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LETTER TO THE EDITORS

Chronic headache with vasospasm treated with nimodipine

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Dear Sirs,

Vasospasm has mainly been implicated in the pathophysiology of headaches associated to reversible cerebral vasoconstriction syndrome (RCVS), subarachnoid hemorrhage and infectious diseases [1, 2].

We report a patient with a chronic headache associated to vasospasm that responded to treatment with nimodipine.

A 49-year-old female patient, seamstress, presented to an outpatient clinic due to an almost daily headache for over a year. She had a bilateral fronto-parietal mild-to-moderate headache (numeric rating scale 2–5), that alternated between pulsatile and tension-type, without photophobia or phonophobia, nausea or vomiting. It started during the morning and persisted during the day. She did not wake up during the night due to the pain. Sometimes the pain was exacerbated by Valsalva maneuvers. Nonsteroidal anti-inflammatory drugs were of no relief. She could not remember the exact onset day and it did not start suddenly. This headache was clearly different from others she had had before that were sporadic and had migrainous characteristics (pulsatile, associated with nausea and photophobia). The patient denied intake of vasoactive medications or drugs. She was under treatment with betahistine for a vertiginous syndrome diagnosed years before and had a past history of surgery to "concha bullosa". Blood pressure was normal. Neurological examination was unremarkable. Brain computerized tomography was normal. She was treated with amitriptyline 12.5 mg/day for 1 month and afterwards amitriptyline 25 mg/day during 2 months with no benefit. Transcranial doppler (TCD) showed a turbulent flow and an increased mean velocity in the right middle cerebral artery (MCA) (155 cm/s) (Fig. 1). She was started on nimodipine 60 mg each 4 h. After 6 days of treatment, the headache remitted. This was accompanied by normalization of the mean velocities. The patient was asymptomatic for 3 weeks. Meanwhile, a brain MRI and a MR angiography were performed, under nimodipine treatment, and were unremarkable (Fig. 2). At this time, nimodipine was withdrawn. In the following day, headache recurred. There was a new increase of TCD mean velocities in both MCAs and distal basilar artery (165 cm/s, 155 cm/s, 155 cm/s, respectively). Reintroduction of nimodipine was followed by headache remission and velocities normalization. After 5 months of treatment, TCD remained normal and nimodipine was suspended. Headache did not recur.

To evaluate a possible infectious cause or a systemic disease we did blood analysis including hemogram, c-reactive protein, ionogram, renal function, antinuclear antibodies, thyroid hormones, HIV and viral hepatitis were negative. Cerebrospinal fluid opening pressure was normal and analyses including immunoglobulinG index, serologies (Brucella, Lyme, syphilis) and neurotropic viruses (VZV,CMV,EBV,HSV1,HSV2,VHH6, VHH7,Enterovirus, Parechovirus,Adenovirus) were unremarkable.

In this clinical case, we could demonstrate an association between vasospasm and the headache.

TCD is a non-invasive technique that is used to monitor vasospasm in patients with subarachnoid hemorrhage (SAH) and RCVS, with a high positive predictive value for vasospasm [3, 4]. It has been suggested that TCD can detect not only dynamic changes in vasoconstriction but also small changes more circumstantially than magnetic resonance angiography [1].

