Giant cell arteritis as a cause of intermittent claudication

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DISCUSSION

Giant cell arteritis, also known as cranial, temporal or granulomatous arteritis, is a vasculitis of unknown aetiology, predominantly affecting people over 50 years of age. Incidence figures vary at around 1–76 per 100 000 of the general population (Chakravarty and Scott, 1993).

Giant cell arteritis produces a variety of symptoms, most commonly tender scalp and temporal arteries, headache, visual symptoms, fever, polymyalgia rheumatica and jaw or tongue claudication. The erythrocyte sedimentation rate is raised in 97% of cases.

Large artery involvement is a much less common finding. In a retrospective analysis of 248 patients with giant cell arteritis, Klein et al (1975) discovered that 23 patients had large artery vasculitis (9%). However, only three patients had isolated lower limb claudication and in none of these was it the presenting symptom. In 14 of the 23 patients, steroid therapy improved the symptoms of claudication which was usually in the upper limb.

To our knowledge there are only 12 case reports of giant cell arteritis in the literature presenting with lower limb claudication. It was first reported by Finlayson and Robinson in 1955. Of the 12 cases, the mean age is 51 although interestingly, a 5-year-old boy has been reported to have undergone a left above knee amputation from lower limb gangrene caused by giant cell arteritis (McEnery, 1977).

If the diagnosis is made early and steroids promptly commenced, it is possible to avoid amputation for this rare presentation of giant cell arteritis. Of the twelve case reports, the diagnosis was made early in four patients. High-dose steroids were commenced and amputation was avoided. In seven patients diagnoses were delayed, leading to four amputations and two deaths, and in the case of the 5-year-old boy diagnosis was not delayed but still lead to amputation.

Tarnoff et al (1995) reported a case for which a femoro-popliteal bypass graft was performed for femoral artery occlusion. The vein grafts themselves occluded and failed, but steroids, which were then started after a diagnosis of giant cell arteritis was made, greatly improved the symptoms of the patient.

CASE REPORT

A 57-year-old lady who works in the post office presented initially in May 1994 with a 5-month history of left calf claudication at 50 yards.

Six months previously she had also complained to her GP of joint pain, tiredness, and muscle aches. Rheumatoid factor was negative and erythrocyte sedimentation rate was elevated at 102. A diagnosis of polymyalgia rheumatica was made and she commenced a course of low-dose steroids, from which she gained dramatic relief. However, the pain in her legs continued and 6 months after the onset it caused sufficient handicap to interfere with her lifestyle.

At presentation to the vascular clinic, she was no longer on steroids. She smoked ten cigarettes per day and had no other atherosclerotic risk factors. On examination she had palpable femorals with no pulses distally. An arteriogram was arranged, which showed bilateral femoropopliteal occlusions with both popliteal arteries being patent above the knee, with poor run off. A diagnosis of peripheral vascular disease was made and confirmed by ankle:brachial pressure indices of 0.7 and 0.76 for the dorsalis pedis arteries on the right and left side respectively. She was advised to stop smoking and was put on an exercise regime. An outpatient review was to be carried out in 2 months time.

Her joint and muscle symptoms returned, so she was referred to the rheumatologist. A temporal artery biopsy was arranged. This showed multi-nucleated histiocytes consistent with giant cell arteritis. She was subsequently started on prednisolone 60 mg and methotrexate 10 mg.

Three weeks after commencement of treatment she made a dramatic improvement in her symptoms of joint pains and muscle aches. Unexpectedly, the claudication disappeared, despite the continuance of her smoking habit. Her claudication symptoms were therefore attributed entirely to large vessel vasculitis as a result of giant cell arteritis. Her ankle:brachial pressure indices improved to 0.89 and 0.92 for the right and left side respectively.

CONCLUSIONS

Giant cell arteritis is a rare cause of lower limb claudication, which may easily be misdiagnosed. The diagnosis is made by biopsy of an artery somewhere else, usually the temporal artery. This case report highlights the importance of early diagnosis for this rare manifestation of giant cell arteritis. Treatment with high-dose steroids should be promptly commenced to avoid debilitating claudication and amputation. HM

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