

from country to country.^{2–5} Some of the confusion arises from the way the term 'brain death', as defined by the Harvard criteria published in 1968, has been translated in Spanish.⁶ We are fully aware of the neuroanatomical differences between whole brain death, brainstem death, and neocortical or cerebral death. The first two definitions of death (based on neurological criteria) are the most widespread, and also the most polemic.^{7–9} All of these definitions revolve around what they consider to be the 'brain' in 'brain death'. On this topic, Spanish law as cited by Iriarte et al. clearly establishes that death must be diagnosed and certified based on "irreversible cessation of cardiopulmonary functions or brain functions".¹⁰ For this reason, we feel that using 'cerebral death' as a synonym for whole brain death is confusing to both health care professionals and society at large. We understand that '*muerte cerebral*' or 'cerebral death' is widely used by Spanish speakers, but the term should not be employed in medicine. If the concepts employed in our definitions are inappropriately explained, the definitions themselves are more likely to be misunderstood.

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J.J. Egea-Guerrero*, J. Revuelto-Rey, E. Gordillo-Escobar

Unidad de Neurocríticos, Unidad de Gestión Clínica de Cuidados Críticos y Urgencias, Hospital Universitario Virgen del Rocío, Sevilla, Spain

* Corresponding author.

E-mail address: juanjoegea@hotmail.com

(J.J. Egea-Guerrero).

Recurrent reversible posterior encephalopathy syndrome with a response to nimodipine[☆]

Síndrome de encefalopatía posterior reversible recurrente con respuesta a nimodipino

Dear Editor:

Posterior reversible encephalopathy syndrome (PRES) is the association of a set of clinical characteristics (headache, epileptic seizures, changes in alertness, visual loss and other focal deficits) and radiological signs indicating reversible changes in white matter, especially in the parietal-temporal-occipital area.^{1,2} It has been linked to high blood pressure and immunosuppressive agents, among other factors. Recurrence is infrequent; a recent study of 25 patients with

long-term follow-up³ reported it in 8% of the study cases.

Various physiopathological mechanisms have been suggested, including vasoconstriction, increased perfusion, and endothelial damage. A study of recurrent cases may help us answer some of the unresolved questions about this syndrome. We present the case of a patient with recurrent PRES episodes which were effectively treated with nimodipine. The patient was a 60-year-old female who came to the emergency department in May 2009. She had a history of smoking, high blood pressure, high cholesterol, and ischaemic heart disease. She presented with headache, drowsiness, nausea, vomiting, and aphasic comprehension which had developed over 2 days, coinciding with an attack of hypertension (blood pressure 220/110 mm Hg). Once the hypertension had been resolved, the patient gradually regained her baseline state and was asymptomatic 48 to 72 hours after being admitted. The patient had been treated with chemotherapy during the preceding 2 years for stage IV ovarian adenocarcinoma. Magnetic resonance (MR) scan showed predominantly parietal-occipital white matter lesions with no restrictions in the diffusion sequences. The lesions disappeared one month later (Fig. 1). Following that, the patient presented with 3 similar episodes in January, April, and June 2010; all episodes coincided with a marked increase in blood

