

2. Semeraro F, Forbice E, Romano V, Angi M, Romano MR, Filippelli ME, et al. Neurotrophic keratitis. *Ophthalmologica*. 2014;231:191–7.
3. Bonini S, Rama P, Olzi D, Lambiasi A. Neurotrophic keratitis. *J Neuroophthalmol*. 2004;24:345–6.
4. Pushker N, Dada T, Vajpayee RB, Gupta V, Aggrawal T, Titiyal JS. Neurotrophic keratopathy. *Eur J Ophthalmol*. 2002;12:60–5.
5. Magone MT, Seitzman GD, Nehls S, Margolis TP. Treatment of neurotrophic keratopathy with nasal dilator strips. *Br J Ophthalmol*. 2005;89:1529–30.
6. Solomon A, Meller D, Prabhasawat P, Jonh T, Espana EM, Steuhl KP, et al. Amniotic membrane grafts for nontraumatic corneal perforations, descemetocoeles, and deep ulcers. *Ophthalmology*. 2002;109:694–703.
7. Lambiasi A, Manni L, Rama P, Bonini S. Clinical application of nerve growth factor on human corneal ulcer. *Br J Ophthalmol*. 2005;89:1529–30.
8. Tan MH, Bryars J, Moore J. Use of nerve growth factor to treat congenital neurotrophic corneal ulceration. *Cornea*. 2006;25:352–5.
9. Park JH, Jeoung JW, Wee WR, Lee JH, Kim MK, Lee JL. Clinical efficacy of amniotic membrane transplantation in the

treatment of various ocular surface diseases. *Eye (Lond)*. 2003;17:989–95.

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Focal leptomenigeal uptake, a new radiological finding in pseudomigraine with pleocytosis[☆]



Captación leptomeníngea focal, un nuevo hallazgo radiológico en la seudomigraña con pleocitosis

Dear Editor:

Headache with neurological deficits and cerebrospinal fluid (CSF) lymphocytosis (HaNDL) is a syndrome characterised by¹ episodes of moderate or severe headache lasting a few hours,² cerebrospinal fluid with lymphocytic pleocytosis and normal neuroimaging results,³ episodes of headache accompanied by transient neurological deficit, and⁴ episodes of headache and neurological deficit recurring less than 3 months apart.¹ Although neuroimaging findings are usually normal,² some exceptions have been reported. We present the clinical case of a patient who met all diagnostic criteria for HaNDL syndrome and exhibited previously undescribed radiology findings.

Our patient was a 30-year-old man with no personal or family history of migraine who visited our department due to somnolence, mixed aphasia, right homonymous hemianopsia, and sensorimotor deficits in the right limbs. He had visited our department in the previous weeks reporting episodes of holocranial headache, sensory alterations,

phonophobia, and photophobia; these symptoms had led to a diagnosis of migraine with sensory aura. Upon arriving at our hospital, our patient underwent a cranial CT scan and a neurosonological study, which yielded normal results. A few hours later he presented fever. A lumbar puncture performed after ruling out other possible focal infections disclosed clear CSF with 50 leukocytes (98% mononuclear), glucose levels of 69.1 mg/dL, and protein levels of 1.45 g/L.

A few days later, he experienced 2 episodes of holocranial headache associated with sensory deficits of the left upper limb which lasted a few hours. An additional lumbar puncture revealed clear CSF with 92 leukocytes (97% mononuclear), glucose levels of 61.7 mg/dL, and protein levels of 0.9 g/L. After that, he remained asymptomatic.

A brain MRI performed during his hospital stay revealed isolated leptomenigeal uptake in the posterior fossa (Fig. 1). Results from a series of blood and CSF screenings (including microbiology and cytology tests, a blood culture, autoimmune tests, and tumour marker tests) were normal. A thoracic-abdominal CT scan revealed no abnormalities.

A follow-up MRI scan performed 3 months later showed that leptomenigeal enhancement had resolved (Fig. 1). To date, 2 years later, our patient has experienced no further episodes.

HaNDL syndrome has been associated with focal slowing in EEG and focal alterations in blood flow as shown by different techniques (Doppler, CT perfusion, perfusion MRI, SPECT). The literature reports isolated cases of brain MRI abnormalities, including alterations in diffusion-weighted sequences at the level of the corpus callosum,³ diffuse leptomenigeal enhancement,⁴ or grey matter alterations associated with CSF enhancement in the temporal and occipital regions.⁵ However, isolated leptomenigeal enhancement in the posterior fossa had never been described.

