TUBERCULOMA OF STRIATED MUSCLE

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For reasons not yet fully explained haematogenous tuberculosis of skeletal muscle is very uncommon. Reports of such lesions appear to be particularly scanty in the medical literature of the English-speaking nations. The problem has, however, interested some individuals who have sought to throw light on its pathogenesis and on its clinical manifestations.

True primary tuberculosis of muscle is said not to exist (Milch 1934). Haematogenous tuberculosis of skeletal muscle in the sense that it is a haematogenous infection secondary to infection elsewhere in the body is a rarity, and muscle itself is considered to occupy an important position among the tissues resistant to tuberculosis. To-day it is agreed that haematogenous tuberculosis of the striated muscle does occur. Culotta (1929), in 2,224 necropsies on tuberculous patients, discovered only four instances.

CASE REPORT

A woman aged forty-four was admitted to hospital complaining of a swelling on the front of the right thigh.

She had first noticed the swelling about twelve months before, and it had gradually increased. There was never any pain or sign of acute inflammation. She did not remember any injury to the right thigh, nor had she received an injection into the thigh.

Several days before admission she had noticed a sensation of "pins and needles" in the fingers and arm on the left side, and on waking in the morning she was found to have a left-sided hemiplegia. There had been no cough, no loss of weight and no night sweats.

On examination an obvious oval swelling was seen on the front of the thigh with its centre at the junction of the middle and lowest thirds. It extended downwards to within six centimetres of the upper border of the patella. The swelling was approximately 11.5 centimetres long by 5 centimetres at its widest. It was firm and well defined, and the overlying skin was of normal temperature and appearance, and moved easily over it. With the muscles relaxed the swelling was mobile transversely but not longitudinally. This mobility disappeared when the muscle contracted. There were full movements of all the joints. There was no swelling or pain on movement of the knee, nor was there any evidence of muscle weakness or atrophy. A few "shotty" glands were palpable in the groin. There was no oedema of the leg and the veins were not dilated. All reflexes were present. The swelling clearly involved the rectus femoris muscle, and a provisional diagnosis of fibrosarcoma was made.

Investigations—Haemoglobin 93 per cent; erythrocyte sedimentation rate 9 millimetres in first hour; leucocytes 12,100. The urine was heavily loaded with albumen and the centrifuged deposit showed thirty red blood cells to a one-sixth inch field. The urine was sterile. The blood urea was 38 milligrams per cent. Radiographs of the chest, spine, pelvis, hip, knees and femora showed no abnormality. Intravenous pyelography showed no abnormality in outline of the renal pelvis.

Operation—The tumour was found to be an encapsulated mass lying in the substance of the rectus femoris. The muscle was defined and separated from the neighbouring muscles, and the tumour with the whole muscle belly was removed in one piece. There was no communication with the underlying bone or with the knee joint. The wound was closed without drainage. It seemed probable from the macroscopic nature of the swelling that it was tuberculous; so streptomycin was given after the operation.
Fig. 1
Fresh specimen partly cut across and opened out showing tuberculous mass embedded in muscle.

Fig. 2
Separate halves of same specimen after fixing and mounting.
**Pathological examination**—The specimen consisted of muscle tissue and tendon, in which was embedded a tumour mass (Fig. 1). On sagittal section typical watery tuberculous pus was released, with flecks of caseous material. Examination of the cut surface of the mass (Fig. 2) showed it to be well encapsulated, the edge being demarcated from the surrounding muscle by a well developed fibrous layer. The cut surface appeared to consist of a framework of thick fibrous trabeculae containing large abscesses lined with shaggy caseating granulation tissue.
Histological examination (Dr D. B. Richards)—The section showed several areas of caseating necrosis surrounded by a zone of epithelioid cells and giant cells with a peripheral zone of lymphocytes. Surrounding these areas a wide zone of collagenous fibrosis enclosed single atrophic muscle fibres. In this fibrous tissue were a few discrete tubercles composed of epithelioid cells and giant cells without any central caseation or peripheral lymphocytic aggregation. The microscopic picture was that of tuberculosis of muscle.

