Cytotoxic Lesions of the Corpus Callosum: Two Case Reports

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ABSTRACT

The corpus callosum (CC), conventionally divided into four parts (rostrum, genu, body, and splenium), connects the corresponding centres in the right and left cerebral hemispheres. Cytotoxic lesions of the corpus callosum (CLOCC) are unusual clinical conditions and manifest with diverse presentations. We, herein, present two cases of CLOCC. In the first case, a 61-year female patient presented to the Emergency Department (ED) with nausea, dizziness, and syncope. The second case involved a 35-year male patient admitted to the ED with diarrhoea and urinary incontinence. The definitive aetiology of CLOCC could not be determined. The possible causes included systemic, metabolic, toxic, infectious, and seizure-related conditions. Both of the cases exhibited hyponatraemia. Diagnosis involves identifying acute diffusion restriction in the splenium of the CC (SCC) using diffusion magnetic resonance imaging (MRI). CLOCC should be considered in patients with undiagnosed neurological signs and symptoms.

Key Words: Hyponatraemia, Corpus callosum, Cytotoxic lesions, Magnetic resonance imaging.

How to cite this article: Bulut M, Gunhan E. Cytotoxic Lesions of the Corpus Callosum: Two Case Reports. JCPSP Case Rep 2023; 1: 135-137.

INTRODUCTION

The corpus callosum (CC) connects the right and left cerebral hemispheres and consists of thick myelinated fibers. The CC, which is approximately 8 cm long, forms the floor of the longitudinal cerebral fissure and the roof of most of the lateral ventricles. It is responsible for interhemispheric motor, sensory and cognitive information flow. Conventionally, the CC is divided into four parts: rostrum, genu, body, and splenium. The splenium, located posterior to the corpus callosum, contains fibers that interconnect temporal, parietal, and occipital cortices. The splenium plays a pivotal role in visuospatial information transfer, language, reading, calculation, intelligence quotient (IQ), behaviour, and consciousness.

Cytotoxic lesions of the corpus callosum (CLOCC) are uncommon clinical conditions that may arise due to various factors, including systemic, metabolic, toxic, infectious, and seizure-related conditions. Although the incidence of CLOCC is not exactly known, different rates have been reported in several studies (1.1-3%). CLOCC may present with non-specific symptoms including fever, vomiting, diarrhoea, headache, urinary retention, and mild mental changes. In some cases, severe manifestations like profound mental changes and epileptic seizures may occur. The aetiology of CLOCC is not fully known.

Herein, we report cases of two patients with CLOCC who presented to ED with distinct clinical presentations, supported by the relevant literature.

CASE REPORTS

Case 1:

A 61-year female patient presented to ED with symptoms of dizziness, nausea, and syncope. She had been experiencing nausea and dizziness for four days and sought emergency care after a syncopal episode. The patient had no known comorbidities. Initial vital signs were recorded as follows: blood pressure (BP), 100/60 mmHg, puberate (PR), 98 beats/minute; body temperature, 36.2°C; respiratory rate (RR), 16 breaths/minute; and oxygen saturation (SpO2), 99% on room air. Neurological and systemic examinations revealed no abnormalities. The Glasgow Coma Scale (GCS) score was 15. The laboratory results showed WBC to be 4.06x10⁹/L, glucose to be 127 mg/dl, creatinine at 1.01 mg/dl, sodium at 125 mmol/L, and c-reactive protein (CRP) at 260 mg/L. Bacteria and leukocytes were detected in urinalysis. A brain computerised tomography (CT) scan exhibited no abnormalities. The diffusion-weighted magnetic resonance imaging (MRI) revealed a round area of acute diffusion restriction in the Splenium of CC (SCC) (Figure 1a). Apparent Diffusion Coefficient (ADC) mapping demonstrated hypointense characteristics in the corresponding anatomical parts (Figure 1b).

In the ED, the patient received prompt fluid resuscitation for the management of hyponatremia. After the fluid replacement, the serum sodium was 130 mmol/L. The patient was consulted to the neurology department. The patient was prescribed antibiotics, acetylsalicylic acid, and vitamin B12, and was scheduled
for a follow-up appointment at the neurology outpatient clinic. At the follow-up, the patient’s sodium level had normalised to 138 mmol/L, and her complaints had improved.

Figure 1: (a) Nodular acute diffusion restriction in the splenium of the corpus callosum on diffusion MRI; (b) The corresponding anatomical parts exhibited drop-out hypointense character on the apparent diffusion coefficient (ADC) mapping.

Case 2:
A 35-year male patient presented to ED by ambulance, with symptoms of nausea, diarrhoea, and urinary incontinence. The urinary incontinence had been ongoing since the previous day. The patient reported a prolonged history of coughing and was Hepatitis B virus carrier. No medication had been used by the patient. On admission, vital signs were recorded as follows: BP, 120/80 mmHg; PR, 98 beats/minute; RR, 16 breaths/minute; body temperature, 37°C; and SpO₂, 98%. Neurological examination revealed dysarthria with tongue muscle weakness.