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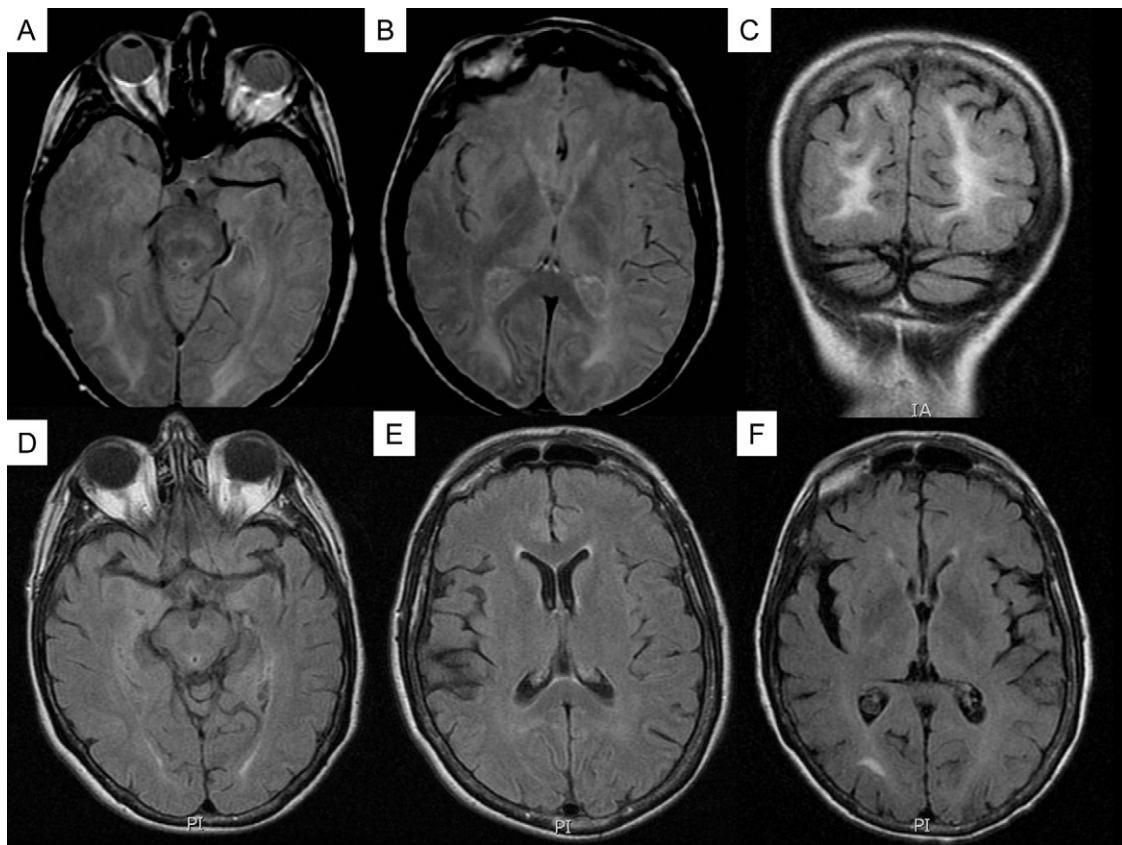


Figure 1 (A–C) Hyperintense parietal-occipital lesions in T2-weighted and FLAIR sequences in the first episode. (D–E) Disappearance of lesions in MR image taken one month later. (F) New right occipital lesion, hyperintense in FLAIR sequence, in a second episode 6 months later.

pressure. The MR scan showed changes similar to those described in PRES, which disappeared once the episode had resolved. The patient discontinued chemotherapy following the first episode and started antihypertensive treatment with beta blockers, ARBs, alpha blockers and diuretics; blood pressure control between episodes was good. Treatment with nimodipine was begun in June 2010; at 15 months of follow-up, no new episodes had occurred.

There are currently 2 hypotheses regarding PRES pathophysiology. The first postulates that high blood pressure would cause arterial vasoconstriction, thereby producing ischaemia and cytotoxic oedema. The second supports there being a disturbance in cerebrovascular autoregulation secondary to uncontrolled high blood pressure. That condition subsequently leads to vasodilation, endothelial dysfunction and vasogenic oedema. The fact that parietal-occipital areas are predominantly involved is probably due to the sparse sympathetic innervation in posterior circulation. MRI findings seem to support the second hypothesis, as they revealed no restrictions in the diffusion sequence. However, cerebral vasoconstriction may be common in PRES, and it may also be related to reversible cerebral vasoconstriction syndrome (RCVS). Ducros et al. describe radiological findings for PRES in 10% of all cases of patients with RCVS.⁴ In turn, vasoconstriction has also been described in some published PRES case studies.⁵ It is not clear whether high blood pressure triggers the episode, or if in fact the episode causes high blood pressure.

Recurrence is uncommon in this syndrome. To our knowledge, there are only 15 published cases of recurrence at present, with a prevalence of 8% according to the series published by Roth et al.³ Various factors may be involved, including high blood pressure, chemotherapy, kidney failure, and eclampsia; however, persistence of these conditions has not been associated with episode recurrence.⁵ For example, our patient left chemotherapy following the first episode. Prior infection has been suggested as a possible episode trigger, but no such infection was present during any of the episodes in the clinical case we present here.

Most published cases of recurrence list 2 episodes, and the case we describe here has the highest number of recurrences recorded in the literature. Favourable response to nimodipine may be due to its role in preventing vasospasm, or caused by its effect on the endothelium. In fact, nimodipine has been put forth as a possible treatment for RCVS,⁶ but no clinical trials have been carried out for this use. Although ours is merely an isolated case, we suggest trying nimodipine as preventive treatment for new recurrent episodes of PRES.

