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Case Report

Transient visual loss: Transient lesion in the splenium of the corpus callosum

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ABSTRACT

Background: The corpus callosum is the most important pathway enabling the exchange of information between the two cerebral hemispheres. Transient splenium lesions may develop in association with various pathologies and infections. We report a case presenting to the emergency department with transient visual loss and in which a transient lesion was determined in the splenium of the corpus callosum (SCC).

Case report: A 24-year-old woman presented to the emergency department due to sudden onset visual loss. An area of restricted diffusion was determined in the SCC at diffusion magnetic resonance imaging (MRI) of the brain. The patient was admitted to the ward with a diagnosis of SCC lesion. The lesion had resolved entirely at control MRI performed 2 weeks later.

Conclusion: There may be many causes of transient SCC lesions, and patients may present with different clinical manifestations, particularly altered consciousness and rarely visual loss like our present case.

1. Introduction

The corpus callosum (CC) the widest part of the white matter bundle and connects the cortical and subcortical regions of the brain and also the cerebral hemispheres with one another. It thus contributes to the functional combination of sensory and motor functions.[1] Transient lesions of the splenium of the CC (SCC) may appear in association with various causes. Various different agents can give rise to transient lesions of the SCC, such as seizures, antiepileptic drug toxicity and withdrawal syndrome, infective encephalitis, high altitude cerebral edema, excessive use of alcohol, hemolytic uremic syndrome, hypernatremia and metabolic disorders such as hypoglycemia, and case reports concerning these appear, albeit rarely.[2] These lesions are known to cause different clinical symptoms, and particularly altered consciousness.The clinical course, cognitive functions and outcomes in lesions of the SCC are relatively better than those of other lesions.[3] The present case is the first report in the literature of a transient lesion of the SCC causing only transient visual loss without additional neurological findings.

2. Case presentation

A 24-year-old woman presented to the emergency department due to sudden onset visual loss. She reported that she began experiencing visual loss in the form of attacks approximately 1 hour before presentation, that visual loss lasted approximately 30 sec in both eyes, with the eyes open, and then resolved. We learned from her history that she had given birth 3 months previously and had flu-like symptoms and occasional fever for the previous 5 days. She had no history of drug use. Blood pressure at time of presentation was 90/60 mmHg, body temperature 38.5 °C, respiration rate 14/min, heart rate 87 beats/min and oxygen saturation at room temperature 97%. Visual loss attack was witnessed during examination. The patient then suddenly panicked and reported being unable to see us. When asked what she could see, she replied that she could see nothing in front of or around her. She referred merely to a confused, fixed image that she was unable to describe. This resolved entirely after approximately 30 sec. Her Glasgow coma scale score at physical examination was 15, the pupils were isochoric during

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and after attack, and light reflexes were present in both eyes. Neurological examination was completely normal during and after attack. The oropharynx was hyperemic, and pulmonary sounds were natural. Other system examinations were normal. Her electrocardiogram was normal. No anomaly was observed in laboratory values; Hb: 11.8 g/dl, WBC: 6740 μL, Neutrophil: 3430 μL, Lymphocyte: 2160 μL, Platelet: 177000 μL, Glucose: 109 mg/dL, Na: 141 mmol/L and other biochemical values were all within normal limits. Tomography of the brain performed in the emergency department was normal, while an area of restricted diffusion 12 × 8 mm in size in the midline was determined in the SCC at diffusion magnetic resonance imaging (MRI) of the brain (Fig. 1). A lesion not causing marked signal intensity alteration on T1 sequences and a mildly elevated signal on T2 sequences was determined at MRI of the brain (Fig. 2). The patient was administered intravenous (iv) saline solution for dehydration and iv paracetamol for fever in the emergency department. The infectious diseases department was consulted, and meningoencephalitis was not suspected since there is no neurological findings, thus lumber puncture was not done. The patient was assessed by the neurology department, admitted to the ward with a diagnosis of SCC lesion and was given

