PERONEAL COMPARTMENT SYNDROME

Report of a Case

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The anterior tibial syndrome is well known. It was described by Vogt in 1943 and numerous reports have since been published (Horn 1945; Hughes 1948; Carter, Richards and Zachary 1949; Blandy and Fuller 1957; Paton 1968). The etiology of the condition remains obscure but the clinical features are well recognised: gradually increasing pain in the muscles of the anterior tibial compartment, beginning during exercise and progressing after exercise has ceased, leading to induration, oedema and redness in the affected area and impairment of function in the affected muscles and in the anterior tibial nerve. It is less well known that the muscles and nerves of the peroneal compartment may be affected in a similar way; only four cases have been recorded (Blandy and Fuller 1957; Reszel, Janes and Spittell 1963; Lunceford 1965).

CASE REPORT

A man aged thirty, a research metallurgist and a keen amateur footballer, suffered a dull aching pain in the antero-lateral part of his right leg while playing football in May 1967. He continued the game in spite of increasing pain, expecting that the pain would disappear when he stopped running, but it did not do so and it became increasingly severe throughout the evening and after going to bed. During the night the pain was so severe that he decided to seek immediate advice. He was examined in the receiving room of the hospital. Radiographs showed normal bones and a diagnosis was not made. He was given an analgesic and allowed to go home. The pain persisted. Later in the morning he was seen by his own doctor who advised rest in bed and prescribed more analgesics. By midday he had severe "pins and needles" on the dorsum of the foot and the front of the leg, and he was unable to bear the weight of the bedclothes. Later the leg became "numb." He returned to the hospital where he was seen in the orthopaedic department.

On examination the foot was warm and of good colour and its pulses were normal. The skin over the antero-lateral aspect of the leg was oedematous, with a red patch in the middle of the area. The part was extremely tender and the underlying muscles were hard and swollen. The tenderness and induration were more lateral than would be expected in a case of anterior tibial syndrome and they seemed to correspond with the line of the peroneal muscles. There was lack of appreciation of pinprick in the distribution of the superficial peroneal nerve, but normal sensation in the first interdigital cleft. Active dorsiflexion and plantar-flexion of the ankle and toes were possible, as was a small range of inversion and eversion; but any movement, active or passive, increased the pain.

Operation—Immediate exploration was undertaken through a long vertical incision twenty-seven hours after the onset of symptoms. The muscles of the anterior compartment were found to be healthy in appearance, pink and contractile. In contrast the peroneal muscles were swollen and dark in colour (Fig. 1). When the overlying fascia was incised these muscles bulged through the incision and were seen to be engorged and necrotic. The upper half of the peroneus longus was friable and disintegrated when touched; it was therefore excised. The peroneus brevis, which was similar in appearance, was left. The fascia was not sutured but the skin was closed without difficulty. Suction drainage was employed.

Progress—Relief of pain was immediate and the wound healed by first intention. Three days after operation some active eversion of the foot was possible and four weeks later it was
surprisingly strong. It was, however, weaker than in the other leg, both subjectively and objectively, so a raise was fitted to the outer border of the shoe heel. With this the patient regarded function as normal. Ten weeks after operation perception of pinprick and touch was possible on the dorsum of the foot but was not quite normal. The patient later emigrated and no further follow-up was possible.

**Histology**—The excised specimen of peroneus longus was examined by Dr E. E. Payne. It showed widespread severe necrosis of muscle fibres. There were large areas of haemorrhage and oedema with infiltration by macrophages. Small parts of the muscle showed evidence of regenerative changes. The upper end of the muscle was more severely affected than the lower end. The vessels were patent and their walls were not unduly thickened. The picture was that of haemorrhagic infarction of a whole muscle.

**Fig. 1**
Findings at operation. The fascia has been split. The upper half of the wound shows the anterior tibial group of muscles, which are normal in appearance. Below, the peroneal muscles are seen bulging through their fascia: note the dark colour.

**COMMENT**

There are striking similarities between this condition and the better known anterior tibial syndrome. They are alike in their mode of onset, clinical features and morbid anatomy, and differ only in the physical signs, which depend on the particular muscles affected. In the anterior tibial syndrome there is weakness of dorsiflexion of the ankle and of extension of the toes, with involvement of the anterior tibial nerve leading to paralysis of the extensor digitorum brevis and loss of sensation in the first interdigital cleft. In the peroneal compartment syndrome there is weakness of eversion with loss of sensation in the distribution of the superficial peroneal nerve. It appears, too, that prolonged involvement of the peroneal compartment can involve the anterior tibial nerve as it passes through this compartment, and so cause paralysis of the anterior tibial group of muscles and extensor digitorum brevis, with sensory impairment in the first interdigital cleft. This complication occurred in the cases described by Reszel et al. (1963) and by Lunceford (1965), and in Blandy and Fuller's (1957) Case 3. In these cases the muscles of the anterior compartment were normal in appearance despite loss of function before operation.

From the similarity of the two conditions it is reasonable to suppose that they have a similar etiology. The cause of the anterior tibial syndrome is uncertain; the various arguments have been well put by Hughes (1948), Carter et al. (1949) and Paton (1968). The chief concepts are of segmental arterial spasm and of ischaemia of muscles and nerves caused by increase in tension within a fascial compartment. Although Hughes did not regard increase in tension
as a major factor, it is evident at operation in an acute case that the enclosed muscles are swollen and that the overlying fascia is tense.

So far as treatment is concerned it seems clear that decompression fasciotomy should be undertaken as soon as the diagnosis is made, even though it be made late. The sooner decompression is done the better will be the result; even late intervention may do good by relieving pain and salvaging some function. It has been suggested that muscles may regenerate to some extent after vascular injury (Bowden and Gutmann 1949). In our own case eversion of the ankle was only slightly weaker than on the normal side despite the removal of a large part of peroneus longus. Peroneus brevis could be felt contracting strongly a month after operation, which was surprising in view of its necrotic appearance at operation.

SUMMARY

1. A case of ischaemic necrosis of the peroneal muscles (peroneal compartment syndrome) is described. Recovery or regeneration of a seemingly necrotic peroneus brevis was noted.
2. The similarity between peroneal compartment syndrome and the anterior tibial syndrome is noted.
3. Treatment by early fasciotomy is advised.

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REFERENCES