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Cystic adventitial disease of the common femoral and popliteal arteries

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Cystic adventitial arterial disease (CAAD) is usually situated in the popliteal artery and is a well recognized cause of intermittent claudication in otherwise healthy, young, non-smokers. Three cases of CAAD have recently been encountered, involving the popliteal artery in two patients and the common femoral in one. Two of these patients were hypertensive smokers in their sixth decades and only one was an otherwise healthy non-smoker, but all three had a characteristically rapid onset of symptoms. All had angiographic appearances suggestive of CAAD, confirmed by ultrasound and CAT scanning in one patient. Two were treated by resection of the affected artery and a replacement graft, both with excellent results. One popliteal lesion was bypassed with a vein graft which occluded after 3 months. CAAD may occur more commonly than generally realized. It can present in patients whose condition suggests an atheromatous cause for their symptoms. Since good results can be expected from appropriate surgical treatment in most cases, CAAD should be considered in the diagnosis of all patients with claudication, particularly when the onset has been rapid.

Keywords: Artery, cystic adventitial disease, popliteal artery, femoral artery

Cystic adventitial arterial disease (CAAD) is an unusual but well recognized cause of intermittent claudication. It occurs with an estimated frequency of 1 in 1200 claudicants[1]. The lesion consists of one or more fluid-filled spaces within the adventitia. These cysts can compress the arterial lumen as they enlarge or rupture into one another, resulting in distal ischaemia of relatively sudden onset. At the present time CAAD has been described in the popliteal artery in 122 cases, and in other vessels in a further 22 instances[1–6]. The typical patient with CAAD is described as a non-smoking, non-atherosclerotic, athletic male in this fourth or fifth decade[1].

Three patients with CAAD have been encountered within the last two years. Only one of these bore any resemblance to the 'typical patient'.

Case reports

Case 1

This 49-year-old woman presented with a rapid onset of claudication in the left leg and a march tolerance of less than 100 yards. She was a life-long non-smoker. There were no symptoms in the right leg which was normal on clinical examination. No pulses were present below the femoral on the left. Arteriography demonstrated a smooth stenosis of the left popliteal artery suggestive of CAAD at the level of the knee joint (*Figure 1*). This artery was explored but found to be so densely surrounded by fibrous tissue that resection or even adequate mobilization was not considered possible and a bypass vein graft was inserted. The graft functioned well for 3 months but then occluded. Subsequent arteriography showed no distal vessel suitable for a second graft and the patient underwent a lumbar sympathectomy. Her march tolerance 18 months after this procedure is in excess of 200 yards.

Case 2

This 52-year-old man had smoked cigarettes and been hypertensive for years. He presented with a rapid onset of claudication affecting the right leg and buttock. Examination revealed a thickened right femoral artery with a bruit. There were no pulses in the right leg distal to this vessel. Angiography confirmed the presence of a short stenosis in the right common femoral artery with distal dilatation (*Figure 2*). At operation the common femoral artery was found to be greatly thickened by fibrous tissue adherent to the hip joint capsule. The involved segment was resected and replaced with a vein graft, with complete resolution of the patient's symptoms. Histological examination of the resected specimen confirmed the presence of CAAD. Three years later there has been no recurrence of symptoms.

Case 3

This patient was a 51-year-old man. He had smoked 20 cigarettes a day for 20 years, required β-blockade to control his hypertension, and had suffered a right hemiplegia 3 years earlier. He presented with a rapid onset of claudication in the left calf. Together with the effects of his hemiplegia this severely restricted his mobility. Examination revealed a

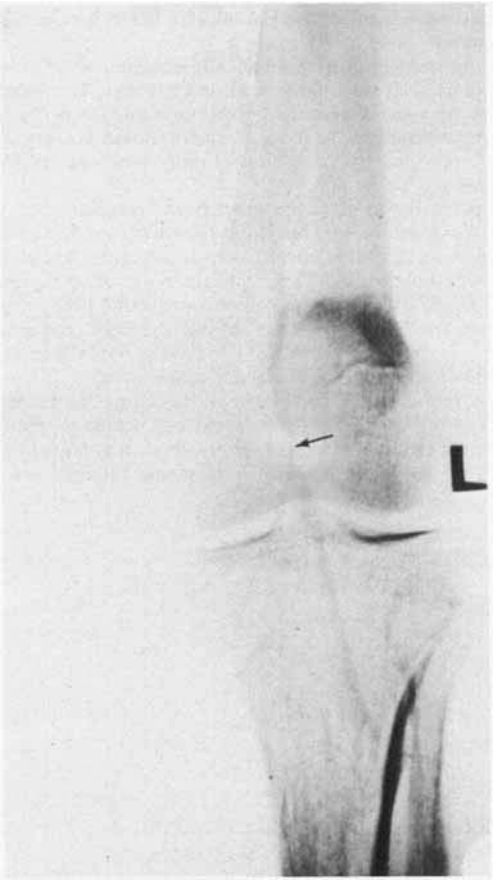

Figure 1 *A smooth stenosis (arrowed) is present at level of the left knee joint*