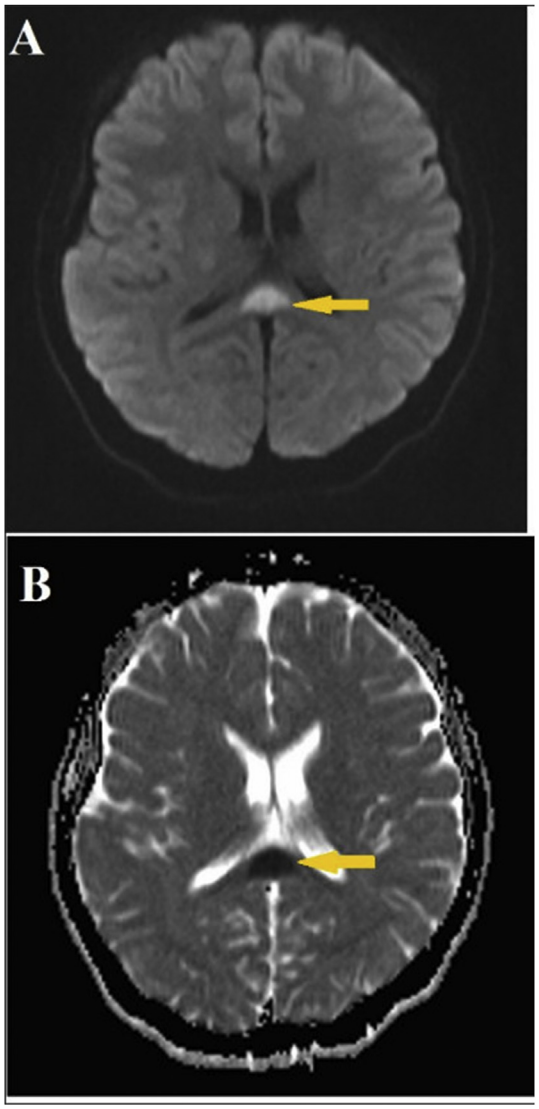

Fig. 1. (A) Axial diffusion-weight (DW) and (B) axial apparent diffusion coefficient (ADC) brain MRI images.

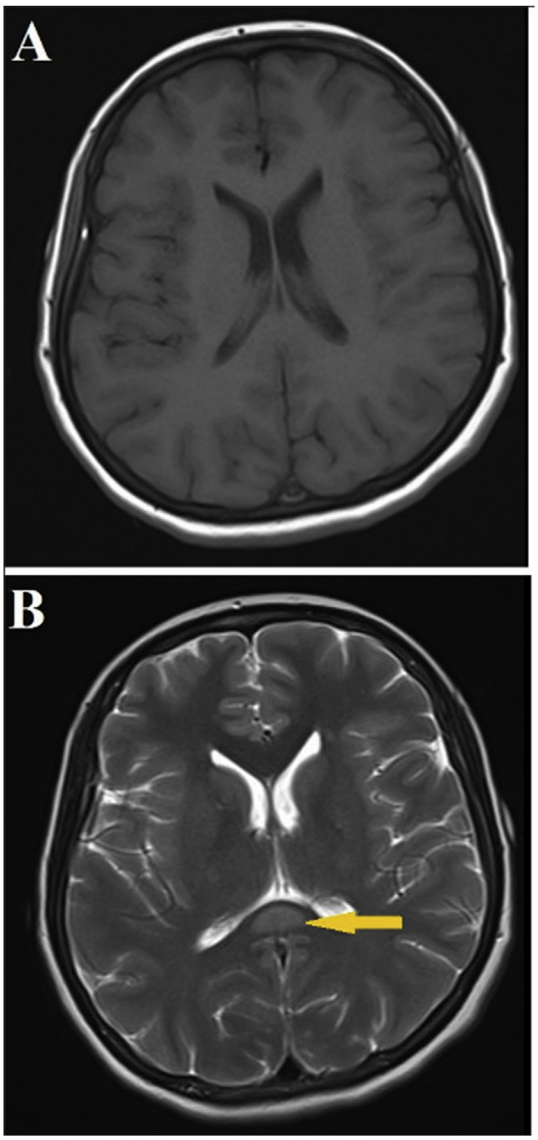

Fig. 2. (A) Axial T1 weighted and (B) axial T2 weighted brain MRI images.

