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CASE REPORT

ERGOT POISONING IN PARAPLEGIA

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Summary. Ergot derivatives are widely used for migraine headaches. However, ergotamine tartrate may cause intoxication which may even become fatal. A case of ergot poisoning in a paraplegic patient is reported, where deep vein thrombosis and ischaemia of the lower limbs were the presenting signs.

Key words: Spinal cord injury; Paraplegia; Ergotamine poisoning; Deep vein thrombosis; Ischaemia.

Introduction

THE INDICATIONS for the therapeutic prescription of ergotamine are limited. An overdose or complications are, however, quite common since ergot derivatives are toxic. Instances of acute or chronic ergot poisoning have been repeatedly published (Martindale, 1977). The present report introduces for the first time complications of ergot when used in a paraplegic patient.

Case report

The patient was a 51-year-old man who had become paraplegic as a result of trauma, below the 8th thoracic segment when he was 42 years old. At the age of 37 he was treated for migraine with Temigran (TEVA) which contained: ergotamine tartrate 2 mg, chlorcyclizine 20 mg, caffeine 100 mg, codeine phosphate 25 mg, and dipyrone 100 mg. This treatment was continued by the patient with no proper follow-up. In 1978, 8 years after he had become paraplegic, he was treated for the first time for deep vein thrombosis (DVT) of the left leg and for sacral pressure sores. The pulses of the dorsalis pedalis, tibialis posterior and popliteal arteries were absent. Cyanosis and mild swelling of the foot were noted. These clinical findings disappeared within 2 weeks but 3 weeks later fractures of the lateral condyle of the femur and left tibial plateau were detected, without any obvious trauma. Two months later DVT and ischaemia of the left lower limb reappeared. At this juncture the possibility of ergot intoxication was considered and the Temigran medication was withdrawn. Within a few hours the objective signs of DVT and ischaemia began to subside and finally disappeared within 48 hours, at which time the pulses were detected in all of the arteries of the left leg. The patient remained asymptomatic for the next 4 years.

Discussion

Spinal cord injury (SCI) results in a particular neurohormonal pathological state which can make the administration of many drugs a challenging problem. Considering the ease with which the SCI patient may lose endocrine or neurological balance, each drug should be administered only after careful consideration of its possible side-effects and agonistic and antagonistic interactions with other drugs.

Ergotamine tartrate is often the drug of choice for the treatment of migraine. The beneficial effect of the drug is probably due to its action on smooth vascular muscle, as a result of its alpha-adrenergic blocking activity and other secondary actions (Martindale, 1977; Goodman and Gilman, 1980). Ergotamine also causes marked cerebral arterial vasoconstriction which closely resembles that induced by sympathetic mediators (Peters, 1972). When administered in low concentration ergot alkaloids may augment the effects of norepinephrine or the contractile response to acetylcholine or angiotensin (Goodman and Gilman, 1980). The possible side-effects of ergot administration include: postural hypotension, increased gastro-intestinal motility, nausea, vomiting, diarrhoea, reflex tachy- or bradycardia, anginal pain, myalgia, itching, anal burning, weakness, and ischaemic ECG changes. Ergot alkaloids may cause a significant rise in blood pressure as a result of peripheral vasoconstriction with damage to the capillary endothelium. Prolonged use of ergot may cause vascular insufficiency and gangrene of the extremities (Martindale, 1977; Goodman and Gilman, 1980), in which case the patient may complain of coldness, numbness, and pain in one or more of his limbs. Later, the arterial pulses may become faint or even disappear, and, unless adequate measures are taken, permanent damage may occur, resulting in gangrene or spontaneous amputation. Such a syndrome has been reported following therapeutic doses of ergot which were previously well-tolerated by the patient. This sudden toxicity may be due to an accumulation of the drug because of impaired liver function or increased sensitivity to ergot during febrile and septic states.

The striking circulatory changes may be easily observed in the normal patient. However, as the paraplegic patient has lost sensation in the lower limbs, he will be unaware of such presenting symptoms as numbness, itching, paresthesia, coldness, or pain, and the diagnosis may be delayed until irreversible vascular changes occur.

A question may be raised as to whether or not the DVT was a complication arising from the fracture or from ergot poisoning in the presented patient. DVT occurring after a pathological fracture in a paraplegic patient may persist for prolonged periods of time with an increasing degree of swelling and cyanosis. However, in this patient the signs of DVT began to subside within a few hours after the discontinuation of ergot and completely disappeared 48 hours later.

Since in SCI there is an exaggerated response to norepinephrine (Erickson, 1980), and since even a low concentration of ergot may increase the effect of norepinephrine, ergot derivatives are contraindicated in patients with SCI lesions. Moreover, the use of ergot should be avoided in any patient who is unable to feel the first adverse signs of the drug.

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