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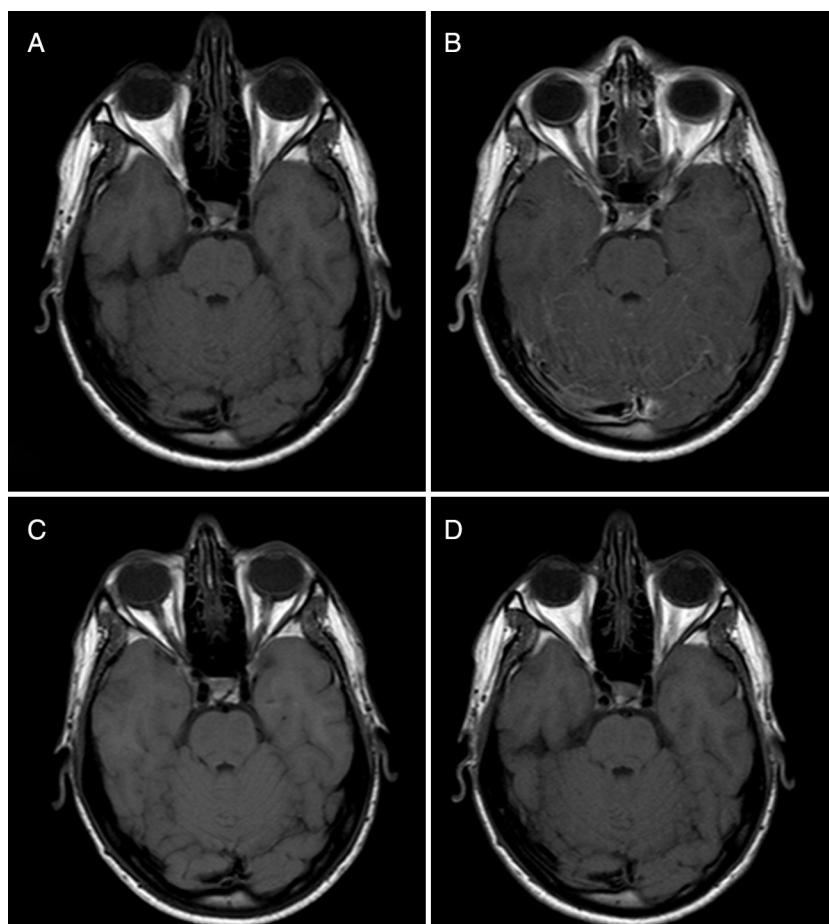


Figure 1 Baseline and follow-up MRI scans (before and after gadolinium administration). The comparison between baseline MR images before (A) and after (B) gadolinium administration reveals slight leptomenigeal enhancement in the superior and vermis folia. Three-month follow-up MR images before (C) and after (D) gadolinium administration evidence resolution of leptomenigeal enhancement.

In our case, differential diagnosis included other diseases that may be accompanied by headache and leptomeningeal enhancement, including infectious diseases (bacterial, fungal, viral, or tuberculous meningitis), tumours (meningeal carcinomatosis, lymphoma), autoimmune diseases (neurosarcoidosis, anti-NMDA receptor encephalitis),⁶ vascular diseases (subarachnoid haemorrhage, vasculitis),⁷ drug-related complications (antibiotics or NSAIDs), and intracranial hypotension.

However, results from microbiology tests and PCR for herpes virus family were negative. Likewise, CSF cytology, tumour marker tests, and autoimmune tests (including antineuronal antibodies) yielded no abnormal results. The thoracic-abdominal CT scan revealed no tumours or pulmonary abnormalities. A brain MRI scan showed no signs of ischaemia or haemorrhage. Our patient denied excessive use of NSAIDs in the days preceding the episode. CSF opening pressure was normal. In conclusion, our patient's clinical progression and the normal results delivered by the aetiological study pointed to HaNDL syndrome associated with previously undescribed brain MRI alterations.

Conflicts of interest

The authors have no conflicts of interest to declare. This study has received no funding of any kind, has not been presented at any congress, nor has it been published in another journal.

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References

1. Headache Classification Subcommittee of the International Headache Society. The International Classification of Headache Disorders: 2nd edition. Cephalalgia. 2004;24 Suppl 1: S9–160.

