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Khrongwong Musikatavorn a; Suchai Suteparuk a

a Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

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CASE REPORT

Ergotism unresponsive to multiple therapeutic modalities, including sodium nitroprusside, resulting in limb loss

KHRONGWONG MUSIKATAVORN, M.D. and SUCHAI SUTEPARUK, M.D.
Department of Medicine, Faculty of Medicine, Chulalongkorn University, Bangkok, Thailand

Keywords Ergotism; Vasospasm; Sodium nitroprusside; Oral nifedipine; Limb loss

Unlike epidemics in past centuries, patients suffering from peripheral vascular ischemia related to ergotism now rarely lose a limb because of vasodilator therapies. We report a patient with ischemia from ergotamine tartrate who failed to recover with medical therapy, resulting in limb amputation.

Case report

A 28-year-old man was admitted to King Chulalongkorn Memorial Hospital with severe pain in his legs of one day duration. Seven days prior to admission, he had a fever without chills, bitemporal headache, and nausea. He went to a hospital and received ergotamine tartrate with caffeine (1 mg and 100 mg, respectively, three times daily), amitriptyline (10 mg at bedtime), domperidone (10 mg daily), and ibuprofen (400 mg three times daily). After five days of medication, his legs became severely painful, especially below the ankles, such that he could not walk. Eventually, his feet became cold and purple with some ecchymoses on the dorsal parts. He denied smoking and illicit drug abuse. His past medical history and family history were unremarkable.

Upon physical examination in the emergency department, his temperature was 37.3°C orally, respiratory rate was 18 breaths per minute, and heart rate was 100 beats per minute and regular; his blood pressure could not be obtained using an external sphygmomanometer. He was very distressed with pain. His feet were cold, cyanotic below both ankles, and very painful when moved. Both hands were cold but not painful or cyanotic. Radial, popliteal, tibialis posterior, and dorsalis pedis pulses were absent bilaterally. Other peripheral pulses (brachial 1+ bilateral and femoral 3+ bilateral) were diminished. His blood count, blood urea nitrogen, creatinine, and electrolyte levels were normal. The electrocardiogram and chest x-ray were normal. The creatinine kinase (CK-MM) level was 8194 U/L (normal 0–190 U/L).

He was given a large volume of intravenous crystalloid fluids. Angiography of the femoral arteries revealed diffuse spasm of bilateral superficial femoral, popliteal, anterior and posterior tibial arteries. Papaverine (30 mg) was infused in each proximal superficial femoral artery, with only a slight increase in caliber of the left femoral artery, four centimeters in length. The right was unchanged. Blood ergotamine levels were not measured. The patient was given intravenous sodium nitroprusside (SNP) with a starting dose of 30 \( \mu \text{g/min} \) and oral nifedipine (10 mg every 8 hours) immediately after the angiographic study. Enoxaparine 60 mg every 12 hours was given subcutaneously to prevent thrombus formation. Eighteen hours later, the dosage of SNP was increased to 100 \( \mu \text{g/min} \) without any palpable pulse. Epidural sympathetic blockage with bupivacaine provided pain relief. On the third hospital day, SNP was increased to 200 \( \mu \text{g/min} \) but pedal and posterior tibial pulses were still undetectable. Serum creatinine kinase (CK-MM) was increased to 12,070 U/L. Oral beraprost 20 mg every 8 hours was added. Despite these therapies, the patient’s left foot was still gangrenous and he underwent transmetatarsal amputation of his left foot on the fourth hospital day. Peripheral pulses became palpable the following day and his right foot was preserved. He was discharged with a program of physical rehabilitation after an 82-day hospitalization.

Comment

Ergotamine can cause vasoconstriction and vasospasm by stimulating alpha-adrenergic and 5-HT2A receptors. The peripheral vascular effects of ergotamine are greater than the
selective 5-HT1B/1D receptor agonist (triptans), since triptans have no activity at adrenergic and 5-HT2A receptors (1). Ergot’s toxicity includes acute peripheral vascular ischemia (gangrenous ergotism) (2) and claudications (3). Effective interventions have been proposed. According to reports from Carliner (4) and Anderson (5), complete resolution of peripheral ischemia can be achieved with intravenous sodium nitroprusside. The maximal SNP dosage administered in our case was not as high as that reported by Carliner (200 μg/min versus 247 μg/min). There are also cases successfully treated by intra-arterial infusion of SNP (6,7) and oral nifedipine (8,9). Continuous epidural anesthesia seemed to be effective in a report by Semb et al. (10), although most consider it ineffective, used only to relieve severe ischemic pain. Hyperbaric oxygen therapy has also been reported as successful in cases of ergot-related limb ischemia (11), and there are reports of successful use of intravenous PGI2 and PGE1 (12) and PGI2 analog (Iloprost) (13). Our patient was given oral beraprost without notable beneficial effect.

The peripheral ergotism can be disastrous and it requires early recognition and aggressive medical therapies. Other therapeutic modalities must be used immediately if there is a failure to respond to an initial therapeutic trial.

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References