Case Report

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Diagnosis of Transient Brain Lesion in the Corpus Callosum Splenium in Emergency Service and Elucidation of Accompanying Conditions

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Abstract

Corpus Callosum Cytotoxic Lesion (CLOCCs) once rarely seen in the literature has been more often diagnosed in emergency services nowadays with widespread use of cranial magnetic resonance imaging (MRI). CLOCCs is defined as a clinical and radiological spectrum disorder. Patient's neurological symptoms usually improve completely within 1 month after the onset of the disease without any sequel. This is generally associated with cytotoxic edema of the splenium corpus callosum. It is important investigate the primary causes that lead to this condition and start the appropriate treatment according to the real diagnosis. We present a case diagnosed as CLOCCs secondary to pneumonia upon admission to our emergency service.

Keywords: Corpus callosum, splenium, transient brain lesion

Introduction

Splenium is the name given to the posterior segment of corpus callosum (CC). In Greek, it means bandage wrapped around a wound. According to anatomical monitoring studies, splenium fiber composition is heterogeneous: while anterior segment contains thin late myelinating fibers emanating from parietal and medial temporal conjunction area, posterior segment contains thick early myelinating fibers connecting primary/secondary visual fields. Majority of splenium fibers are mixed type and they connect hemispheres to each other homotophically (1). Basic physiological effects of CC are conceptualized as stimulation and inhibition. Specifically, while stimulation means tendency of a region in one hemisphere to activate a symmetrical region, inhibition means opposite. Except agenesis of CC, there is not specific pathology it is included (2).

Corpus Callosum Cytotoxic Lesion (CLOCCs), first introduced by Tada et al in the year 2004 with the name of Reversible Splenium Lesion (MERS) is clinically defined as a clinical and radiological syndrome with mild encephalitis/encephalopathy symptoms (3). This phenomenon, later more broadly named as CLOCCs, was defined as a clinical and radiological spectrum disorder (4). Patient's neurological symptoms usually improve completely within 1 month after the onset of the disease without any sequel. However, studies conducted over time have revealed 3 following features. First, condition is not always mild and might be severe rarely. Second, except viral encephalitis/encephalopathy various diseases and conditions also are defined ad CLOCCs. Third, CLOCCs is not always completely reversible. In the light of these 3 features, CLOCCs illustrated by cranial magnetic resonance imaging (MRI) is considered to be secondary to other disorders (5).

In cranial magnetic resonance imaging, it is visualized as hypointense in T1 and FLAIR and hyperintense in T2 sequences (h). In diffusion weighted MRI, CLOCCs displays itself as low diffusion fields. CLOCCs can't be visualized with contrast weighted imaging. Lesions tend to be on the midline and relatively symmetric. Involvement of corpus callosum shows one of 3 typical patterns: 1- a small round or oval lesion in the center of splenium, 2- a lesion that is in the center of splenium but extends from callosal fibers laterally to the adjacent white matter or 3- a lesion that is central to splenium but extends to anterior corpus callosum. These lesions are generally reversible, but not always (6). On the other hand, cranial computed tomography (CT) might be nonspecific. Cytotoxic edema is the most common cause of the etiopathogenesis of such lesions (7).

Pathophysiological hypothesis of CLOCCs which is believed to cause cytotoxic edema through cytokines is as

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follows: first tumor necrosis factor alpha, and cytokines like interleukin-1(IL1) and interleukin-6 cause endothelial damage. Also, tumor necrosis factor alpha and IL1 stimulate astrocytes to produce vascular endothelial growth factor, and this in turn weakens tight connections of brain vascular system and impairs blood-brain barrier (8). In addition, IL1 might induce astrocytes to uptake glutamate, and this triggers glutamate-glutamine cycle and then increases extracellular glutamine levels. Intracellular ATP consumption that causes mitochondrial dysfunction and oxidative stress is induced by activated glutamate-glutamine cycle. As a result, influx of excessive extracellular fluid and Na+ into cells is induced and consequently cellular swelling occurs. These all finally lead to cytotoxic edema (9).

Clinical impression is related to underlying pathology rather lesion itself. So, patients might not only present with encephalopathy symptoms but also with symptoms of central nervous system infection, and nonspecific symptoms such as pneumonia, sepsis, nausea, vertigo, fever or symptoms of metabolic disorders (10). Our patient also presented with nausea and confusion.