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Figure 2 *The right common femoral artery is stenosed (arrowed) with distal dilatation where it overlies the hip joint*

Figure 3 *A typical 'scimitar sign' stenosis (arrowed) is present in the left popliteal artery, proximal to the knee joint*

diminution of the left popliteal, dorsalis pedis and posterior tibial pulses. Arteriography demonstrated a smooth narrowing of the left popliteal artery typical of CAAD (*Figure 3*). The cystic nature of the lesion was confirmed by ultrasound and also by CAT scanning. At exploration the artery was found to be surrounded by fibrous tissue, connecting it to the adjacent veins and the knee joint. The involved segment was resected and replaced with a vein graft. Typical features of CAAD were found on histological examination of the resected specimen. The patient's claudication was completely relieved and he remains symptom free one year later.

Discussion

CAAD is considered to be a rare cause of intermittent claudication and its aetiology remains uncertain. Suggested explanations include the sequestration of 'synovial rests' in the arterial wall, protrusions of ganglion-like projections of synovial membrane into the adventitia, or the effects of trauma due to movement of a neighbouring joint[1]. The trauma theory does not explain the relative youth of most patients however, nor the extreme rarity of bilateral cases. The most likely explanation appears to be the ganglion theory, which is supported by several reports of continuity between adventitial cysts and the knee joint cavity[3,4]. No such continuity was demonstrated in any of our cases but each lesion was closely attached to the neighbouring joint by an unusual amount of fibrous tissue, which made mobilization of the vessel impractical in one case.

Several authors have stressed that CAAD occurs in otherwise healthy, young, non-smokers[1,7]. The clinical presentation has been described as being so distinctive that 'non-recognition is almost impossible'[4]. This may be true in most of the published cases, but in two of our patients (*Cases 2* and *3*) the condition presented in hypertensive smokers in their sixth decades. One of these had already suffered a cerebrovascular event, and the most likely clinical diagnosis in both would have been atheromatous narrowing of the common or superficial femoral arteries. Only the rapid onset of symptoms in both patients suggested a different aetiology, the sudden development of claudication being characteristically found in CAAD[1]. The first patient was the only one who resembled the 'typical CAAD patient' in any other way.

Certain clinical findings may suggest the diagnosis of CAAD when the lesion affects the popliteal artery. These include the obliteration of foot pulses when the knee is flexed[8], and the presence of an arterial bruit in the popliteal fossa[9]. The definitive diagnosis depends on the angiographic findings however. Characteristic appearances consist of a smooth narrowing of the arterial lumen, described as the 'scimitar sign'[10], along with normal distal vessels. These features enabled a pre-operative diagnosis to be made in both of our patients with popliteal disease, although histological confirmation was only obtained in one. The precise nature of the stenosis in the patient with femoral artery involvement was not appreciated until the lesion had been resected. Imaging techniques such as ultrasound or CAT scanning may prove useful in evaluating these less obvious cases since more conservative forms of treatment may be appropriate, provided that the diagnosis is certain.

A variety of surgical procedures have been described for the treatment of CAAD[1]. Non-resectional techniques include open cyst aspiration, evacuation or excision, with or without a patch angioplasty. Provided that perfect distal flow can be demonstrated intra-operatively these techniques produce satisfactory short-term results. Recurrence may occur in the long-term however, and is particularly likely to develop where a connection to the joint cavity has not been recognized and divided[4]. Patch angioplasties may be necessary to avoid narrowing the lumen but these have been shown to be prone to aneurysm formation and should probably be avoided[1]. Replacement or bypass of the affected vessel is required when it is completely occluded, or when cyst decompression is unlikely to produce an adequate lumen. Owing to the unusual amount of fibrosis around the arteries in our patients, replacement was considered to be necessary in two, and bypass in the third. The usually healthy distal arteries enable excellent results to be obtained in most cases and it is ironic that the only 'typical patient' in our series had the only treatment failure.

The presentation of three patients with CAAD within a short time, suggests that the condition may be more common than generally realized. Although typical cases should be easily recognized, CAAD also occurs in patients who would initially be expected to have atheromatous disease. Since this condition usually responds well to surgical treatment, and may even be treatable by percutaneous needle aspiration[11], it is particularly important that cystic adventitial disease is borne in mind when evaluating patients with intermittent claudication, especially when the onset has been relatively rapid.

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