MYOSITIS OSSIFICANS AS A COMPLICATION OF TETANUS

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A forty-three-year-old Indian stepped on a sharp piece of wood and suffered a small puncture wound on the sole of the right foot. A doctor gave him daily injections of penicillin for three days, but no antitetanus serum. Ten days after the injury he was admitted to hospital complaining of pains in his legs, and trismus was observed. He was immediately given an intravenous injection of 100,000 units of antitetanus serum and 600,000 units of procaine penicillin. Later the same day he complained of pain in his back, and this was shortly followed by tatic nerve spasm in his back and legs. Tetanus was diagnosed. The penicillin was repeated daily for ten days, but no additional serum was given. An intravenous glucose saline drip was started in the right cephalic vein, and paraldehyde was given intramuscularly as required, on an average 8 cubic centimetres five times daily. On the third day the spasms became more frequent and severe, and half a gramme of thiopentone was added to each bottle of saline. After another four days the patient had recovered sufficiently to allow the thiopentone to be discontinued, and the frequency of the paraldehyde injections was reduced. From now onwards the patient made a steady recovery.

On the tenth day after the onset of the spasms the patient complained that both knees and the right elbow were stiff. The stiffness of the right elbow was attributed to a rather painful arm after the intravenous drip, which had been continued for seven days. No thrombosis was noticed. The patient was encouraged to move the affected joints as much as possible, but the stiffness increased, and four days later he was referred to the physiotherapy department for active and passive movements of the knees and right elbow. Examination revealed considerable thickening about the left knee, which felt bony hard, and there was rather less about the right knee. Neither active nor passive movement of the knees was possible. There was marked limitation of movement in both hips, and in the right elbow. Radiographs taken three weeks after the onset of the spasms revealed extensive soft flocculent calcareous infiltration in the soft tissues behind the lower end of the left femur, extending downwards some two inches below the knee joint; and similar, but less extensive, changes behind the right knee. The calcareous infiltration seemed to be mainly in the hamstring muscles or gastrocnemii, and in the medial and lateral ligaments of the knees, though there were similar changes in the medial vasti and adductors on both sides. There was faint flocculent calcareous infiltration in the periarticular tissues of both hips—again a little more pronounced on the left side—and in front of the right elbow in the situation of the lower part of the brachialis. In view of these surprising findings, characteristic of myositis ossificans, the serum levels of calcium and phosphorus were checked and found to be 11 milligrams per cent and 3.1 milligrams per cent respectively. The alkaline phosphatase was 7 units (King-Armstrong), and the Kahn test was negative.

Progress—Complete rest in bed was ordered, with posterior plaster splints to both knees. This treatment was abandoned after two months because no improvement had been achieved, and physiotherapy was instituted. This was discontinued after two months. A year after the onset of tetanus the patient was well, apart from complete immobility of the right elbow, left knee and left hip. There was a limited degree of movement in the right knee (180–165
Fig. 1
Early calcification visible in capsular structures of both hips three weeks after onset of tetanus.

Fig. 2
Radiographs of the knees taken at the same time as the radiograph in Figure 1, showing distribution of ossification in soft tissues.
FIG. 3
Radiograph of pelvis one year later. Broad bands of ossification ankylosing both hips.

FIG. 4
Knees one year later, showing consolidation into dense bony bands.
degrees) and in the right hip, where it was estimated as 10 per cent of normal. Radiographs showed thick dense bony bands behind both knees, bridging the joints, and similar changes in front of the hip joints and right elbow joint. There appeared to be ossification in the medial ligaments of both knees, more pronounced on the left (Figs. 1 to 5). It was proposed to try to excise the bony bridge from behind one knee, but the patient decided to return home to India and has not been seen again.

ETIOLOGY

We do not know of any previous description of myositis ossificans and ossification in the ligaments and capsules of joints after tetanus. Calcification consists in a slow passive deposition of calcium salts in tissues that are either necrotic or of low metabolic activity, and presents as an amorphous shadow in a radiograph. Calcinosi seen in scleroderma and with Raynaud's phenomenon, and the calcification of tendons around the shoulder are good examples. The early changes seen here, although presenting an amorphous picture in the radiograph, do not comprise calcification any more than do the similar appearances in the early stages of active callus formation in fractures. The indefinite opacity soon matures to one of bone formation, and clearly represents a process of ossification in the involved structures, whether muscles, ligaments or other mesodermal tissues.

Trauma usually precedes such changes; they may occasionally be associated with a fracture, but more often with a dislocation or subluxation, especially of the knee, elbow or hip. The condition is uncommon in open injuries.

Changes which, clinically and radiologically, are indistinguishable from post-traumatic myositis ossificans have been reported with lesions of the central nervous system—more commonly in those affecting the motor system such as traumatic paraplegia, spastic diplegia and anterior poliomyelitis, than in lesions of the sensory system such as tabes and syringomyelia. The condition does not seem to occur with lesions of peripheral nerves, and we have not

FIG. 5
Right elbow. The changes in the left elbow are almost identical.
encountered it in a fairly large experience of leprosy. It is best described as self-limiting metaplastic ossification in mesodermal tissue, but the term myositis ossificans is universally accepted if the muscles are involved. This local traumatic myositis ossificans must not be confused with myositis ossificans progressiva, which usually, and for no known reason, starts in childhood.

