnormalities described by Aumann and Brüning (1980) require differentiation from aseptic necrosis (Friedrich disease) and from an early stage of unilateral sternocostoclavicular hyperostosis. If there is demonstrable swelling of the sternoclavicular joint but no redness, it is unlikely that arthritis is present.

**Necrosis?**

Densities at the inferior medial end of the clavicle may be purely regressive in nature, but in symptomatic patients they may signify aseptic necrosis at that location (Friedrich disease). This condition presents clinically with a soft to firm swelling over the sternoclavicular joint. The earliest radiographic change is sclerosis involving the inferior medial end of the clavicle (stage I, Fig. 3.86a). With further progression, radiographs demonstrate fragmentation or demarcation of the necrotic end (stage II, Fig. 3.86b).

The necrotic process culminates in stage III, which displays the features of manubrioclavicular osteoarthritis (Heinemeyer et al. 1979, Lingg and Heinemeier 1981). Differentiation is mainly required from the condition known as osteitis condensans of the clavicle (Brower et al. 1974) and from early sternoclavicular hyperostosis (see below). Some cases of “aseptic necrosis” (Friedrich disease) that we have observed appeared to represent an initial stage of sternocostoclavicular hyperostosis, which became more obvious as the years progressed (see below). These patients developed dermatological changes such as pustulosis palmoplantaris or classic psoriasis, or the changes were already present when the patients were first seen.

Interestingly, Friedrich disease is observed predominantly in females.

---

Fig. 3.85a–d  Nonpainful subluxation of the right sternoclavicular joint. While dressing, the patient suddenly noticed a prominence of the medial end of the right clavicle (star). CT shows an obvious anterior-superior subluxation of the clavicular head (b). New bone formation is apparent in the area where the capsule and ligaments attach to the clavicular head on the right side. Compare this finding with Fig. 3.94, and contrast with the features of aseptic necrosis in Fig. 3.86.
Inflammation?

Generally there should be little difficulty in distinguishing inflammatory diseases of the clavicle from normal variants. We include a section on inflammatory conditions because, at most, there may be some confusion with initial findings.

Infantile Cortical Hyperostosis

After the mandible, the clavicle is the second most frequent site of involvement by infantile cortical hyperostosis (Caffey disease). Most cases are bilateral.

Chronic Recurring Multifocal Osteomyelitis

Another inflammatory disorder with a predilection for the clavicle is chronic recurrent multifocal osteomyelitis. Initially, this disease usually presents radiographically as a subacute to chronic osteomyelitis without sequestrum formation (Figs. 3.87, 3.88).

Generally the diagnosis is established by the radionuclide detection of additional sites of involvement, especially in the metaphyses of the long bones of the lower extremity. Not infrequently, the condition is associated with pustulosis palmo-plantaris (Freyschmidt and Freyschmidt 1996).
Fig. 3.87a–c  Chronic recurrent multifocal osteomyelitis. The patient, a 13-year-old boy, presented clinically with massive prominence of the left clavicle but no inflammatory redness of the skin. Note the grotesque enlargement and increased density of the left clavicle and the concomitant involvement of the distal radius (c). The lucency on the radial side of the metaphyseal-diaphyseal junction is a postsurgical defect.

Fig. 3.88  Chronic recurrent multifocal osteomyelitis. The process started in the right clavicle. There is a relative paucity of destructive changes and new bone formation (arrow). Metachronous changes in other skeletal regions appeared during subsequent years. Differentiation is required from aseptic necrosis of the medial end of the clavicle (Fig. 3.86).

Osteitis condensans of the Clavicle
Osteitis condensans of the clavicle is a nonbacterial inflammatory disease (Brower et al. 1974, Franquet et al. 1985). Usually there is isolated involvement of the clavicular head, which has a homogeneous roentgen appearance with no destructive changes or obvious periosteal reactions (Fig. 3.89a). We doubt whether this entity actually exists as such and suggest that it may represent aseptic necrosis without fragmentation or incipient sternocostoclavicular hyperostosis.

Paget Disease
In polyostotic cases of Paget disease (osteitis deformans), the clavicle may be involved in addition to other bones. The classic features are bony enlargement and a coarsened trabecular pattern with loss of clear delineation between the cortex and medullary canal (Fig. 3.89c,d).

