THE ANTERIOR TIBIAL SYNDROME

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Acute ischaemia of the anterior tibial muscles with consequent replacement fibrosis or, in severer cases, necrosis of muscle is a well recognised entity. In 1942 Child collected fourteen cases from the literature and added one of his own. Since that time a further twenty-two cases have been reported (Sirbu, Murphy and White 1944, Horn 1945, Phalen 1948, Hughes 1948, Pearson, Adams and Denny-Brown 1948, Carter, Richards and Zachary 1949). Ischaemic necrosis secondary to severe injury is the most common type of this condition, which may thus complicate fractures involving the bones of the lower leg. Other cases have followed exercise. A more chronic form of the condition with symptoms persisting for a long time has not been clearly defined. Hughes (1948) suggested in relation to acute ischaemic necrosis that the syndrome was probably fairly common, but that many cases were wrongly diagnosed or even overlooked. Such an observation seems likely to be even more accurate in explaining the absence so far of any description in the literature of a chronic or persistent form of the condition. That such does exist is suggested by the following case.

CASE REPORT

A professional footballer, aged twenty-four years, was admitted to the Aberdeen Royal Infirmary with a complaint of pain in the front of both lower legs occurring on exercise. This had been present for two years. The onset was gradual, and it had coincided with his

becoming a full-time professional footballer and undertaking more intensive training. Gradually the pain had become more annoying because of its increased frequency and severity. It had begun first in the left leg, radiating from the lateral aspect down the shin towards the ankle. He described the pain as a "tightness" rather than a cramp, increasing

FIG. 1
Reflex vasodilation test. There is no significant delay in heating of the left great toe or dorsum of foot.
in severity with exercise and always forcing him to rest. The pain in the right leg was exactly similar, although not quite so severe. During the past six months the severity of the pain had forced him to rest after ten minutes of active training or playing football. With the pain he had also noticed numbness over the front of the ankles, a complaint that he tried unsuccessfully to treat by immersing his feet in hot water. There was no pain at rest or after walking or moderate exercise. There was no history of claudication in the calf or foot, or of pallor on exercise, or of injury or phlebitis. He was right-footed. Part of his regular routine training was to develop the left foot. On examination the patient's highly developed musculature, particularly about the legs, suggested "over-training." There was no evidence of ischaemia at rest and no change in the skin or nails of the feet. There were two small muscle herniae over the upper third of the anterior tibial muscles on each side. These gaps in the fascia were about equal in size, oval in shape, and admitted the tip of the forefinger. Ankle movements were full and powerful, and no peritendinitis was noted. There was no sensory loss. All the distal pulses were present, equal and easily felt. Examined after severe exercise, the pulses were still palpable. However, although there was no localised exercise pallor, the left foot over the dorsum felt colder than the right, and skin temperature recordings showed a fall of 2 to 4 degrees as compared with the right dorsum.

The blood Wassermann and Kahn tests, full blood examination, radiographs of the chest and legs, and electrocardiography showed no abnormality. Skin temperature recordings on the feet after heating the trunk showed full vasodilation of the skin vessels (Fig. 1). Arteriography was not considered justifiable.

The diagnosis of exercise ischaemia of the anterior tibial muscles was made. It was decided to try the effect of producing slight inversion of the foot along with dorsiflexion of the ankle. Accordingly, the front studs of the patient's football boots were placed on transverse bars, and a rubber pad was placed along the medial aspect of each sole. Tried for four weeks, this had no effect on the exercise pain.

Since the patient did not wish to change his occupation, operation was advised.

Operation—The fascia over the left anterior tibial compartment was incised, the incision including the fascial gaps. Repair was effected by a graft of fascia lata, measuring 8 inches by 2 inches. Biopsy of the anterior tibial muscle, which looked normal, showed no abnormality. The muscle tissue showed no ischaemic features and there was no evidence of fibrosis or organisation. After six weeks, during which it was clear that the patient's symptoms in the left leg had improved greatly, a similar operation was undertaken on the right side. Again the muscle tissue examined was normal.

