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Peripheral ischaemia after chronic ergot poisoning

Isquemia periférica tras intoxicación crónica por ergóticos

Dear Editor:

The ergots (ergotamine and dihydroergotamine) are forms derived from rye fungi with multiple side effects. However, despite this (favoured by their low cost and easy availability), they are widely used, enjoy great popularity and are among the most extensively used drugs by patients for medication overuse headache (MOH).[1]

Ergotism is a vasomotor syndrome represented by a case of claudication that is clinically no different from occlusive arteriopathies[2] and whose aetiology is usually iatrogenic due to the consumption of ergot in the MOH.

We report the case of a 20-year-old woman with a history of migraine without aura since age 14. The patient had consulted her primary care physician 3 years before for this reason. She was prescribed treatment with naproxen; not obtaining a good response to treatment, she began to self-medicate with ergot (Cafergot®), a drug that she used almost daily for 1 year (up to 20 days a month).

Her family history highlighted that her maternal grandfather and her mother also suffered migraines.

The patient complained of pain in both legs of about 3 months' duration, which started with walking and eased with rest (intermittent claudication), as well as pain in both calf masses and in the lateral areas of both legs. She did not present any signs of arthritis in any joints. The pulses in the upper extremities were positive and symmetric, as in the lower extremities at the femoral level. The pulses were weak upon palpation in the popliteal regions and absent in the posterior tibial and foot regions. She had cold feet and delayed capillary refill.

The patient was referred to the hospital emergency service, where she underwent an electrocardiogram, haemogram, coagulation studies and lumbar spine and sacroiliac radiographs, all of which were normal. The venous Doppler ultrasound of the lower extremities was normal, whereas a non-specific injury with involvement of the iliac and femoropopliteal sectors appeared in the arterial system of both lower extremities (right ankle-brachial index=0.6; left ankle-brachial index=0.55).

The patient was diagnosed with peripheral arteriopathy by ergotamine.

She discontinued treatment with ergots and non-steroidal anti-inflammatory drugs were prescribed for her headache. After 1 week without taking ergot, we observed normality in arterial pulses and capillary refill. Ankle-brachial index = 1 and control echo-Doppler was normal. One month later, the patient attended a review session, where it was observed that vascular normality and the absence of headache persisted.

In the eighties, different authors showed that analgesic drugs also contributed to the development of chronic forms of headache, and the concept of headache due to overuse of analgesic drugs thus appeared.[3,4] Given the difficulties in classifying all patients according to the definition of the International Headache Society (IHS), Silbertein et al introduced the concept of chronic daily headache, which was subdivided into 4 types (transformed migraine, chronic tension headache, recent onset chronic headache and hemicrania continua), each of which could be associated or not with the use of analgesic drugs.[5] In 2006, the IHS recognised the 4 subtypes of migraine proposed by Silberstein et al, and distinguished between the abuse of simple, combined, opioid and ergot analgesic drugs. The distinction between primary chronic headache and MOH was still maintained.[6] The 2006 IHS criteria defined MOH as headache present for 15 or more days in a month that had developed or worsened during medication overuse and when there was regular overuse for more than 3 months of one or more drugs used as acute treatment for headache (ergots, triptans, opioids or combined analgesic drugs consumed at least 10 days per month and/or simple analgesics consumed at least 15 days/month).[7]

The largest class of drugs associated with MOH in our area is that of simple analgesics (34.7%), followed by combined analgesic drugs (27.8%) and ergots (22.2%).[5]

Ergots, duet to their potent vasoconstriction activity, cause vasospasm in vascular smooth muscle. They act mainly on the peripheral circulation of the extremities, although they can act at any arterial level (aorta, iliac, femoral, renal, carotid, coronary).[8] The rapid disappearance of the drug in blood and the lingering effects in the arterial tree suggest that both the separation of the drug from its

receptor and the presence of active metabolites prolong the persistent vasoconstrictive effect.[8]

In peripheral ischemia due to ergots, the underlying problem is the persistent vasoconstriction, so treatment should be primarily vasodilative, although the first step in treatment should be the suspension of ergots.[9] Vasodilator therapy is needed in severe cases. Sodium nitroprusside has traditionally been used as the antidote for ergotism,[10] but its use would be limited by its side effects, requiring pump infusion[2] and the possibility of vasospasm after removing the drug.[8] Calcium channel blockers (nifedipine or nitroglycerin) have also been used for their vasodilator activity. Epidural or spinal anaesthetics have been used, but with not totally satisfactory results.[11]

Surgical treatment should be reserved for very advanced chronic ergotism or as a complement if there is a large area of tissue necrosis.[12]

Currently, the combination of alprostadil (PGE1)-·-cyclodextran (Sugiran) and heparin (sodium or low molecular weight) or pentoxifylline is considered more useful.[6]

In conclusion, ergots are not only one of the most common causes of headache due to medication overuse, they can also lead to serious complications such as the case presented here.

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Optical neuritis secondary to a rhinosinus disease

Neuritis óptica secundaria a rinosinusopatía

Dear Editor:

Rhinosinusopathy may involve severe morbidity-mortality if not treated or when treatment is ineffective.[1] Complications associated with a slow evolution depend critically on the condition of adjacent structures. The orbital cavity, due to its peculiar shape, is the most susceptible anatomical structure. We present a patient with right optic neuritis secondary to homolateral rhinosinusopathy.

The patient was 27 years old and had no prior history of interest. The patient was admitted from the emergency department due to retro-orbital pain of 5 days' evolution that increased with eye movements; in the latest few hours, the condition was associated to limitation of the right visual field. Neurological examination and fundoscopy were consistent with normality, with the exception of the previously referred scotoma. Visual field examination revealed a right visual field loss. Neuroimaging studies —

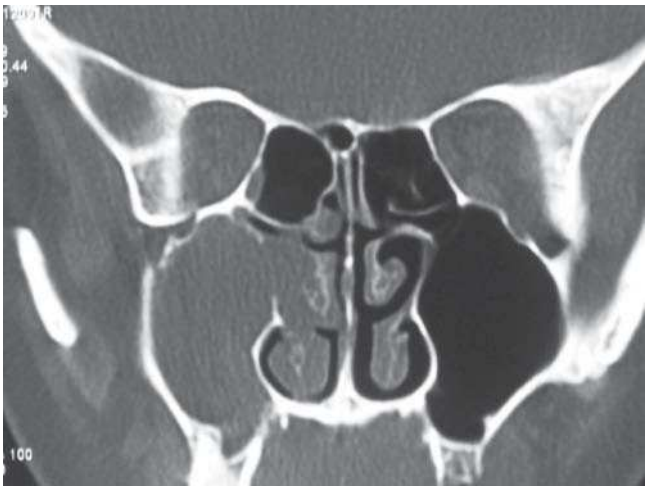

Figure 1 Computed tomography scan of paranasal sinuses: space-occupying lesion in the frontal and maxillary sinuses and ethmoid cells.