Bacterial Diseases
Bacterial osteomyelitis of the clavicle (usually caused by Staphylococcus aureus) is rare but occurs with some frequency in immunocompromised patients (diabetes mellitus, chronic hemodialysis, drug abuse, etc.). Hunter et al. (1983) described the development of clavicular osteomyelitis following the insertion of a Swan–Ganz catheter.

In congenital syphilis, the clavicle is a site of predilection for osseous involvement (Fig. 3.90).

Bacterial arthritis of the manubrio-clavicular joint is also relatively common in immunocompromised patients. The pattern of involvement with destruction of the bone ends, accompanying periosteal reaction, etc. is the same as in other forms of bacterial arthritis.
Fig. 3.89a–d So-called osteitis condensans of the clavicle and Paget disease.
a, b So-called osteitis condensans of the clavicle in a 17-year-old male. Follow-up (b) for one year showed increasing sclerosis of the medial end of the clavicle. The case presented clinically with swelling and little pain. The absence of other skeletal manifestations, even years later, distinguishes this condition from chronic recurrent multifocal osteomyelitis (CRMO). Note the nutrient canal in the lower mid-clavicle (arrow).

c, d Involvement of the clavicle by polyostotic Paget disease in a 61-year-old man. Note the coarsened trabecular pattern throughout the left clavicle and in the coracoid process (c). The humeral head also exhibits structural change. The radiouclide scan (d) shows the most intense uptake in the left clavicle and coracoid process, apparently because the disease is still in a florid stage at those locations. Additionally, giant-cell tumorlike lesions were found in the humeral head and proximal diaphyseal-metaphyseal junction of the left humerus. This case is fully documented in Freyschmidt (1997, Fig. 14.42c–n).

Fig. 3.90 Stage III syphilitic osteitis of the right clavicle in a 2-month-old infant with congenital syphilis. The large tubular bones were also involved. Note how destructive lesions in the medial third of the clavicle are combined with extensive periosteal new bone formation, which apparently preceded the destructive changes (stage II).
**Sternocostoclavicular Hypertostosis**

Sternocostoclavicular hypertostosis is an inflammatory disease that we and other authors have observed with increasing frequency in the sternoclavicular region during recent years (Sonozaki et al. 1981, Kasperczyk and Freyschmidt 1993, Dihlmann 1993, Freyschmidt and Freyschmidt 1996, Freyschmidt and Sternberg 1998, Schilling and Kessler 1998). The swelling and redness are based on an inflammatory destructive process that involves the sternum, the medial ends of the clavicles, and the ligament and tendon attachments, especially between the ribs and clavicles (Figs. 3.91, 3.123).

This abacterial process is always associated with concomitant reactive-reparative and ankylosing bone formation in the affected region, leading to a progressive limita-

Fig. 3.91a–h  Sternocostoclavicular hypertostosis.
a Advanced changes with staghorn-like ossification, especially of the manubriocostal attachments. Note the destructive changes in the manubrioclavicular joints. There was also inflammatory widening of the manubriosternal synchondrosis (not demonstrated here: see Fig. 3.123 e, h).

b Predominantly destructive form of sternocostoclavicular hypertostosis with complete destruction of the sternoclavicular joints accompanied by bone proliferation.

c Series of CT scans in sternocostoclavicular hypertostosis (c). The right clavicle and manubrioclavicular joint are predominantly affected in this patient. The whole-body radionuclide scan (f) shows predominant involvement of the right medial clavicle and manubrium. It also demonstrates foci in both lower thoracic vertebrae, in both lower lumbar vertebrae, and in the sacrum consistent with a nonspecific spondylitis/spondylodiscitis (g). Note the typical sites of syndesmophyte formation on the affected vertebral bodies, indicating that this disease should be classified as a seronegative spondylarthritis.
Fig. 3.91d, e  These scans show the typical bull’s head pattern of radionuclide uptake in the sternocostoclavicular region. Scan d goes with the radiograph in a, and scan e goes with the radiograph in b.