Progress—Convalescence was uneventful. The patient has been followed up for four years since the operations. At no time has there been any recurrence of exercise pain. Although the muscles of the anterior tibial compartment have remained bulky, there was no evidence of muscle herniae (Fig. 2). The patient is still playing professional football, and his career
DISCUSSION

There appears to be little doubt about the diagnosis in this case. The nature of the anterior tibial pain suggested exercise ischaemia because it was constantly related to excessive exercise. The presence of the small muscle herniae indicated the possibility of excessive pressure in the anterior tibial compartment, presumably during this exercise, and it is important that the patient had bulky, highly developed muscles. The success of the operations gives further confirmation of the diagnosis. It is probable that the onset of ischaemic pain can be related to the increased training programme, although it is doubtful whether anything other than a general correlation between increase in exercise and onset of symptoms is worthwhile. It is surprising that the muscle showed no evidence of fibrosis, yet, if it had, both exercise pain and muscle bulk would have been expected to diminish.

Pathogenesis—The possible etiological factors in this condition have been discussed at length by Hughes (1948) and by Carter, Richards and Zachary (1949). Always present are the anatomical factors of the configuration of the anterior tibial compartment with its rigid walls and the vascular pattern of the muscles of that compartment. It seems probable that the former is of major importance, and it is unlikely that the so-called "open" lower end of the compartment is anything more than a potential opening well occupied by tendons and thus unsatisfactory as a "safety-valve." With increasing tension within the compartment "circulation within the intramuscular vascular networks is embarrassed" (Hughes 1948), and ischaemia leading to necrosis results. This necrosis may not be uniform, but it seems to affect the tibialis anterior and extensor hallucis longus to a greater extent than the extensor digitorum longus (Carter, Richards and Zachary 1949). It is doubtful whether this is of any significance, and whether the source of the muscles' blood supply is important, because if tension is a major factor the pressure will be uniformly distributed on the smaller intramuscular vessels. The increase in tension can be brought about in different ways. It can occur after a direct injury with or without fractures of one or both bones of the lower leg, and nineteen of the reported cases are of this nature. Carter, Richards and Zachary (1949) reported two cases in which a rise in tension followed blood transfusion, presumably with leakage of fluid into the compartment. However, the most common and interesting cause of increased tension is the muscle swelling which normally follows exercise, but which may be excessive or exaggerated by "rupture of a few muscle fibres or by haemorrhage into the muscles" (Carter, Richards and Zachary 1949) after over-exertion. This over-exertion is commonly marching or playing football. Howard (1937) described the changes in fatigued muscle and found an increase in bulk to be an important factor. It is pertinent that gross oedema with consequent increased bulk demands a patent arterial supply, a factor against the possibility of spasm or thrombosis as the cause of the necrosis.

Further evidence against the possible role of arterial thrombosis as a factor in etiology comes from this case, in which there was good anterior tibial pulsation after two years of ischaemic pain. Carter, Richards and Zachary (1949) also noted a normal circulation arteriographically in four cases, and dorsalis pedis pulsation in these and their remaining five cases at a late stage. Furthermore, as Hughes (1948) pointed out, such widespread necrosis as is usually present cannot result from any short thrombosis of the anterior tibial artery at the level of the upper end of the interosseous membrane, a suggested site of trauma and thrombosis, because of the alternative arterial supply available to the muscles from perforating branches of the posterior tibial artery and from the peroneal artery in the lower third of the leg. A long segmental block would be necessary to explain the necrosis on the basis of arterial thrombosis. There has been no evidence of this in the reported cases in which the artery has been resected, and in the cases in which it has not been resected it has been noted to pulsate at the front of the ankle.
There remains the possibility of arterial spasm or abnormal tone in an otherwise normal vessel. Hughes (1948) subscribed to this view, believing that the reason for the spasm was the accumulation of metabolites during exercise, a cause and effect which appears unphysiological. To explain fully the pattern of acute ischaemia which Hughes (1948) described, spasm has to act over a long segment of the anterior tibial artery, and yet be selective, in that the first part of the artery and its recurrent branch must not be involved (Hughes 1948). He also quoted Lewis (1936) and Cohen (1944) as stating that "whatever condition may be responsible for the acute ischaemia, it must have persisted for six to twenty-four hours in order to produce necrosis." This may be asking too much of "spasm." It also seems unlikely that prolonged spasm would not be followed at least in some cases by thrombosis, of which there has been no evidence.

