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Hemispatial neglect secondary to a traumatic brain injury[☆]



Heminegligencia espacial secundaria a traumatismo craneoencefálico

Dear Editor:

Hemispatial neglect is characterised by systematic omission of stimuli in the contralesional hemifield. This disorder affects such daily activities as getting dressed, eating, reading, or writing. Patients display a decreased ability to attend to, explore, and respond to novel or significant stimuli on the side opposite to the injury. This impairment cannot be attributed to presence of homonymous hemianopsia, which sometimes coexists with hemispatial neglect.¹ This frequent complication of right-sided vascular lesions² has also been described in patients with multiple sclerosis,³ neurodegenerative diseases,⁴ or head trauma.^{5,6} We present a case of hemispatial neglect after severe head trauma.

Our patient is a 52-year-old right-handed man with no relevant medical history who experienced severe head trauma after falling in a suicide attempt. When the emergency medical services arrived on the scene, the patient had a Glasgow Coma Scale score of 6, a bleeding scalp wound, and numerous associated contusions. Brain CT performed when he was admitted to the emergency department displayed a comminuted fracture of the right parietal bone; the largest fragment was depressed. Subdural haematoma was identified in the right hemisphere, with a subarachnoid haemorrhage and the haemorrhagic contusion site in the parietal lobe. We found no midline shift, and the ventricular system was symmetrical and of normal size. Bone fragments

were excised and the parenchyma of the right cerebral convexity was resected. A subsequent brain MRI (Fig. 1) displays a right frontal parenchymal lesion; a right subdural supratentorial haematoma with a discrete mass effect on the right hemisphere and right lateral ventricle and no midline shift; multiple microbleeds in the cortical and subcortical areas of both hemispheres, and around the lateral ventricles; and signal changes in the body and splenium of the corpus callosum.

One month after the traumatic injury, the patient was transferred to our hospital to undergo neurorehabilitation. Upon admission, he was disoriented and showed bradypsychia and psychomotor restlessness. Four months after head trauma, it was possible to conduct a formal neuropsychological examination, according to which the patient was oriented in time, space, and person and displayed preserved language skills. He showed verbal memory alterations (Rey Auditory Verbal Learning Test: short-term memory 35/75, long-term memory 2/15, and recognition 4/15) and executive dysfunction (Letter-Number Sequencing Test: 4/21; Wisconsin Card Sorting Test: number of categories 2/6, number of perseverative errors 32; Phonemic Verbal Fluency Test [3 min]: total number of words 19). During the examination, the patient was observed not paying attention to stimuli in the contralesional hemifield; homonymous hemianopsia had not been observed in the neurological examination. He was subsequently administered a specific neuropsychological testing protocol for assessing visuospatial attention (Bells Test, Ogden Figure Copying Task, Line Bisection Test, Baking Tray Task, and Reading Task); results confirmed presence of left hemispatial neglect. Ten days later, visuospatial tests were administered again; the patient showed slight improvements in task performance (Fig. 2).

Although hemispatial neglect commonly presents in patients with lesions in the right hemisphere, recurrence after head trauma is infrequent. Left hemispatial neglect has traditionally been associated with vascular lesions in the right parietal lobe.⁷ At present, this condition is linked to right-hemisphere lesions, more specifically in the superior temporal gyrus, temporoparietal

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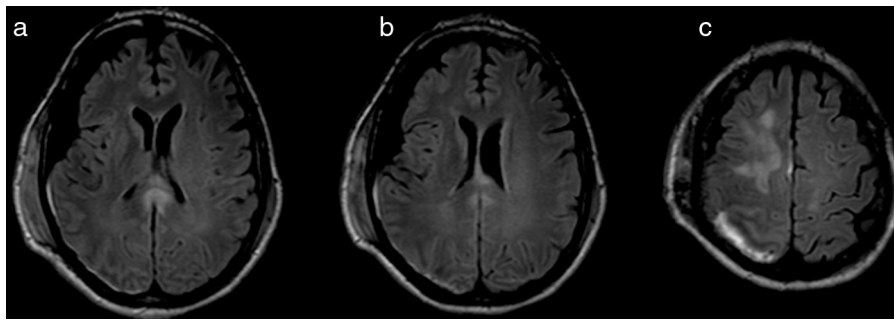


Figure 1 Axial T2-weighted FLAIR MRI sequences. (a) and (b) Resection of the parenchyma of the right convexity and signal changes in the splenium of the corpus callosum. (c) Hyperintensities on the right superior area of the parietal cortex may indicate presence of blood and tissue damage associated with subdural haematoma.



