A Dural Fistula as a Treatable Cause of Cognitive Impairment

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Keywords

dementia, vascular, cerebrovascular disorders, hypoxia-ischemia, brain, stroke, vein of Galen malformations, behavioral neurology, clinical specialty

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Figure 1. Clock drawing test before (A), and after treatment (B).

Case

A 67-year-old man with no relevant medical history was admitted to our hospital because his family perceived behavioral problems that developed gradually in a few days. He was afebrile and normotensive. On neurological examination, his attention was generally intact, and he was cooperative. Nonetheless, his Mini-Mental State Examination (MMSE) score was 12 (losing points on orientation, calculation, and pentagram drawing), he was highly associative, he confabulated, and his visuoconstructional function deficits were observed as illustrated by the clock drawing test (Figure 1A). Furthermore, no focal neurological deficits were found, and symptoms remained stable during admission. The differential diagnosis comprised thalamic infarction (specifically tuberothalamic and paramedian infarction), Creutzfeldt-Jakob disease, and diffuse encephalopathy of various causes.¹ A magnetic resonance image (MRI) scan of his brain showed signal

changes in both thalami (Figure 2A). On the sagittal T2-weighted images, dilated vessels and abnormal flow voids were seen near the vein of van Galen (Figure 2B). Congestion of the deep venous system caused by a dural arteriovenous fistula was considered, and a digital subtraction angiography was performed. A fistula was discovered between the left middle meningeal artery and the vein of van Galen (Figure 2C). It was successfully treated by an endovascular approach (Figure 3C).

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Figure 2. Pre-treatment transversal (A) and sagittal (B) T2-weighted images, and digital subtraction angiography (C). The arrows in A point to signal changes in both thalami, and in B to dilated vessels and abnormal flow voids. The fistula between the left middle meningeal artery and the vein of van Galen is depicted by the arrow in C.

Figure 3. Postprocedural transversal T2-weighted MRI (A), sagittal projection of magnetic resonance angiography (B), and digital subtraction angiography (C).

Although our patient did not improve to full premorbid condition, his cognitive function markedly improved, which was demonstrated by the MMSE (improved to 23) and the clock drawing test (Figure 1B). A postprocedure MRI showed improvement in the thalamic lesions (Figure 3A), and the abnormal flow voids disappeared (Figure 3B). He was transferred to a nursing home for further rehabilitation.

Authors' Note

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Reference

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