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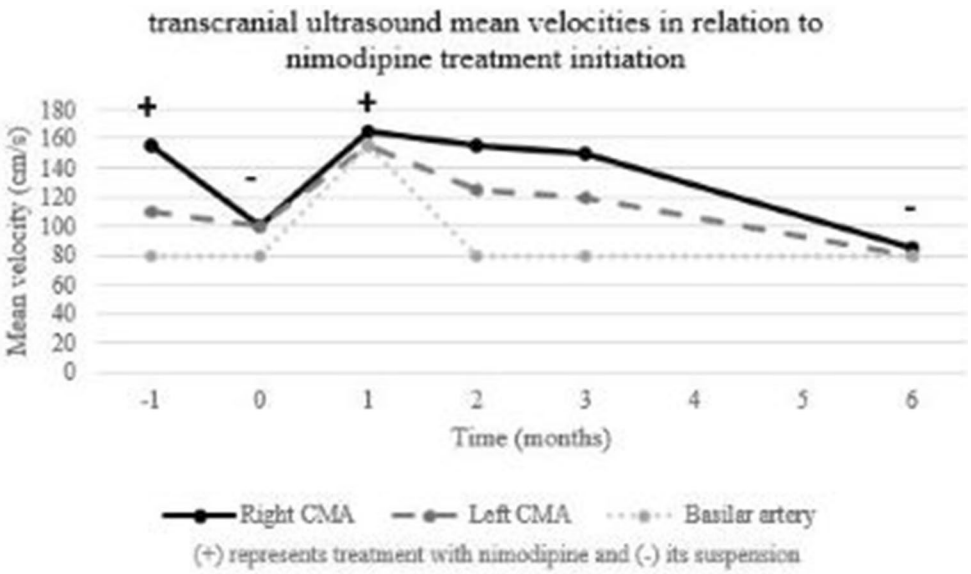

Fig. 1 Graphic showing mean velocities in the middle cerebral arteries and basilar artery during treatment with nimodipine 60 mg 4/4 h. Vasospasm corresponds to a mean velocity in middle cerebral artery > 120 cm/s. Nimodipine was introduced in the − 1 month, suspended in the 0 month and reintroduced thereafter

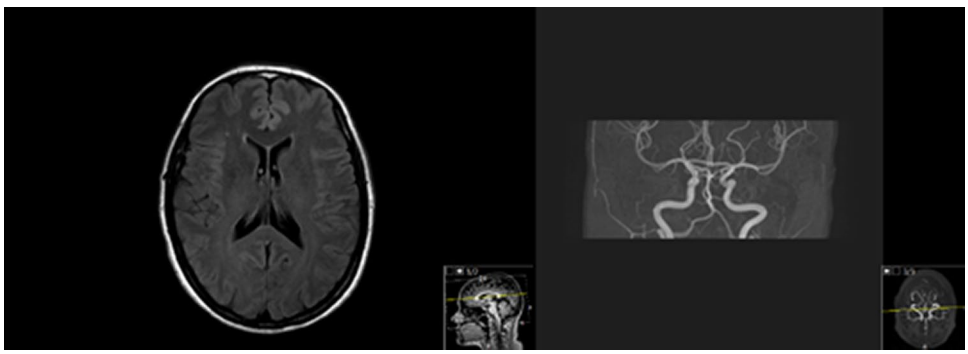

Fig. 2 On the left, a T2/FLAIR sequence image of the brain MRI, which was normal. On the right, MRA with no evidence of vasospasm. The patient was asymptomatic, under nimodipine treatment, when this MRI scan was performed

This patient´s headache does not comply with the ICHD-3 criteria for RCVS as there was no onset with thunderclap and it lasted longer than 3 months [5]. Also, CSF was unremarkable which makes the possibility of an infectious or inflammatory disease unlikely. It also does not comply with the ICDH-3 criteria for new daily persistent headache (NDPH) because the patient could not identify the exact date of symptoms onset. Some heterogeneity has, however, been reported in NDPH. A study that reviewed 71 patients with NDPH according to ICHD-2 described that only 42.4% could recall specific onset day [6]. There is a description in literature of a patient with a NDPH with acalculia that was responsive to nimodipine [2]. The authors attributed the focal symptoms to oligoemia due to focal vasospasm. Nevertheless, CT angiography of the head and neck vessels was normal [7]. They hypothesized that vasospasm could be caused by a very rapid increase in CSF tumor necrosis factor alpha levels, that has been reported to be raised in these patients [7, 8]. Nimodipine could have been useful as it not only inhibits vasospasm but also the production of tumor necrosis factor alpha [7].

Physicians should be aware of the possibility of vasospasm in chronic headaches. TCD can be helpful to evaluate these patients.

Compliance with ethical standards

Conflicts of interest The authors declare no financial or other conflict of interest.

Ethical standards The authors declare that they complied with ethical standards and the study was in accord with the Helsinki Declaration.

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