PATHOLOGY OF RUPTURED PLANTARIS

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Very little is known of the exact pathology of the lesion described as "ruptured plantaris" because the condition does not require operation and its morbid anatomy is therefore seldom seen. The following case report shows, however, that actual rupture of the belly of the plantaris muscle does occur, and that its clinical features tally exactly with the textbook descriptions of "ruptured plantaris."

Case Report

Sgt. X., aged 31, was admitted to hospital after an accident in which he had been thrown from a motor-cycle while travelling slowly. He complained of severe pain in the left calf region, and was under the impression that he had received a direct blow on the calf. Further questioning showed that he had slid across the road and been brought to a stop by the sole of his left foot hitting the kerb, thus producing forcible dorsiflexion of the ankle.

Examination revealed no evidence of direct injury to the calf, and there was no bruising. He was tender over the whole of the upper third of the leg posteriorly, but acutely so over the site of the plantaris muscle. There was a transverse incised wound over the tendo Achillis $2\frac{1}{2}$ in. long, 3 in. above the ankle-joint. There was no other injury.

The wound was excised under gas-oxygen anaesthesia; the incision involved skin and fascia only, the tendo Achillis being exposed but showing no evidence of damage. On the medial side, a little deeper in the wound, it was noticed that the plantaris tendon was lying slack, and gentle traction upon it resulted in the delivery of the entire tendon and half the muscle belly ruptured transversely about its middle. The lower end of the tendon was detached and the whole removed. The wound was sutured.

Pain in the calf persisted for 2 to 3 weeks and staining of the skin of the leg by effused blood, with some oedema, appeared 3 days after the injury. Dorsiflexion of the ankle beyond a right-angle was painful for 4 weeks and was relieved by raising the heel of the boot. Physical treatment was aided in the early stage by local infiltration with novocain. (Campbell, 1938; Smiley, 1939.)

The wound over the tendo Achillis, which allowed the inspection of the plantaris tendon, was presumably incidental, as the deeper structures showed no sign of injury. The plantaris must therefore have been ruptured by indirect violence applied in dorsiflexion of the ankle.

Discussion

The clinical features of "ruptured plantaris" are well known. The average age of 8 cases seen by me was 30, and it is essentially a lesion of relatively young healthy tissues. Classically, a tennis player reaching back for a shot produces a forced dorsiflexion of his ankle; a sudden pain occurs, and he thinks he has been struck on the calf by his partner. Pain increases during the next few hours, and swelling of the leg followed by bruising is seen in 2 to 3 days. A painful acutely tender area over the plantaris muscle belly in the upper third of the calf is present at first, but later pain is felt only when the ankle is dorsiflexed beyond the right angle. The period of recovery lasts about three weeks.

The pathology of the lesion is the subject of much doubt. It is variously supposed to be a subcutaneous rupture of the plantaris tendon, a partial or complete tear of the muscle fibres or the musculo-tendinous junction of the plantaris, or a rupture of some fibres of the soleus or gastrocnemius muscles. The case described shows that, sometimes at least, the symptoms are produced by rupture of the plantaris muscle belly, and the following facts and observations support the view that the clinical syndrome is always so produced.

The plantaris is absent in 7.5% of subjects, according to Gruber, quoted by Quain (1892). Pilcher (1939) found the muscle absent or rudimentary in 16 of 100 consecutive post-mortem examinations, and states that the deficiency is more common in women than in men. The frequency of occurrence of the plantaris muscle is thus compatible with its presence in every case showing the classical clinical picture of rupture.

Greater tension per unit area is developed in the plantaris on forced dorsiflexion of the ankle than in the other calf muscles, because the length of the plantaris belly is only one-

fifth of the total length of muscle and tendon; whereas one-half the total length of the soleus and gastrocnemius is composed of muscle fibres. I exposed the tendon in a number of cases for use as a graft in the repair of hernia, after the method described by Pilcher (1939). In each case, when the ankle was fully dorsiflexed the plantaris was seen to be as taut as a bow-string, while the tendo Achillis still showed elasticity. It seems reasonable to postulate, therefore, that in healthy tissues the sequence of events in increasingly violent dorsiflexion of the ankle is: (1) partial tear of the plantaris; (2) complete tear of the plantaris; (3) rupture of the other calf muscles in addition, or the tendo Achillis. Where the tissues are not healthy the order may be altered, as seen in the easy rupture of the tendo Achillis of elderly or syphilitic subjects. The first two of the above possibilities are the lesions occurring with the strain causing the "ruptured plantaris" syndrome.

An attempt was made to rupture the plantaris in a fresh young healthy cadaver by seizing the tendon with a haemostat and pulling. This caused rupture at the point seized, but when local damage was prevented by looping the tendon round a wooden peg, strong traction caused the muscle to tear through the middle of its belly. This, together with the findings in the case described, proves the weakest part of the muscle to be its contractile fibres.

The site of tenderness in 8 cases of "ruptured plantaris" seen by me was over the plantaris muscle belly. This is strong evidence pointing to the muscle itself as the site of the lesion.

