Polyradiculitis may affect the autonomic nervous system and be accompanied by pupil abnormalities. Horner syndrome has been described from sympathetic affectation, cranial neuropathies of the 3rd cranial nerve and Adie tonic pupil.[2,3] Our patient presented corectopia within a case of polyradiculitis; the photomotor reflex and lack of a tonic reaction exclude the possibility of an Adie tonic pupil.[4,5] The clinical case with an eccentric pupil is compatible with corectopia. This finding has classically been associated with pretectal involvement[1]; however, it should be regarded as a finding of other processes that may affect the autonomic system. Our case indicates that the most reasonable pathophysiology of corectopia is a lesion of the autonomic fascicle of the 3rd cranial nerve in the midbrain or a peripheral lesion rather than a nuclear lesion.

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Congenital arthrogryposis multiplex and gastroschisis in the same patient

Artrogriposis múltiple congénita y gastrosquisis en un mismo paciente

Dear Editor:

Arthrogryposis multiplex congenita (AMC) is a rheumatic disorder characterised by multiple joint contractures that affect the back muscles as well as the upper and lower extremities. The muscles may be absent, reduced in size and/or number, with replacement by fibrous tissue or fat.[1] The degree of involvement varies from patient to patient and the classical deformity is bilateral and symmetrical.[2] Its prevalence is estimated at 1/3,000 newborns, without distinction by race or gender, and a genetic cause is found in only 30%of cases.[2]

The aetiology of this condition remains unknown and many causal agents have been implicated, particularly viral infections and intrauterine restrictive disorders. The possibility of a vascular rupture phenomenon has also been mentioned as a possible cause.[3,4]

Gastroschisis is a congenital defect of the anterior abdominal wall, lateral to the umbilical cord insertion, usually on the right side. Its aetiology is considered multifactorial and maternal factors such as exposure to teratogens, salicylates and nutritional deficiencies in the early weeks of gestation have currently been mentioned. Any of the previously mentioned teratogenic factors probably causes a vascular alteration of the omphalomesenteric artery (primary defect), which destroys a portion of the abdominal wall, through which abdominal contents protrude into the amniotic cavity.[5-7]

There have been very few published reports of the association between AMC and gastroschisis.[8]

We present a male patient with AMC and gastroschisis, son of a mother aged 36, with 4 pregnancies, who consulted at 36 weeks of gestation to receive delivery care. Weight at birth was 2,400g; head circumference was 29cm; size was 43cm. The mother followed no prenatal care and underwent no obstetrical ultrasound. She denied consumption of tobacco, alcohol or psychoactive substances. The patient was assessed by the paediatric neurology service, which requested brain scans. These were reported as normal and paediatric surgery was carried out to correct the abdominal wall defect, which was corrected on the second day of life. Karyotyping with G "banding" was requested; it reported a normal chromosome complement (46XY). Echocardiography was also requested and was reported as normal.

The association of gastroschisis with other birth defects with a possible aetiology of vascular disruption, such as Poland sequence, intestinal atresia and AMC, has been reported.[8] A recent publication aimed to assess the frequency and type of malformations associated with gastroschisis in 24 different registries of birth defects that contributed data to the International Clearinghouse for Birth Defects Surveillance and Research (ICBDSR); this publication collected information from 3,322 patients with gastroschisis, of which 17 also presented AMC. Although the aetiology of these two conditions is not clear, it has been noted that a vascular disruption phenomenon may be involved. This phenomenon may explain the coexistence of these two conditions in our patient.[9]

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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