Several theories have been advanced to explain ossification in soft tissues. Déjerine, Ceillier and Déjerine (1919), who were the first to describe ossification in muscles in traumatic paraplegia, ascribed it to the potential of primitive connective tissues under certain conditions to develop into fibrous tissue, cartilage or bone; and suggested that local tissue oedema, with an alteration in the chemical composition of tissue fluids, was an essential feature. Miller and O'Neill (1949) suggested that ossification in spastic lesions was a form of metaplasia brought on by continuous traction on connective tissues, ligaments and tendon; they thought that calcium might be derived from adjacent bones, which initially show rarefaction.

Brailsford (1941) suggested that a lesion of the central nervous system might promote traumatic stripping of periosteum at the site of muscle attachment, or metaplastic ossification. He quoted extensive ossification in the adductor muscles after tenotomy in a youth with spastic diplegia. Watson-Jones (1943) stated that traumatic myositis ossificans was avoidable, and that new bone formation occurred only within the limits of displaced periosteum—being nothing more than ossification of a subperiosteal haematoma. He denied that ossification was caused by the escape of osteoblasts into muscle, and stated that, if displacement of periosteum and extensive subperiosteal haematoma were prevented, ossification would not be encountered. But the pattern of bone formation in ossification of a subperiosteal haematoma is not like that which we are describing, and we cannot accept this explanation.

Costello and Brown (1951), discussing the ossification in muscle in certain cases of anterior poliomyelitis, considered that when tissue atrophy and degeneration are taking place conditions are suitable for metaplastic ossification, especially if there be added the stimulus of trauma; and that factors such as local calcium, phosphorus and phosphatase concentration, and metabolic changes in tissue fluids, might all play a part. Brailsford (1941) suggested that the factor common to all ossification of this type was a lesion of the central nervous system, either purely local and due to trauma, or more central and due to a lesion of the cord, cauda equina or main peripheral nerve trunks.

Clearly there is much difference of opinion over the cause and origin of localized myositis ossificans and similar lesions in other tissues. Is it necessary to seek factors other than trauma to mesodermal tissues? In our case, with its short, violent history, it is hard to believe that there were any factors beyond the trauma from the severe tetanic contractions. This explanation would allow this case to fall into line with the usual picture of myositis ossificans localisata traumatica, differing only in the extent of the lesions from the involuntary self-inflicted trauma to many muscles and joints.

In the treatment of paraplegia and acute poliomyelitis it is customary to employ passive movement of joints. In the presence of muscle spasm which may accompany these conditions, more force may be applied to produce joint movement than is possibly appreciated at the time. In this respect it is interesting that, in many cases of myositis ossificans complicating poliomyelitis, the deposition of bone has first been noted during the early stages of convalescence on an average some twelve weeks after the onset of the illness, and a few weeks after physiotherapy had begun. In Brailsford's case of spastic diplegia, ossification followed operative trauma to muscle. It is known that repeated sudden muscle action may at times be followed by ossification in tendon attachments, and it is not uncommon to find it in the elbow region of athletes who indulge in field events such as throwing the discus and javelin. Heiss (1929) noticed this feature in a high proportion of Olympic athletes examined radiologically; it usually took the form of spurs firmly attached to bone in the region of capsular attachments.
Similar changes have been noted in relation to the lateral condyle of the femur in shoemakers, and in brachialis in pneumatic drill workers. Ossification is occasionally encountered around the hip joints of footballers.

CONCLUSIONS

Ossification of the type we have described usually follows severe trauma: and in this connection a dislocation is usually associated with more trauma to muscle attachments, ligaments and capsular structures than an uncomplicated fracture. In cases associated with lesions of the central nervous system there is almost invariably a history of trauma, or of physiotherapy, or of corrective operations. In those affecting the sensory system it is invariably associated with arthropathy of Charcot type, and repeated trauma to insensitive joints or tendons is probably responsible.

Our patient had received physiotherapy for ten days when we first saw him, but this had been instituted because of the increasing stiffness of the joints. We think there is little doubt that, although passive movement in the early stages may have aggravated the condition, the severe muscle spasms of tetanus were the cause; calcareous infiltration in muscle planes was observed in radiographs taken only three weeks after the onset of the spasms, an interval commonly found in post-traumatic myositis ossificans. Furthermore, most of the deposits occurred in the hamstring and flexor muscles of the knee—the muscles most likely to be damaged in the supine position by sudden spasms of knee flexion against the resistance of the distal leg against the mattress—and not in the extensor muscles of the thigh, which would be those most liable to injury on forced passive flexion with the leg free.

SUMMARY

1. Myositis ossificans as a complication of tetanus is described.
2. Ossification in mesodermal tissue is briefly discussed.

REFERENCES