Conclusion and Summary

The lesion described as "ruptured plantaris" is in fact a partial or complete tear of the fleshy fibres of the plantaris muscle. The case report correlates the clinical syndrome with a complete rupture of the plantaris muscle belly. The anatomical features of short muscle belly and long tendon cause this muscle to be subjected to greater tension than the other calf muscles—a fact which is well demonstrated by exposure at operation. The weakest part of the muscle is its belly, as proved by the experiment in the cadaver and the findings in the case described. The classical type of trauma causing the lesion in healthy tissues, together with the location of tenderness, distinguish plantaris rupture from other similar lesions.

I am indebted to Lieut.-Col. A. F. L. Shields, commanding officer of the hospital in which the case occurred, for permission to publish it.

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Medical Memoranda

Cavernous Sinus Thrombosis

There can be no doubt that treatment with penicillin has considerably improved the prognosis of cavernous sinus thrombosis. In a leading article in the *Lancet* (1945) it was pointed out that "chemotherapy is likely to cure just that type of case where the outcome was formerly almost hopeless—spread of infection from anterior veins draining a septic lesion of the face—because a lesion here is plainly visible, and any divergence from routine healing, or signs of more deep-seated mischief, become rapidly obvious, so treatment can be begun in time." In addition, treatment with penicillin may result in a cure even when its administration has been delayed for any reason, and Johnstone (1945) has reported a case in which administration was not begun for 10 days, and yet the patient was completely cured.

It may be of interest to record the following case, in which penicillin was not given until 6 days after the onset of the illness, and in which recovery was complete.

CASE HISTORY

Miss A., aged 23, a ledger clerk, was admitted to hospital on the evening of Jan. 12, 1945, as a case of erysipelas. The illness had begun suddenly on Jan. 8, with headache, limb pains, and vomiting. On the 9th she was put to bed, and since the afternoon of the 11th had been given a total of 6 g. of a sulphonamide. On examination she had an area of erythema extending from the right cheek to the forehead and spreading across the nose. It was extremely tender

and slightly raised. Both eyelids were oedematous. Her temperature was 103.2° F., pulse 120. She was immediately given 2 g. of sulphadiazine, followed by 2 g. 4 hours later, then 1 g. 4-hourly, with pot. cit. and plenty of fluids.

On Jan. 13 her condition was much the same, but her eyelids were more swollen—she could not open her right eye at all. The temperature was swinging between 99.2° and 101° F. during the day. At 10 a.m. on the 14th her temperature was 100.2° F. and the eyelids were worse. The right eye was definitely proptosed, and the left slightly so, and both showed early chemosis. She complained of feeling "dopy," was apathetic, and was rather slow in answering questions. A diagnosis of cavernous sinus thrombosis was made. At 2 p.m. the temperature was subnormal, but there were some petechiae on her eyelids and body. Treatment with penicillin was begun with a dosage of 20,000 units 3-hourly, intramuscularly. The sulphadiazine was discontinued. During Jan. 13 and 14 the patient had several rigors.

On Jan. 15 her temperature had risen again, there was marked chemosis of the conjunctivae of both eyes, with a blue discoloration of the eyelids, and she felt extremely drowsy. She complained that when she managed to open her eyelids a little she found that the vision of her right eye was blurred, but her left one was normal. Her pupils were small but reacted to light; and her external ocular movements were grossly restricted. It was impossible to examine the ocular fundi. Her general condition was worse, and a course of sulphathiazole was started—1.5 g. 4-hourly. All day she was extremely drowsy, with a swinging temperature.

On the morning of the 16th the temperature was normal. She still felt very drowsy, but had had 2 gr. of phenobarbitone in the early hours of the morning. Her eyes were painful but definitely less swollen, and the condition appeared much more localized. A boil on the right side of her nose was beginning to point—there had been no evidence of this previously, as the whole cheek had been swollen; hot fomentations were ordered. Her temperature rose again to 101.4° F. that night, and the next morning to 102.2° F. At this time the local condition was very much better and there was less chemosis, but a few fresh petechiae were present on the trunk and she complained of a stiff neck. All meningeal signs were equivocal. Her spleen was not palpable. By that night the temperature had dropped to normal and risen again to 100° F., and she was complaining of very severe pain in the head and neck. During the night there was a discharge of thick green pus from the boil on the nose, from which *Staph. aureus* was subsequently grown. The temperature was down again in the morning and the patient was much better subjectively. The sulphathiazole was reduced to 1 g. 6-hourly.

On Jan. 19, 20, and 21 the temperature was still swinging, but never reached 100° F. The eyes were practically normal. The sulphathiazole was stopped on the 19th, after a total of 34 g. had been given. On the night of the 22nd, the temperature having been normal for 36 hours, the penicillin was stopped after a total dosage of 1,320,000 units. On Jan. 23 the patient complained of double vision, and had a left external rectus paresis. On the 26th this was no better. The optic disks were apparently normal except for slight blurring of the nasal side of each. On Feb. 7 she complained of tenderness in the distribution of the right supra-orbital nerve, which had troubled her for about a week.