Examination of direct smear of pus—A few scanty acid-alcohol-fast bacilli were seen. Culture was sterile: guinea pigs were not inoculated. Professor J. W. S. Blacklock was able to demonstrate tubercle bacilli in the sections.

Comment—The hemiplegia, which improved considerably, was considered to be unrelated to the tuberculoma of muscle and was thought to be due to a cardiovascular accident secondary to prolonged nephritis.

Formiggini (1911) in a review of fifty-nine cases accepted only twenty-seven as true examples of haematogenous tuberculosis of striated muscle. Hanke (1932) reviewed fifty-five authenticated cases of tuberculosis of muscle considered to be haematogenous. In 1948 Cortesi and Venturi accepted ninety-four published cases as authentic, and since then there appear to have been two further cases described, one by Gallarate (1950) and one by Magaldi (1952).

Tuberculosis can affect skeletal muscles in two distinct forms. There is the secondary and relatively common form, in which the tuberculous process spreads into the muscle by direct extension from neighbouring structures.

The other type, the so-called primary or haematogenous type, is much less common. It has been described by some authors as primary tuberculosis of muscle, but strictly speaking
it is haematogenous in origin because the lesion is considered to be carried to the muscle by the blood stream from a tuberculous focus elsewhere in the body. This focus may not be demonstrable. In the case reported here no other source of infection was found. These truly primary and latent centres almost always reside in the cervical, bronchial or mesenteric lymph nodes (Magaldi 1952).

There is, in addition, the rare possibility of a direct penetration of tubercle bacilli into the muscle tissue by hypodermic injections. Such a case was described by Coope (1946) after penicillin therapy. Magaldi (1952) quoted two similar cases after autopyotherapy and anti-typhoid injections.

The rarity of haematogenous tuberculosis of muscle has been the subject of much investigation. Several hypotheses have been suggested, none wholly satisfactory. It was suggested by Tria (1891), and again by Cagnetto (1925), that the production of lactic acid by the muscle tissue prevents infection, perhaps by a special bacteriolytic action (Tria). Zahnert (1929) ascribed resistance to the richness of the blood supply, and Culotta (1929) to the scantiness of the reticulo-endothelial system associated with the tendency of the bacillus to prefer connective tissue. Plummer, Sanes and Smith (1934) objected that this does not explain the frequent occurrence of tuberculosis in the liver and kidney, both as highly differentiated and well vascularised muscle and having a similar dearth of lymphatic tissue. Pettinari (1936) also pointed out that this hypothesis explains only the slight tendency of the tissue to form giant cells and organise complete tubercles: it does not explain the resistance of the muscle fibres to the tubercule bacillus.

Richet (1927) observed that dogs in which he had reproduced tuberculosis of muscle responded favourably to the administration of fairly large doses of muscle juice and concluded that it contained a special muscle antitoxin with bacteriolytic potency. Hensel (1950) was unable to verify this in rabbits. Prussia (1931) explained Richet's good result by supposing that the nutritional element administered with the muscle juice enabled the animal to overcome the infection by its own natural resistance. Cagnetto (1925) noted that, in rabbits infected with trichinosis and subsequently infected with tuberculosis, the tuberculosis manifested itself predominantly in the muscles most affected by the trichinosis, possibly because this produced a local concentration of histiocytes.

Some authors have suggested that the contractile power of the muscle fibres is a defence against the infection of these fibres. It appears to have been established that cutting the nerve supply to the muscle increases the risk of haematogenous tuberculous infection of that muscle.

A review of these hypotheses suggests a biochemical explanation in which the products of muscle metabolism appear to play an important role in the resistance of muscle to tuberculous infection.

Saltykow (1902) injected tubercle bacilli into the femoral artery of animals. He showed that the first tuberculous lesions were in the intima and walls of the muscle vessels. The lesions then spread to the interstitial tissue. The muscle fibres themselves were involved only in a very late stage, and then only secondarily.

Pettinari (1936) also described the histological characteristics of the disease in muscle. He explained that the disease attacks the connective tissue in the interstices of the muscle. The muscle fibres themselves are not directly attacked, but are affected indirectly by compression and by the action of toxins. They show granular, fatty or hyaline changes. Neither clinically nor experimentally was there ever a violent specific reaction to the tubercle bacilli in the muscle. An initial leucocytosis is rare, and the formation of tubercles usually not well marked. He suggested that this supports the claim that the reason for the refractory nature of striated muscle fibres to infection by the tubercle bacillus is its slight receptivity and not its great power of resistance.