2. Gómez-Aranda F, Cañadillas F, Martí-Massó JF, Díez-Tejedor E, Serrano PJ, Leira R, et al. Pseudomigraine with temporary neurological symptoms and lymphocytic pleocytosis. A report of 50 cases. *Brain*. 1997;120:1105–13.
 3. Segura T, Hernández-Fernández F, Sánchez-Ayaso P, Lozano E, Abad L. Usefulness of multimodal MR imaging in the differential diagnosis of HaNDL and acute ischemic stroke. *BMC Neurol*. 2010;10:120.
 4. Emond H, Schnorf H, Poloni C, Vulliamoz S, Lalive PH. Syndrome of transient headache and neurological deficits with CSF lymphocytosis (HaNDL) associated with recent human herpesvirus-6 infection. *Cephalgia*. 2009;29:487–91.
 5. Yilmaz A, Kaleagasi H, Dogu O, Kara E, Ozge A. Abnormal MRI in a patient with headache with neurological deficits and CSF lymphocytosis (HaNDL). *Cephalgia*. 2010;30:615–9.
 6. García-Monco J, Gómez Beldarrain M, García Gorostiaga I, Kortazar I. Anti-NMDA Receptor encephalitis presenting as a syndrome of transient headache and neurological deficits with cerebrospinal fluid lymphocytosis (HaNDL). *Neurology*. 2012;78:P04.251.
 7. Smirniotopoulos JG, Murphy FM, Rushing EJ, Rees JH, Schroeder JW. Patterns of contrast enhancement in the brain and meninges. *Radiographics*. 2007;27:525–51.
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Epileptic seizure as a trigger of acute coronary syndrome^{☆,☆☆}



Crisis epiléptica como desencadenante del síndrome coronario

Dear Editor,

Epilepsy has an impact not only on the nervous system but also on a long list of organs and vital systems, including the cardiovascular system. Several studies have investigated, and clearly identified, effects of epilepsy on the heart. The earliest and best-known effect of epilepsy on the cardiovascular system is the increase in heart rate due to adrenaline release and increased circulatory demand; the latter is especially evident in tonic-clonic seizures. In addition to increased heart rate, other phenomena may also be present including myocardial ischaemia, stress cardiomyopathy, and conduction disorders.

We present 2 clinical cases of acute coronary syndrome triggered by epileptic seizures and provide a brief review of current literature addressing the effects of epilepsy on the heart.

Patient 1

Our first patient was 81-year-old woman with a history of arterial hypertension, ischaemic stroke, and atrial fibrillation who was partially dependent for activities of daily living. She visited the emergency department due to a

generalised tonic-clonic seizure which resolved spontaneously. The electrocardiogram revealed ST segment elevation in leads II, III, and aVF. The patient also displayed elevated troponin levels (1.89 ng/mL). These findings were compatible with acute coronary syndrome, which was successfully controlled with medication. The patient experienced no associated complications. During hospitalisation, she experienced an episode of disorientation and a prolonged drop in consciousness. A brain CT scan revealed encephalomalacia in the left parietal region, which was a sequela of a previous ischaemic stroke, and no other alterations suggesting ischaemia and/or haemorrhage. An electroencephalography (EEG) conducted during a seizure revealed markedly slow background activity featuring theta-delta waves and rhythmic spike-and-wave discharges at 3 Hz in the left frontotemporal region. In view of these findings, we administered intravenous diazepam, which controlled symptoms and achieved a normal EEG tracing. Our patient was subsequently treated with levetiracetam dosed at 2000 mg/day, and experienced no further seizures. She was discharged a few days later with a diagnosis of acute coronary syndrome secondary to a partial epileptic seizure with secondary generalisation.

Patient 2

Our second patient was 86-year-old man with a good quality of life who was independent for activities of daily living, had no cardiovascular risk factors, and had a history of colonic polypectomy and parathyroid adenoma. He was attended in the emergency department due to loss of consciousness and amnesia. He was accompanied by his son, who reported that the patient had difficulty understanding instructions and spoke incoherently, although the latter symptom improved gradually. His vital signs were recorded by the emergency services and included slight increases in heart rate and arterial pressure. A cranial CT scan revealed no alterations. While undergoing the CT scan, the patient reported non-specific pain under the right scapula. An electrocardiogram

[☆] This study has not appeared previously in print, nor has it been presented in any meetings or congresses.

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