The patient was discharged on Feb. 14 feeling perfectly fit but for slight diplopia on looking to the extreme left, and with what she called her "old face again."

DISCUSSION

But for penicillin this patient would almost certainly have died. Although no blood culture was done, the swinging temperature and the occurrence of petechiae and rigors indicated that she had a septicaemia. There may also have been meningeal involvement in view of the severe pain in the head and neck, with equivocal meningeal signs. Lumbar puncture was not carried out, as the pain was well relieved with 1/6 gr. of morphine and did not recur with the same severity; and little more could have been done therapeutically if it had been definitely known that meningitis was present, except perhaps the administration of penicillin intrathecally.

Prof. Garrod (1945) found that sulphathiazole *in vitro* "somewhat reduces the rate of disinfection by penicillin." Does the present case bear this out *in vivo*? Would the patient's condition have improved more rapidly with penicillin alone? The temperature had not come down to below 100° F. permanently until after the sulphathiazole was stopped; 36 hours after that it had become normal for good, and penicillin was discontinued 36 hours later. But probably the temperature would have settled just as quickly had the sulphathiazole been continued. However, with no bacteriological test and no apparent lesion causing the thrombosis in the early stages, it was not justifiable to do anything but give full doses of penicillin and a sulphonamide.

I am indebted to Dr. J. C. McEntee, acting medical superintendent, for permission to publish this case; and to Drs. Bamforth and Seward, of St. Thomas's Hospital, for generously supplying the penicillin.

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Reviews

ANTIMALARIAL DRUGS

Antimalarial Drugs. General Outline. By O. Temkin and Elizabeth M. Ramsey. (Pp. 110, with Bibliography. No price given.) National Research Council: Division of Medical Sciences. Issued by the Office of Medical Information (under grants of the Carnegie Corporation and the Johnson and Johnson Research Foundation). Washington, 1944.

The war in the Pacific compelled careful revision of our methods for controlling malaria, and the loss of Java and its quinine supplies necessitated the overhauling of our choice and use of antimalarial drugs. The present volume is a careful review of the information available in the published literature about such compounds, and it has been compiled as a basis for the revision which the military situation demanded. The first part of the work describes the quinine situation in wartime, the principles on which the antimalarial compounds pamaquin (plasmoquine) and mepacrine (atebrin) were discovered, and the general pre-war opinions about antimalarial drugs as set out in the Third and Fourth General Reports of the Malaria Commission of the Health Organization of the League of Nations, issued in 1933 and 1937 respectively. A list is included of the main compounds studied during the pre-war years, with indications of their action upon avian, simian, and human malaria. Detailed summaries are given of the literature concerning the pharmacology, antimalarial activity, and toxicology of quinine, mepacrine, and plasmoquine. Subsequent sections deal with other compounds, such as thio-bismol, which show minor degrees of antimalarial action. Nowadays, when new means are being sought to produce radical cure of *vivax* malaria, the summary of information about neoarsphenamine is timely. The antimalarial action of this compound is limited to *P. vivax*, and it does not extend to *P. falciparum*; and even with *vivax* malaria, although neoarsphenamine will usually cut short the immediate attack of malaria, most reports agree that it is no more effective than quinine or mepacrine in preventing a subsequent relapse. Antimonials, mercurials, amidines, and derivatives have all been reported to show antimalarial action under certain conditions, but in no case have the facts suggested that these compounds would be valuable for general clinical use. Owing to new information which has been gained since 1942, and which is still confined to confidential reports and military instructions, the section on "Treatment Plans of Malaria under Present War Conditions" is more valuable as history than as a guide to immediate conduct.

The review should prove valuable to the investigators who are now engaged in studying the use of the compounds under war conditions, and it is mainly for them that it has been compiled; but to the general student it would prove misleading on many points, for it omits all the newly acquired information, which is now restricted to confidential American and British reports, and which considerably modifies pre-war views in many particulars.

GENERAL THERAPEUTICS

The 1944 Year Book of General Therapeutics. Edited by Oscar W. Bethea, M.D., F.A.C.P. (Pp. 447; illustrated. 18s. 6d.) Chicago: The Year Book Publishers; London: H. K. Lewis and Co.

The Year Book of General Therapeutics for 1944 is a useful book which can be strongly recommended to all doctors. It is small, well printed, and well written; it describes recent advances in treatment during the current year. The clarity of the exposition of difficult subjects is remarkable, and new procedures are described with caution and moderation. All the articles consist of summaries of published papers. The scope of the book can be gauged by a brief mention of some of the topics. There is a description of the use of caudal analgesia in midwifery: procaine hydrochloride in 1.5% solution is given by continuous drip by way of the sacral hiatus. There has been great enthusiasm for the method in the U.S., where it has been hailed as providing "dramatic relief of all pain" in labour. The book contains very useful sections on sulphonamides and penicillin, and a good account of the use of penicillin in syphilis. The treatment of burns is included, and antimalarial drugs have a section to themselves.

Janeway's work on the use of gamma globulin in measles is well described. This has arisen out of the work of Cohn