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J.A. Matías-Guiu*, S. García-Ptacek, C.M. Ordás,
A. Marcos-Dolado, J. Porta-Etessam

*Servicio de Neurología, Hospital Clínico San Carlos,
Madrid, Spain*

* Corresponding author.

E-mail address: jordimatiassguu@hotmail.com

(J.A. Matías-Guiu).

Atypical presentation of Terson syndrome: Presentation of a case[☆]

Presentación atípica de un Síndrome de Terson: A propósito de un caso

Dear Editor:

Subarachnoid haemorrhage (SAH) associated with vitreous haemorrhage was first described by French ophthalmologist Albert Terson in 1900.^{1–3} At present, any type of intracranial haemorrhage² accompanied by vitreous haemorrhage is known as Terson syndrome (TS). SAH generally occurs as a result of a ruptured cerebral aneurysm. It is relatively frequent, with an incidence accounting for 5% of all cases of cerebral vascular disease. In contrast, TS is exceptional. The mechanism by which SAH causes intraocular bleeding is a matter of debate.^{1–3} A number of theories state that by means of a direct mechanism, blood is forced through the stem of the optic nerve and into the globe while the SAH is occurring.^{1,3} Other authors suggest that vitreous haemorrhage is the result of venous hypertension and disruption of retinal vessels.³

In TS, most subarachnoid haemorrhages are caused by spontaneous rupture of an aneurysm. The 3 most common locations are the intracranial internal carotid artery, the bifurcation of the middle cerebral artery, and the superior part of the basilar artery. The anatomical location of the aneurysm has not been linked to the side on which TS appears. It has also been shown that anatomical proximity between the aneurysm and the vitreous cavity is not required in order for an intraocular haemorrhage to occur.³

We present a case study of a white male who came to the emergency department due to sudden headache accompanied by vomiting and loss of consciousness. These symptoms resulted from SAH due to ruptured aneurysm, which was diagnosed by computed tomography (CT). The patient

regained consciousness in 24 h, which is very unusual; SAH patients normally remain in a coma for a longer period of time. The aneurysm was embolised several days later using a platinum microcoil. Nothing in the patient's medical history was relevant to the event. He was referred to an ophthalmologist due to loss of vision in the left eye (LE). Findings from the ophthalmic examination were normal, except for a large subhyaloid haemorrhage at the back of the LE that was affecting the macular area (Fig. 1). The haemorrhage was treated with Nd:YAG laser posterior hyaloidotomy. This caused a dense extension of blood in the vitreous cavity which was reabsorbed in 2 months, and the patient's eyesight recovered. This treatment option was chosen because the intraocular haemorrhage did not resolve spontaneously, and because the patient's condition discouraged us from subjecting him to stressful situations such as additional surgical procedures.⁴

It is a well-known fact that the presence of TS is an indicator of SAH severity.¹ Most patients with TS remain unconscious for extended periods of time, as stated above, which is a sign of the magnitude of the condition. However, our patient's case was atypical, as he regained

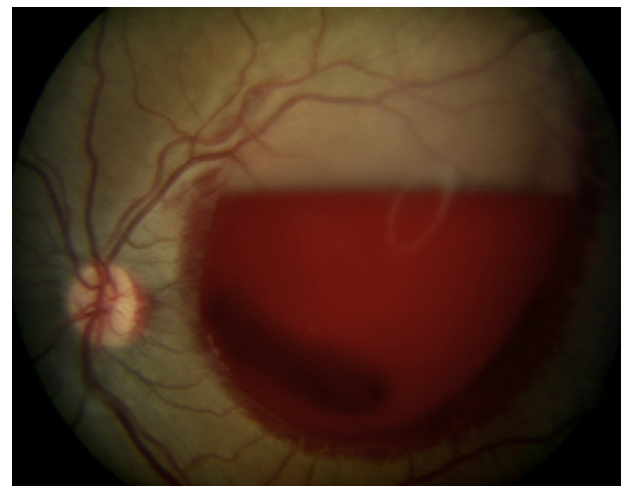


Figure 1 Large subhyaloid haemorrhage of the left eye found in an examination of the back of the eye.

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