Haematogenous involvement of striated muscles occurs almost equally in the upper and lower limbs. Muscles of the abdomen and trunk are involved much less frequently. The
significance of injury has been disputed. Magaldi (1952) cited a number of authors who support and those who oppose the localising action of trauma. The much greater frequency of tuberculosis in the muscle groups liable to injury suggests that repeated trauma may influence its localisation; but the matter is still controversial.

**Histology**—Morphologically, Prussia (1931) and other authors distinguish three types: nodular tuberculosis, inflammatory tuberculosis myositis, and sclerosing myositis.

**Nodular tuberculosis**—These lesions form as small tuberculous nodules of varying size in the interstitial connective tissue, sometimes separated clearly from the surrounding tissue by a fibrous layer. The centre of the lesion caseates and may form a cold abscess. This may spread progressively to involve more tissue. Such an abscess can involve subcutaneous tissue and eventually skin to form a collar-stud abscess. The fibrous barrier may localise the abscess, the outside fibrous tissue gradually merging into the surrounding tissue.

**Inflammatory tuberculous myositis or the granular-fungoid type**—This type is characterised by the production of luxuriant granulation tissue which invades the interfascicular connective tissue. Giant cells and classical tubercle formation may be absent. The lesion can undergo liquefaction, thus forming a cold abscess.

**Sclerosing myositis or tubercular cirrhosis**—This is considered a rarer form, the inflammation causing a diffuse sclerosis with disappearance of muscle fibre. Inflammatory nodes with typical tubercle formation can be found in the sclerotic tissue or may be completely absent. The lesion itself assumes a cirrhotic appearance.

These types are of theoretical interest only as they may co-exist in the same lesion and represent different stages in the same process advancing to necrosis and caseation, depending on the virulence of the germ and the power of resistance of the patient.

**Sex and age incidence**—Culotta (1929) found that the incidence was unaffected by sex.

Magaldi (1952) found it generally an affection of youth, the third being the most affected decade.

**Clinical features**—The disease manifests itself insidiously. The swelling becomes slowly and progressively larger. Pain is usually inapparent and the functional limitation is usually slight. Constitutional disturbances are usually absent: when they do occur they include a slight fever, loss of weight, malaise and sweating. The general condition of these patients remains good unless there are tuberculous lesions affecting other viscera. The mass in the muscle substance is usually single. Normally movable, it becomes fixed on contracting the muscle. The consistency may be firm, soft or fluctuant, depending on the stage reached in the degeneration. The swelling is not usually attached to the skin or surrounding structures, and the regional lymph nodes are seldom involved. Examination of aspirated pus exceptionally reveals tubercle bacilli. There may be slight leucocytosis.

**Diagnosis**—The diagnosis clinically is usually difficult, especially the differentiation from the other and commoner tumours of muscle, such as benign and malignant lesions, the chronic granulomata and the cystic lesions of muscle. The condition is usually diagnosed at operation or after pathological investigation of the specimen. The haematogenous origin of the lesion must then be verified by careful investigation to exclude direct extension from a neighbouring tuberculous focus, thus excluding the commoner secondary type of tuberculous infection of muscle.

**Course**—It seems that the disease is chronic and progressive. Spontaneous healing has not been described. Dissemination may occur through the blood or lymph stream from the muscle focus. Extirpation of the focus, with general anti-tuberculous therapy, is therefore the treatment of choice. The functional result depends on the interference with muscle. The prognosis after satisfactory excision will depend on the general condition of the patient and the extent of any tuberculous lesion elsewhere in the body.
SUMMARY

1. An example of haematogenous tuberculous lesion of striated muscle is described.
2. Its rarity is stressed and pathological details described.
3. Some of the theories for the refractory reaction of muscle tissue to tuberculous infection are discussed, and experimental work is reviewed.
4. The prognosis, treatment, and the relationship of the disease to trauma are considered.

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REFERENCES


