LETTERS TO THE EDITOR

## Cerebral hyperperfusion syndrome in the emergency department

### *Síndrome de hiperperfusión cerebral en urgencias*

**To the editor:**

Cerebral hyperperfusion syndrome (CHS) is an acute cerebral injury by revascularization of chronic carotid stenosis. It occurs in patients with poor cerebral hemodynamic reserve, with no capacity for autoregulation as permanent vasodilatation of the arteriolar bed becomes chronic in an attempt to compensate for oligohemia.[1-4] The inability for early vasoconstriction in the face of postrepermeabilization hyperflux leads to cerebral edema. Acute clinical presentation may occur days after arterial repermeabilization, with initial symptoms attributable to incipient edema, which without proper management progresses to fatal cerebral hemorrhage.[1-4]

Advanced age, contralateral carotid occlusion and, above all, poor hemodynamic reserve are risk factors that cannot be modified but can be anticipated. High blood pressure (HBP) is the main modifiable factor.[1-4]

We present the case of a 65-year-old hypertensive man with peripheral arterial disease and right middle cerebral artery infarction attributed to subocclusive stenosis of the cervical internal carotid artery (ICA), who 6 months after the stroke was admitted for scheduled carotid thromboendarterectomy (TEA), without in-mediated complications. Four days later, at home, he began a clinical picture with episodes of left hemifacial and brachial clonus with oculocephalic version without affecting consciousness. She was treated with antiepileptic drugs from the emergency room. BP was 110/60 mmHg. Computed tomography (CT) reported as "chronic infarction". Once the seizures were controlled, the possibility of discharge as "late symptomatic epilepsy" was suggested. However, a closer examination of the case revealed discrete sulcus effacement with subtle subarachnoid bleeding and incipient right hemispheric vasogenic edema (Figure 1A). In addition, there was an absence of radial pulse in the limb where the pressure was monitored and a weak pulse in the contralateral upper limb, despite which he demonstrated a BP of 176/83 mmHg. This finally led to admission for BP control, despite which there was clinical worsening and progression of edema during the first 24 hours (Figure 1B). After several weeks, clinical and edema resolution was achieved (Figure 1C).

Cerebral HPS has a variable incidence (0.2-18.9%).[3] A typical clinical triad of presentation has been described, with headache, seizures and neurological focality.[3,4] Furthermore, given the profile of these patients, with diffuse atheromatosis, it is important to ascertain the validity of bilateral brachial BP.

The pre-existence of risk factors invites the systematic performance of anticipatory and control tests, among which transcranial Doppler is one of the most important. It provides information on the preoperative hemodynamic reserve or confirms the diagnosis of CHS after surgery, especially when the flow velocity in the middle cerebral artery is 100% higher than the preoperative baseline value.[5]

Plain CT does not provide predictive information, and may initially be normal, but may also present subtle data that aid clinical suspicion.[3,4] Awareness of the entity in the emergency department and subsequent rigorous CT assessment may facilitate its early diagnosis and improve its vital and functional prognosis.

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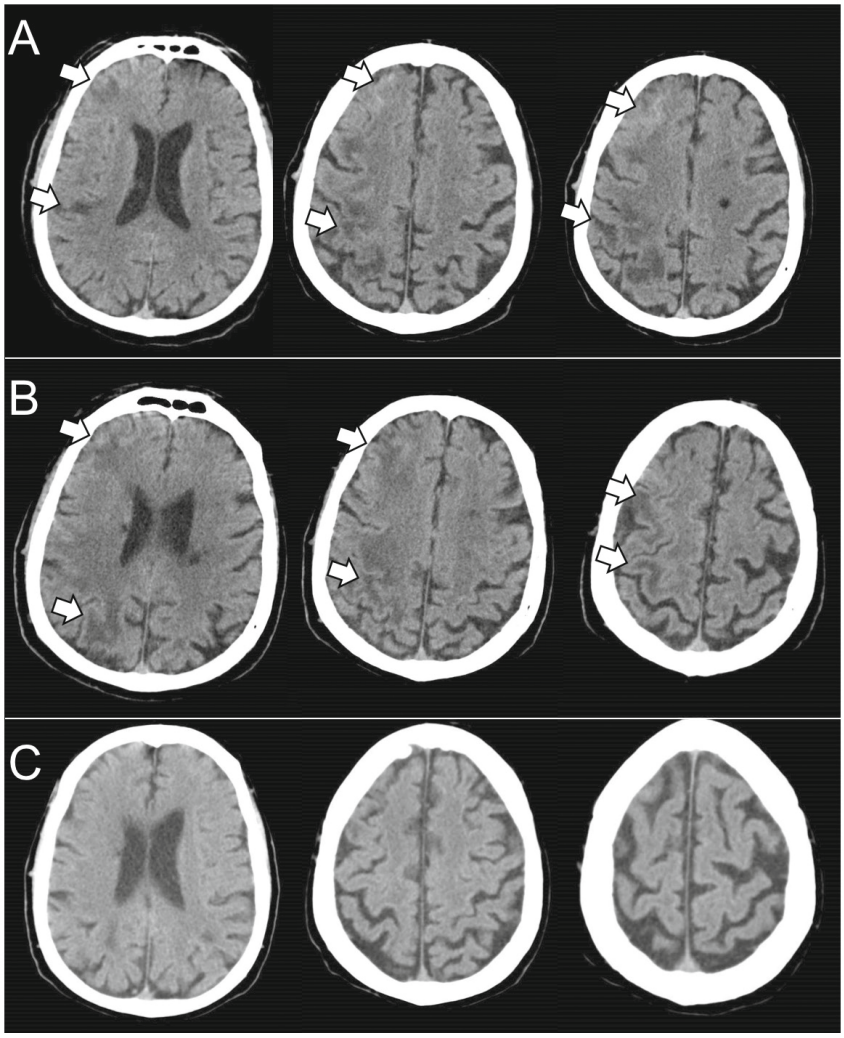


**Figure 1.** Evolution of the patient's brain computed tomography. A: Subtle signs of right frontal and parietal edema (arrows) on arrival at the ED. B: Worsening of edema, 24 hours later, during hospitalization. C: Normalization of neuroimaging at 6 weeks.

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