The laboratory tests were as follows: WBC, 21x10⁹/L; Lymphocytes, 0.53x10⁹/L; glucose, 144 mg/dl; creatinine, 1.23 mg/dl; sodium, 127 mmol/L; vitamin B12, 165 pg/ml; potassium, 2.9 mmol/L; phosphorus, 0.6 mmol/L; CRP, 286 mg/L; AST, 16 IU/L; ALT, 13 IU/L; ALP, 48 IU/L; total bilirubin, 0.80 mg/dL; and direct bilirubin, 0.35 mg/dL. Urinalysis revealed 31 yeast-like cells and 3 leukocytes. Stool microscopy showed no leukocytes, erythrocytes, or occult blood. Brain CT scan displayed no pathology. Diffusion-weighted MRI demonstrated a focal hyperintense lesion with well-defined margins, indicative of acute diffusion restriction in SCC (Figure 2a). ADC mapping exhibited hypo-intensity in the same lesion (Figure 2b).

The patient was consulted to the neurology department and received anticoagulant, proton pump inhibitors, and isotonic fluid therapy in the ED. Subsequently, the patient was admitted to the neurology service and consulted to the infection department. Acute hepatitis or acute exacerbation of chronic hepatitis was ruled out. Thoracic CT was performed due to the cough in the neurology service which revealed lobar pneumonia. The patient was treated with parenteral ceftriaxone. Replacement therapy was given for electrolyte imbalance and vitamin B12 deficiency. The repeat serum sodium level was found to be 136 mmol/L. After 12 days of hospitalisation, the patient’s symptoms resolved, and he was discharged.

DISCUSSION

CLOCC are usually reversible and associated with various conditions. In a review published in 2011, it was reported that the most commonly associated condition was epilepsy, followed by infection. Various local or systemic infections, including viral and bacterial can cause CLOCC. The prevalence of infection-related cases has increased after the 2019 coronavirus disease (COVID-19) pandemic. In a recently published study, neuroimaging was performed on 167 patients with neurological symptoms out of a total of 3404 COVID-19 patients, and it was reported that the most common (60%) lesions were splenial lesions. During the pandemic, an increased incidence of CLOCC was observed on neuroimaging tests conducted on patients with neurological symptoms. In the literature, hyponatraemia was stated as one of the metabolic causes, and both of these patients exhibited hyponatraemia. Although the precise pathophysiology of CLOCC remained unclear, reversible failures of cellular fluid mechanisms, cytotoxic oedema, focal demyelination, and inflammatory changes were proposed. As a result of hyponatraemia, intramyelinic axonal oedema and infiltration of local inflammatory cells may cause CLOCC. The most common symptom that precedes neurological findings is fever. Other common symptoms include nausea, vomiting, diarrhoea, and headache. Changes in consciousness, seizures, mutism, psychosis, delirium, dysarthria, ataxia, dizziness, and visual disturbances were also reported. No fever was detected in both of these patients, but the first patient had dizziness and fatigue while the second patient presented with nausea, diarrhoea, dysarthria and urinary incontinence.

The primary diagnostic tool was brain MRI, revealing hyper-intensity in T1 and hypo-intensity in T2 sequences, with focal diffusion reduction in ADC mapping. However, MRI may not be performed in all patients in the ED, so diagnosis may be difficult. SCC lesions are categorised into three patterns: (a) a small round or oval lesion in the centre of the splenium, (b) a lesion in the splenium extending into the adjacent white matter, or (c) a lesion in the splenium extending into the anterior portion of the CC (the boomerang sign). Multiple sclerosis, intoxication, posterior reversible encephalopathy syndrome (PRES), acquired immunodeficiency syndrome (AIDS), lymphoma, vitamin B12 and folate deficiency should be considered in the differential diagnosis.
The prognosis of CLOCC depends on the underlying causes. Generally, the outcomes are quite good, especially in isolated splenial lesions. Some MRI studies have suggested complete recovery within one month, often within one week of the clinical improvement.

In conclusion, CLOCCs are rare lesions and there are various aetiologies such as infections, metabolic and electrolyte abnormalities, drug use, epilepsy and cerebrovascular diseases. Therefore, SCC lesions should be considered in patients presenting with undiagnosed neurological signs and symptoms. It is important to note that the diagnosis can be made using diffusion MRI as an advanced imaging technique.

COMPETING INTEREST:
The authors declared no conflict of interest.

PATIENTS’ CONSENT:
Patients’ explicit consents had been obtained to publish their cases.

AUTHORS’ CONTRIBUTION:
MB, EG: Contributed to the design of the study, drafting, and revision of the manuscript.
Both the authors read and approved the final version to be published.

REFERENCES


