

Successful Treatment of Ergotism with Nifedipine

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Ergotism is a rare cause of peripheral ischemia, usually associated with ergotamine tartrate therapy for migraine headaches. Recognition of the typical angiographic pattern may be important, as the classic drug history may not be obtained and the cause overlooked unless suggested by the angiographer [1, 2]. We describe the angiographic findings in a case of chronic ergotism that responded to nifedipine (Procardia, Pfizer), a calcium channel blocker.

Case Report

A 53-year-old woman was admitted with the chief complaint of numbness and severe burning pain in the left foot for several months and claudication in the left calf at 0.5 block. She had been on Cafergot (Sandoz) suppositories for more than 10 years and admitted to using one suppository (2 mg) per day for the previous 7 years. The left popliteal, posterior tibial, and dorsalis pedis pulses were absent, and the foot was cold and cyanotic. Ankle/brachial pressure indices for the left posterior tibial and dorsalis pedis were 0.32 and 0.5, respectively. The right lower extremity indices were normal.

Aortography with distal runoff showed narrowed caliber of the distal aorta and iliofemoral system bilaterally, more marked on the left (fig. 1A). The left superficial femoral and popliteal arteries were markedly narrowed. Trifurcation vessels were threadlike with a minimal peroneal artery patent to the ankle. No collaterals or thrombi were identified.

Cafergot was tapered to 0.5 suppositories per day and was discontinued 2 days later. Sympathetic block was unsuccessful. Migraine prophylaxis with propranolol was initiated, and oral nifedipine, 10 mg three times daily, was started. Within 2 days, all symptoms disappeared except for minimal toe paresthesias. Repeat Doppler indices at this time were normal bilaterally. Repeat angiography after 5 days of nifedipine therapy demonstrated marked improvement in left leg runoff (fig. 1B). Three months later, the patient is nearly normal without pain or claudication but with minimal residual numbness in the toes.

Discussion

Three major angiographic findings are noted in ergotism: arterial spasm, collateralization in severe cases, and thrombus formation [3, 4]. The usual findings are relatively symmetric involvement with more marked narrowing in the distal parts

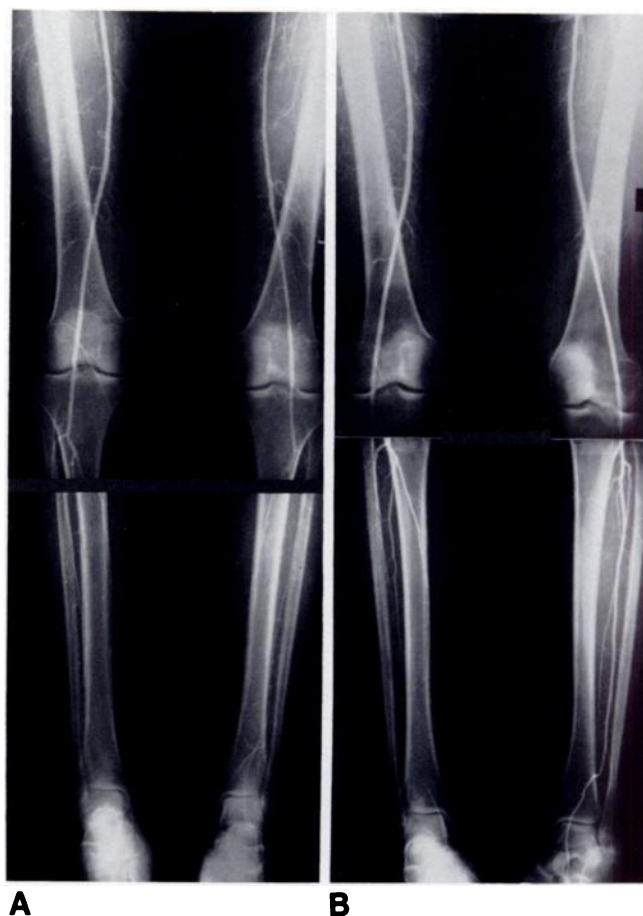


Fig. 1.—A, Distal runoff, before treatment. Narrowed vessels bilaterally with long focal segment of marked narrowing in distal left superficial femoral artery (SFA). Left trifurcation vessels are threadlike with anterior and posterior tibial arteries terminating at mid calf. Small peroneal artery is patent to ankle. B, Distal runoff, after treatment with nifedipine. Resolution of focal spasm in distal left SFA. Normal-sized anterior tibial artery is patent to ankle. Runoff is now faster in left leg.

of the limb. The lower extremities are almost always involved [3]; isolated upper extremity spasm is rare.

This case is somewhat typical, as the symptoms were unilateral. A recent report on possible synergistic effects of

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ergotamine and propranolol suggests that resolution of this patient's symptoms may have been prolonged by their concurrent administration [5]. Propranolol has also been indicated as a cause of Raynaud phenomenon [6].

Ergotism often responds to conservative therapy, which includes drug withdrawal, fluid therapy, and heparinization [2, 4, 7]. In limb salvage situations, intravenous nitroprusside effects prompt vasodilation [7].

In our case, vasodilation was achieved with oral nifedipine, a known peripheral and coronary artery vasodilator. This drug is a calcium channel blocker that inhibits the influx of calcium ions into cardiac and smooth muscle during the repolarization phase, thus decreasing contractility. In humans, the peripheral vasodilatory effect of nifedipine usually results in a 5–10 mm Hg decrease in systolic blood pressure. Experimentally, nifedipine can reverse ergonovine-induced coronary artery spasm [8].

Nifedipine is rapidly and fully absorbed after oral administration, with peak blood levels occurring in 30 min. Its half-life is about 2 hr. Elimination is via hepatic conversion to inactive metabolites and renal clearance.

Our case illustrates many of the problems associated with chronic ergotism: Chemical sympathectomy was ineffective; migraine prophylaxis with propranolol may have prolonged vasospasm, and the patient required long-term therapy for migraine. Nifedipine, 10 mg three times daily, seemed to reverse vasospasm promptly by Doppler and angiographic

criteria. When the limb is not in immediate danger, nifedipine, a calcium channel blocker, may be used to reverse ergotism. This drug is effective orally and does not require expensive intensive care monitoring as does nitroprusside infusion.

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