h  Clinical appearance of sternocostoclavicular hypertostosis in the patient imaged in a and d: marked redness and swelling in the sternocostoclavicular region, accompanied by a conspicuous inflammatory prominence over the manubriosternal synchondrosis.
associated spondylarthritic changes, which may even progress to the classic picture of ankylosing spondylitis. In contrast to classic forms of spondylarthritis, this form involves the shoulder girdle more than the sacroiliac region, and the shoulder girdle is the primary site of involvement by inflammatory destructive and proliferative changes. Unusual tumorlike lesions occur in the tubular bones (Kasperczyk and Freyschmidt 1993). A detailed description of the features of pustulotic arthro-osteitis can be found in Freyschmidt and Freyschmidt (1996, 1998) and other sources. Most patients present at a relatively late stage, because usually the diagnosis is not routinely considered. Only CT and radionuclide scans can reliably detect the disease in its early stages. Whenever initial destructive and proliferative changes are found in the sternocostoclavicular region on clinical and radiological examination, a radionuclide scan should be obtained to check for the typical “bull-head” pattern of intense uptake in the sternocostoclavicular region (Freyschmidt and Sternberg 1998). The advantage of the radionuclide scan is that it also permits the early detection of other inflammatory skeletal changes.

**Tumor?**

Basically all types of primary and secondary bone tumors may involve the clavicle. Early osteosclerotic and even osteolytic changes are often very difficult to detect on plain films. The following figures show typical examples of neoplastic changes in the clavicle:

- Figure 3.92: Langerhans-cell histiocytosis in a small child
- Figure 3.93a: Fibrous dysplasia
- Figure 3.93b: Bone metastasis

**Pseudotumors of the clavicle** can develop as a sequel to radical neck dissection (Fini-Storchi et al. 1985). Ultimately they are the result of a stress fracture with hemorrhagic areas and reactive changes.

![Fig. 3.92a, b](image)

**Fig. 3.92a, b** Medial destruction of the left clavicle in a 3-year-old child with Langerhans-cell histiocytosis (eosinophilic granuloma). The cufflike periosteal reaction bridging the gap caused by bone destruction is a typical feature of Langerhans-cell histiocytosis. The CT scan (b) shows bony debris (sequestrum) within the periosteal cuff. The changes resolved spontaneously during the following year.

![Fig. 3.93a, b](image)

**Fig. 3.93a, b** Fibrous dysplasia and metastatic bone destruction.

**a** Fibrous dysplasia appears as a lucent (cystlike) area in the middle third of the clavicle with slight expansion of the bone. The lesion is sharply margined. The medial portion shows a typical ground-glass appearance, distinguishing it from a true bone cyst.

**b** Metastatic destruction of the acromial segment of the clavicle in a patient with renal cell carcinoma.
Degenerative arthritis of the sternoclavicular joint is not an unusual finding in radiographic examinations (Figs. 3.94, 3.95). Lately we have seen it quite often on digital thoracic images (owing to the large dynamic range). The patients denied having symptoms referable to the degenerative changes, so we interpreted the condition as an incidental finding or age-associated variant. Painful degenerative changes are uncommon (Fig. 3.94).

The problem of regressive disk changes in the sternoclavicular joint and the features of subluxation combined with osteoarthritis are covered under Fractures, Subluxations and Dislocations.

Calcifications and ossifications of the ligaments attached to the clavicle are illustrated in Figs. 3.26 and 3.50.

Fig. 3.94 Painful degenerative arthritis with joint space narrowing and sclerosis of the articular surface in a 63-year-old man.

Fig. 3.95a–d Classic sternoclavicular osteoarthritis in a 61-year-old woman who had done heavy physical labor all her life. She presented clinically with prominence of the right medial clavicle. She had occasional pain and cracking on joint motion but no swelling. The manubrium may be congenitally asymmetrical or "tilted," as its transverse axis is not horizontal. The CT scans show marked productive new bone formation on the superiorly subluxated medial end of the clavicle. The joint space (c, d) shows a definite vacuum phenomenon along with small subchondral degenerative cysts that confirm the presence of osteoarthritis.

References

Fischer, E.: Persistierende Klavikulaapophyse. Fortschr. Röntgenstr. 86 (1957) 532