Isolated Choroid Plexus Infarction Presenting as Psychomotor Slowing

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A 70-year-old right-handed woman with 9 years of education was brought to our clinic by her husband, who reported that over the past 20 days, the patient had experienced a visible slowing of physical and emotional reactions. She had a history of hypertension and hyperlipidemia. The patient's husband reported that she did not have any history of depression or headache and had a fairly good memory. Furthermore, there was no preceding history of head trauma, encephalitis, antipsychotic or antiemetic medication use, or exposure to toxic substances. However, she was noticeably slower in performing activities which normally would require little thought or effort such as responding to simple questions, preparing meals and clearing dishes from the table, or returning phone calls. Her mini-mental state examination score was 28/30 (two points lost on attention and calculation task). Neuropsychological tests revealed an impairment in frontal/executive function, but not of memory, language, and visuospatial functions. The patient's cranial nerves were intact. Motor examination revealed normal muscle strength and tone, along with normal deep tendon reflexes. Sensory examination results were also normal for all modalities, and the Babinski sign was not elicited. In addition, abnormal behaviors were not seen on the neuropsychiatric inventory questionnaire. Her score on the Geriatric Depression Scale was 2/30.

Extensive serology and hematological tests including complete blood count, blood chemistry, Vitamin B12/folate, and thyroid function tests were normal. Rapid plasma reagin and Treponema pallidum hemagglutination assays were also normal. However, brain magnetic resonance imaging (MRI) demonstrated a focal hyperintense lesion in the right choroid plexus on diffusion-weighted imaging [Figure 1a]. The above lesion was confirmed to be an acute ischemic stroke based on low-signal intensity on the apparent diffusion coefficient map and hyperintensity on fluid-attenuated inversion recovery sequence [Figure 1b and c]. Steno-occlusive lesions were not visible in the intracranial and neck vessels on magnetic resonance angiography. An electroencephalogram was reported as normal. Investigation of the cardioembolic source of stroke included an echocardiogram, which showed normal left ventricular function without structural abnormalities. No arrhythmias were found on 24 h Holter monitoring.

Since the patient exhibited a subacute cerebral infarction and the cardioembolic source was not identified by the examinations, she was given 100 mg of aspirin for secondary prevention of ischemic stroke. However, little is known about the typical symptoms of isolated choroid plexus infarction, and the choroid plexus is associated with cerebrospinal fluid (CSF) production, so we performed a lumbar puncture to evaluate the patient's CSF circulation. The CSF analysis demonstrated a clear color with 0 cells/mm³, 29 mg/dl protein, and 78.5 mg/dl glucose (serum glucose: 110 mg/dl). However, opening CSF pressure was 56 mmH₂O, which was lower than normal range. At follow-up after 4 weeks, she had noticeable recovery with minimal symptoms of psychomotor slowing. Follow-up lumbar puncture revealed a normal opening pressure (130 mmH₂O).

To the best of our knowledge, an isolated choroid plexus infarction presenting as psychomotor slowing has never been reported. The choroid plexus of the lateral ventricle is supplied by the medial branch of the posterior choroidal artery. All previously reported cases were accompanied by multiple infarcts including the thalamus in the posterior cerebral or basilar artery distribution.^[1-3] Furthermore, the patients in previous cases presented with various neurological deficits including motor weakness, sensory loss, and hemianopia. However, it was interesting that our patient complained of a visible slowing of physical and emotional reactions without the above-mentioned symptoms. Unlike previous reports, extensive diagnostic evaluation in the present case revealed an isolated focal choroid plexus infarction only, without significant atherosclerosis in the relevant vessels and cardioembolic sources.

To date, the obvious causal relationship between psychomotor slowing and isolated choroid plexus infarction has not been discussed. However, since CSF is formed primarily in the choroid plexus within the ventricles, it can be speculated that a disturbance in the production of CSF may have occurred after the choroid plexus infarction. Consequentially, we suggested that the symptom of psychomotor slowing, in this case, might be related with frontal lobe dysfunction due

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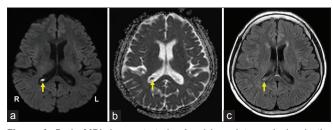


Figure 1: Brain MRI demonstrated a focal hyperintense lesion in the right choroid plexus (a) on DWI and (b) low-signal intensity on the ADC map. (c) The same lesion was hyperintense on FLAIR sequence. MRI = Magnetic resonance imaging, DWI = Diffusion weighted imaging, ADC = Apparent diffusion coefficient, FLAIR = Fluid-attenuated inversion recovery, MRA = Magnetic resonance angiography

to low-CSF pressure. Previous reports have suggested that the mechanism of frontal lobe dysfunction due to low-CSF pressure could be explained by brain sagging and anatomic distortion.^[4,5] In this case, we did not find any clue of intracranial hypotension on the patient's brain MRI, such as pachymeningeal thickening and sagging brainstem. However, considering the symptom onset, the course of recovery, and the result of the neuropsychological test, we assume that her symptom of psychomotor slowing suggesting frontal lobe dysfunction was closely related to the choroid plexus infarction. Further studies with more cases are needed to understand its underlying mechanism.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

REFERENCES

- Liebeskind DS, Hurst RW. Infarction of the choroid plexus. AJNR Am J Neuroradiol 2004;25:289-90.
- Koral K, Dowling MM, Rollins NK. Choroid plexus infarction in a child. Pediatr Neurol 2007;37:452-3.
- Nabavizadeh SA, Tangestanipoor A, Mowla A, Hurst R, Mamourian AC. Infarction of the choroid plexus in basilar artery occlusion. J Neurol Sci 2015;358:467-8.
- Capizzano AA, Lai L, Kim J, Rizzo M, Gray L, Smoot MK, et al. Atypical presentations of intracranial hypotension: Comparison with classic spontaneous intracranial hypotension. AJNR Am J Neuroradiol 2016;37:1256-61.
- Walker L, DeMeulemeester C. Spontaneous intracranial hypotension masquerading as frontotemporal dementia. Clin Neuropsychol 2008;22:1035-53.