Figure 2 Performance on one of the tests included in the protocol for assessing visuospatial attention (Baking Tray Task).¹³ (a) Healthy subject: cubes are evenly distributed over the board. The dividing line shown in the picture was added intentionally to this figure; it is not visible during the test. (b) Our patient's performance 127 days after head trauma. The image shows a marked tendency to place the cubes on the right side of the board. (c) Performance 10 days after the previous testing session.

junction, and inferior parietal region.⁸ Likewise, several authors have suggested that structural changes in certain areas of the right frontal lobe may cause hemispatial neglect.⁹ Danckert and Ferber⁸ support the idea that left hemispatial neglect is caused by extensive lesions affecting the functional system formed by those brain regions. According to these authors, a focal lesion in the inferior parietal or right superior region is not very likely to cause this condition by itself. Mesulam proposes that contralateral neglect is caused by dysfunctions in the neurocognitive network whose cortical epicentres are located in the frontal cortex (involved in the intentional component of neglect) and parietal cortex (involved in the attentional component).¹⁰ In our patient, hemispatial neglect was probably due to presence of multiple lesions affecting function of Mesulam's proposed neurocognitive network, which plays a critical role in processing extrapersonal space. Those lesions include structural changes in the splenium of the corpus callosum (Fig. 1). Current hypotheses postulate that hemispatial neglect may be explained by a disconnection mechanism between the 2 parietal cortices resulting from damage to the posterior corpus callosum.^{11,12}

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Preganglionic or postganglionic efferent pupillary defect? Clinical versus neuroimaging diagnosis[☆]



Defecto pupilar eferente, ¿preganglionar o posganglionar? Diagnóstico clínico vs neuroimagen

Dear Editor:

Tonic pupil syndrome is a disorder secondary to lesion to the parasympathetic pathway at the ganglionic or postganglionic level.¹ Manifestations include mydriasis with abolished or minimal pupillary reflex (with vermiform movements of the iris due to contraction of normally innervated segments of the iris sphincter, only visible with a slit lamp). Constriction with near effort is present (light-near dissociation) and results from subsequent aberrant reinnervation of the iris sphincter by fibres which were originally destined for the ciliary muscle. This reaction is tonic, that is, slow and sustained. Furthermore, the syndrome also manifests with cholinergic supersensitivity due to postganglionic denervation.²

The origin of the injury may be local (viral ciliary ganglionitis, open or closed trauma, or orbital tumours), or systemic-neuropathic (syphilis, alcohol abuse, diabetes, amyloidosis, or paraneoplastic alterations). However, its most frequent variant is idiopathic tonic pupil or Adie syndrome,¹ which predominantly affects women (70%) in the third to the fifth decades of life. It is caused by a painless degeneration of the ciliary ganglion and posterior funiculi, sometimes associated with a slow viral infection.³

We present the case of a 44-year-old woman with flash blindness and a 7-year history of anisocoria. She reported having suffered head trauma 20 years previously and migraine-like headache that was being treated with flunarizine. The brain magnetic resonance imaging (MRI)

ordered by her neurologist showed a kink at the ostium of the right posterior cerebral artery, originating from the carotid system. Since the kink was touching the superior branch of the ipsilateral oculomotor nerve (OMN), doctors suspected compression of the parasympathetic fibres and referred the patient to our department. We examined her and observed anisocoria due to right mydriasis that was more pronounced under photopic conditions. Direct photomotor reflex and consensual response were abolished in the right eye (RE) but remained intact in the left eye (LE). The near response was normal with a tonic reaction. Extrinsic eye movements were normal with no pupillary response to isolated RE adduction. Biomicroscopy results showed an irregular right pupil with minimal sectoral movements in response to light. Instillation of 0.125% pilocarpine into the conjunctival sac provoked miosis in the RE and no response in the LE (Fig. 1). Bilateral osteotendinous hyporeflexia was also observed. In view of these findings, we proposed the diagnosis of Adie syndrome. Neurologists requested a new gadolinium contrast MRI with thinner slices which revealed displacement of the right OMN without compression (Fig. 2). We therefore opted for watchful waiting.

Pupil abnormalities are examined relatively frequently in neuro-ophthalmology clinics. Finding the lesion location and cause is necessary to assign an accurate diagnosis and subsequently, an appropriate treatment approach. Non-reactive mydriasis may be caused by lesion to the parasympathetic pathway, which can be located at one of several levels: nuclear, preganglionic, ganglionic, and postganglionic.¹

In this case, differential diagnosis includes lesion to the OMN (preganglionic) and Adie's tonic pupil (ganglionic/postganglionic). Furthermore, differential diagnosis was complicated in this case by results of an imaging scan showing an anatomical variant of the origin of the posterior cerebral artery which might compress parasympathetic fibres of the right OMN. A compression injury to this nerve at the point where it leaves the midbrain may affect parasympathetic fibres, causing mydriasis on the affected side due to a preganglionic defect.³

However, clinical signs do not indicate compression injury to the OMN at that level. Light-near dissociation, tonic near-response, and vermiform movements of the iris may indicate tonic pupil. Although the supersensitivity test with pilocarpine has traditionally been used to diagnose tonic pupil, positive results are not exclusive to

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