subcutaneous enoxaparin treatment, 40 mg once a day. These symptoms persisted in the form of attacks for approximately 2 hours, but did not subsequently recur. The patient was observed on the ward for 3 days and then discharged when no change was determined at MRI, electroencephalography was normal and symptoms resolved. The patient experienced no further similar symptoms at that the lesion had resolved entirely at control MRI performed 2 weeks later.

3. Discussion

The pathogenesis of transient lesions in the SCC and the reason for their particular location in the SCC are still unclear. It may be that, while the remaining regions of the CC are supplied by the wider carotid artery vascular system, a decreased flow occurs in the SCC since it is supplied by terminal branches of the posterior cerebral artery.[4] Transient lesions in the SCC are very probably develop secondary to focal demyelination associated with antiepileptic drugs and cytotoxic edema and inflammatory changes.[5] CC lesions are known to cause impairments in higher brain functions

such as left apraxia, agraphia, alien hand syndrome, hemifacial metamorphosis, visual and tactile anomia and hearing loss.[6] Since CC infarctions generally occur together with other cerebral ischemic lesions there are no typical symptoms and findings.[1] Lesions of the SCC have been reported to cause paresthesia, hypoesthesia, headache, dysarthria and encephalopathy (altered behavior and confusion).[7] Confusion was determined as the most common symptom associated with SCC lesions in two separate studies investigating the clinical outcomes of such lesions, by Doherty et al. and Park et al.[2,3] Mutism, hallucination, psychosis and impaired hemispheric connections are findings more specific to lesions of the SCC. It is still unclear how these lesions cause mutism and hallucination.[2] Ghosh et al. reported the first case in the literature of transient visual hallucination only without additional neurological findings in association with acute posterior CC infarction.[8] Similarly, in our case there were no additional neurological findings, consciousness was unaffected and only subsequently resolving visual loss in the form of attacks was present. Visual loss attacks occurred in both eyes and with the eyes open, and ocular movements were not affected. During this time the patient was unable to fully see the person opposite or her surroundings, and reported seeing a confusing, fixed image that she could not describe. In contrast to the case reported by Ghosh et al., our patient experienced no hallucination in the form of nonexistent people or objects during these attacks. The visual loss attacks resolved entirely approximately 3 hours after onset and did not recur. In addition, the lesion in the CC also resolved entirely at follow-up 2 weeks later. According to the available databases, ours is the first case of transient visual loss caused by transient lesion of the CCS.

Encephalitis/encephalopathy may be observed in association with the influenza A virus, the most common upper airway infectious agent causing flu-like symptoms, and lesions exhibiting diffusion restriction in the SCC have been reported in these patients.[9,10] The transient nature of the lesion and other clinical and laboratory findings make it possible to differentiate infectious pathologies from other causes. However, it is difficult to estimate the infectious agent with certainty by means of clinical and radiological findings.[10] Due to the flu-like symptoms and fever in our case, the influenza A virus may have caused this lesion as an agent. However, cerebrospinal fluid specimens were not taken since there was no altered consciousness or encephalitis in our patient, and due to the clinical improvement observed. When our patient's clinical findings were compared with case reports in the literature, we thought they might be compatible with post-infectious (most probably postviral) transient lesion of the CCS.

İn conclusion; as discussed, there may be many causes of transient SCC lesions, and patients may present with different clinical manifestations, particularly altered consciousness. However, the reason for the involvement of this location in particular is still unclear. While there may be several causes of visual loss, transient lesion of the SCC is one condition that should be considered at differential diagnosis.

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Conflicts of interest

None declared.

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