In all the published cases the onset has been acute, and it appears that this is typical of the condition, although in cases without direct injury or a history of excessive exercise it is not clear why this should be so. A previous history of ischaemic muscle pain in the anterior tibial compartment in these cases would be expected, and yet generally it has not been forthcoming. Three of the patients reported, however, did have previous ischaemic symptoms. One of Hughes's (1948) patients complained of "vague pain" over the antero-lateral aspect of both lower limbs for nine months, and Horn (1945) discussed a patient who had noted "pains in both legs during forced marching." Carter, Richards and Zachary (1949) gave clinical details of only two patients, but one of these had a past history of pain in the front of both legs during route marches, and "even before joining the Army at the age of seventeen, had had pain in his legs after severe exercise." In other cases there was no specific mention of a previous history, and it is possible that it may have been overlooked.

It is evident from the case reported here that muscle ischaemia and thus exercise pain in the anterior tibial compartment may be persistent and represent a chronic type of the condition. Under this heading of chronic ischaemia the condition of peritendinitis crepitans has also to be considered. It appears that this condition may develop with excessive or unaccustomed exercise of the anterior tibial muscle group and, according to Howard (1937), who studied the local histological and chemical changes, it is a manifestation of chronic ischaemia of muscles. A milder and much commoner form of the condition, often seen in athletes undergoing training, may be that of "shin splints," described by Pearson, Adams and Denny-Brown (1948). The pretibial tenderness and pain of "shin splints" generally subside as training progresses, but on occasion they demand greater attention and care.

To summarise, it seems probable that increased tension in the anterior tibial compartment is the most important factor in etiology. This rise of tension may be produced in many ways—by direct injury with or without associated fractures of the lower leg bones, by a misplaced transfusion, or by excessive and perhaps unaccustomed exercise of various types. In all cases the ischaemia may be acute and cause replacement fibrosis or muscle necrosis, but in cases without direct injury it may be chronic, producing either persistent pretibial pain on exercise with or without herniation of muscle through the anterior crural fascia or a peritendinitis involving the muscles of the anterior tibial compartment.

Treatment—The possible progression of persistent ischaemia to acute complications should have a bearing on treatment, in that it is important not to dismiss lightly the complaint of exercise pain in the anterior tibial compartment, particularly when this occurs in footballers and athletes, when the exercise that produces it is likely to be repeated frequently. Should the condition be diagnosed in the chronic phase, the patient should be advised to give up whatever excessive exercise produces it, or, if this is impossible, decompression of the anterior tibial space should be undertaken. In such cases the presence of muscle herniae through the anterior crural fascia is of particular significance and indicates that increased tension within the space has existed for a long time. Although not mentioned by recent investigators, the presence of such herniae has been noted before. Sirbu, Murphy and White (1944) described ischaemic
necrosis occurring five days after the repair of a small hernia in the anterior crural fascia. This suggests that repair without fascial transplantation should not be undertaken, as it serves only to increase tension within the compartment.

In the acute phase decompression is an essential emergency measure, and the earlier it is undertaken the more effective it will be. Intervention is worthwhile as late as the fourth day after the onset, because complete recovery has been reported thereafter (Sirbu, Murphy and White 1944), indicating that even with acute symptoms deprivation of blood supply may not be total.

**SUMMARY**

1. A case of persistent exercise ischaemia affecting the anterior tibial group of muscles is described.
2. Pathogenesis and treatment are discussed. Decompression of the anterior tibial compartment is required if the patient is unwilling to give up whatever activity causes the exercise pain.

I wish to thank Mr G. Gordon Bruce for his encouragement and Mr Andrew Fowler for his permission to publish this case.

**REFERENCES**