Lesson of the Week

Autonomic dysaesthesia due to ergot toxicity

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Modern cases of ergot poisoning (St Anthony's fire) are frequently iatrogenic, and occur when ergot alkaloids are taken in excess. The principal feature is intense vasoconstriction, but sensory neurological disturbances such as paraesthesiae or causalgic-like pains are often described.[1] We report on a woman who developed such an autonomic dysaesthesia affecting the lower limbs, which was initially mistaken for a development of her pre-existing chronic back pain.

Diagnosis of ergot poisoning in a woman with dysaesthesia was confused by a history of chronic back pain.

Case report

A 42-year-old married woman was referred for treatment of chronic low back pain and left-sided sciatica. This had developed over eight years and had followed a laminectomy and spinal fusion, performed for root compression. She was prone to attacks of migraine and had been taking a combined preparation (Migril) containing ergotamine tartrate 2 mg, cyclizine hydrochloride 50 mg, and caffeine hydrate 100 mg. Her physical examination was normal and there were no new radiological changes. She had had many different treatments, including paravertebral somatic blocks, extradural steroid injections, and facet rhizotomies with gradual improvement in symptoms.

In the second year of attendance and six months after she had started taking Migril, she developed a new pain, typical of an autonomic disturbance. It first appeared at her ankles and over eight months progressed upwards to affect the whole of both legs as far as the hips. It was a constant burning sensation that she likened to being on fire, and was so severe that it almost completely masked her back pain. Blockade of sympathetic nerve pathways always proved effective but the duration of relief was short (table). The response to intravenous regional sympathetic blocks with guanethidine[2] was particularly interesting. The duration of the effect was remarkably constant and a unilateral injection always produced bilateral relief. A placebo block using normal saline alone failed to relieve symptoms and after the previous effective blocks was immediately detected by the patient. A specific vascular cause for her symptoms was then considered.

Her ergot consumption had never been questioned, but in discussion she said that when the pain had started there had been considerable domestic difficulties, and she had been suffering frequent migraine attacks. To relieve her symptoms she had started taking one Migril at night prophylactically and more during the day if headaches occurred. She regularly took up to 12 tablets (24 mg) a week. This was considered to be excessive consumption of ergotamine, and she was referred to the migraine clinic. The ergotamine was withdrawn under cover of the serotonin antagonist pizotifen, initially in a dose of 3 mg daily. Since then she has improved. Intense vasoconstriction was not a feature of her ergotism although she frequently complained of cold legs. She had no other sequelae such as ischaemia or gangrene.

Relief of causalgic pain produced by "sympathetic" blocks

Procedure		Duration of relief (h)
Extradural injection 0·5% bupivacaine	× 1	3
Intravenous sympathetic block with 20 mg guanethidine	× 4	26-28
Intravenous pethidine 50 mg	× 1	½

Comment

Ergotamine has traditionally been used to relieve acute attacks of migraine, but it is unsuitable for prophylaxis.[3] The therapeutic ratio is narrow and if exceeded leads to dependence. Toxicity has been reported with doses as low as 10 mg in a week[1] and fatalities have resulted from doses of 26 mg or more.[4] Intermittent claudication, paraesthesia, and burning sensations in the arms and legs, particularly the latter, have been described.[5] It is thought to be secondary to ischaemia but has been reported in the absence of intense vasoconstriction.[1] Blockade of sympathetic pathways relieved the patient's pain and in each case the duration of relief was consistent with the length of action of the drug. The unusual bilateral relief obtained from the intravenous sympathetic blocks probably resulted from the release of excess guanethidine into the circulation on deflating the tourniquet. This patient also shows that careful evaluation is needed before treating symptoms alone. In cases where the character and distribution of symptoms frequently change, an organic basis for the pain may be overlooked.

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(*Accepted 9 October 1980*)