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 —

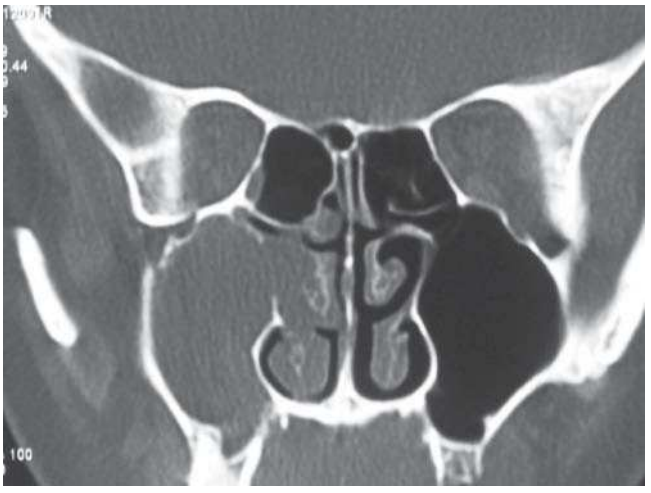

Figure 1 Computed tomography scan of paranasal sinuses: space-occupying lesion in the frontal and maxillary sinuses and ethmoid cells.

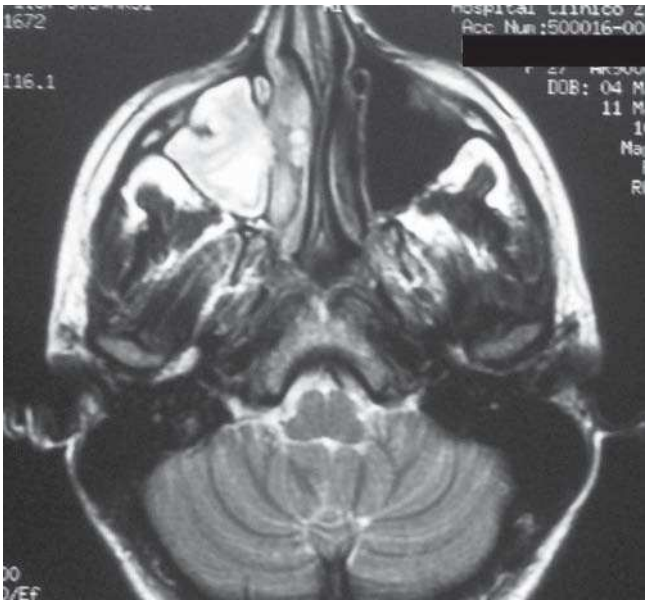

Figure 2 Cerebral magnetic resonance image, T2-weighted sequences: space-occupying lesion in right sinuses compatible with rhinosinusopathy.

maxillofacial computed tomography (CT) (fig. 1) and cerebral magnetic resonance imaging (MRI) in T1, T2, DP and flair weighted sequences (fig. 2) — showed a space-occupying lesion in the maxillary and frontal sinuses, ethmoid cells and nostril of the right side. A rhinofibrolaryngoscopy confirmed collection of mucus and pus in the right paranasal cavities. Oral antibiotic therapy was started with the diagnosis of rhinosinusopathy and optic neuropathy by proximity. This improved the symptoms and normalised the visual field study.

The orbital cavity is composed of 7 separate bones: frontal, zygomatic, maxillary, ethmoid, spheroids, lachrymal and palatine. This anatomical structure is caudally adjacent to the maxillary sinus, frontal sinus and anterior cranial fossa at the level of the skull, and with the ethmoid cells at the medial level. The bone walls between the orbit and the cells (lamina papyracea) and the maxillary sinus (floor of the orbit) are extremely thin, which favours the spread of infections in that area, especially from the ethmoid cells through the venous systems that lack valves. The signs and symptoms shown by the patient translate into different degrees of orbital affectation (external ocular musculature, optic nerve, etc.), as well as into different therapeutic strategies. Rhinosinusitis may lead to local, orbital, intracranial and distant complications. The main orbital complications according to the Chandler classification[2] are inflammatory oedema, orbital cellulitis, subperiosteal abscess, orbital abscess and cavernous sinus thrombophlebitis, even though the latter would be intracranial. Diagnostic studies usually require a high-resolution CT scan of the paranasal sinuses, rhinofibrolaryngoscopy to confirm the inflammatory-infectious nature of the space-occupying process from the anatomopathological point of view, and a careful evaluation of ocular motility and visual acuity. A multidisciplinary approach may be necessary in certain cases and depending on the anatomical structure involved[3] (as was the case in the patient presented), as well as prompt, aggressive treatment to prevent disease progression and to minimise potential sequelae. In this patient, specific antibiotic therapy was sufficient to improve not only the symptoms but also the visual field study results. We conclude by stressing the importance of rhinosinusopathy in the diagnosis of optic neuritis and the need for rapid management to slow its progression and prevent possible complications.

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