Case Report

A 57-year-old male patient with congenital speech and hearing impairment and who has been immobile for 2 years were admitted to emergency service with complains of decreased eating and drinking and change in consciousness for 1 week. According to the information taken from his relatives, he also had nausea, vomiting and cough for last few days, but no fever, no epileptic seizure and use of any antiepileptic drug. He had no history of drug use except angiotensin converting enzyme inhibitor for hypertension treatment. His general condition was bad, blood pressure was 110/70mmHg, heart rate was 90 beats/min, respiratory rate was 20 breaths/min, body temperature was 37.7 0 C. He was confused. Glasgow coma scale was 12. Abnormal findings in biochemistry tests of patient whose whole blood count was normal were as follows: Urea: 69 (N:8-48), BUN: 32 (N:4-23), AST: 148 (N:5-50), ALT: 89 (N:5-50), Total Bilirubin: 2.74(N:0.3-1.2), Direct Bilirubin: 2.74(N:0.3-1.2), LDH: 521 (N:5-248), Sodium: 130 (N:136-146), Chlore: 97 (N:101-109). On the PA chest X ray, there was an infiltrative field in right upper zone (Figure 1). On the thorax computerized tomography (CT) taken afterwards, a consolidated field with air bronchogram was observed in the right lung upper lobe posterior segment (Figure 2). Brain CT was normal (Figure 3). On cranial diffusion MRI, an acute diffusion restriction 5mm in diameter was observed in splenium CC (Figure 4a-b). There was no other lesion in cortex and white matter. Whole abdominal ultrasonography (USG) was normal.

In neurology consultation of the patient, CLOCCs was diagnosed, and as there was no history of seizure disorder,

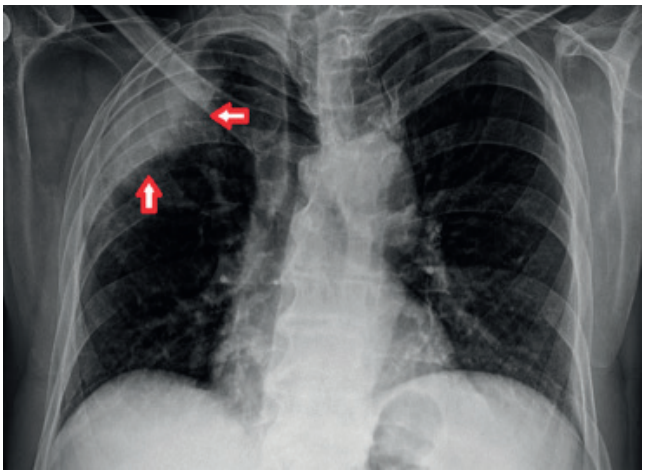

Figure 1. The PA chest X ray, there was an infiltrative field in right upper zone.

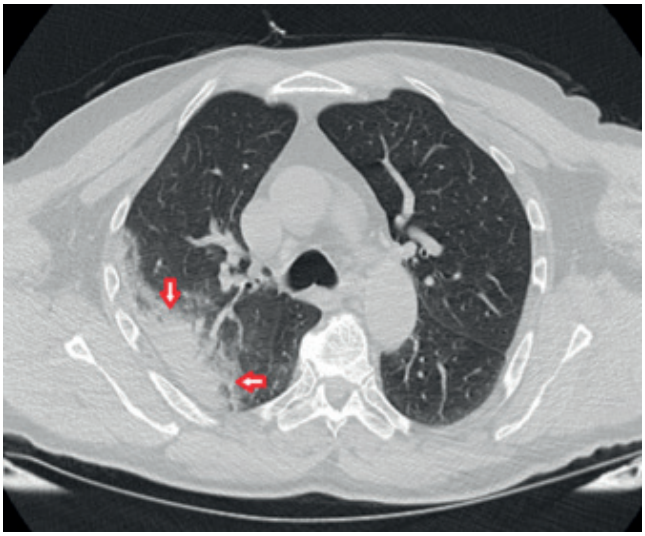

Figure 2. The thorax (CT) taken afterwards, a consolidated field with air bronchogram was observed in the right lung upper lobe posterior segment.

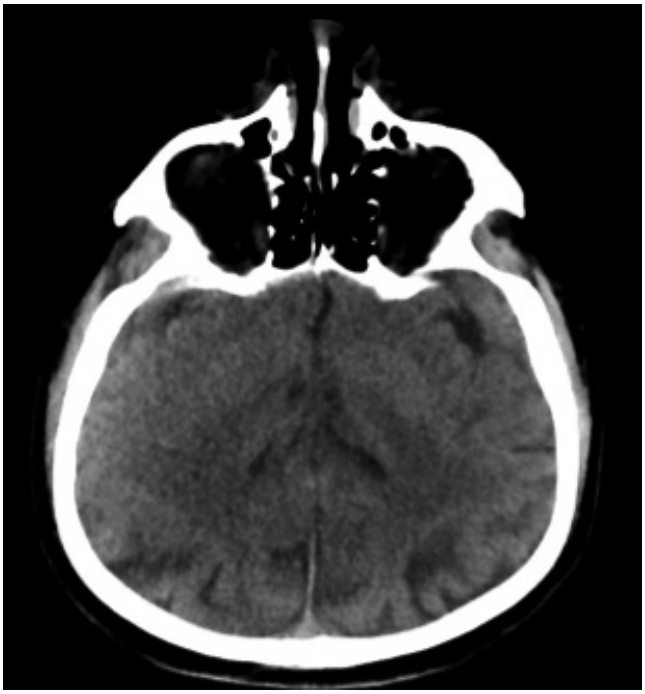

Figure 3. Brain CT was normal.

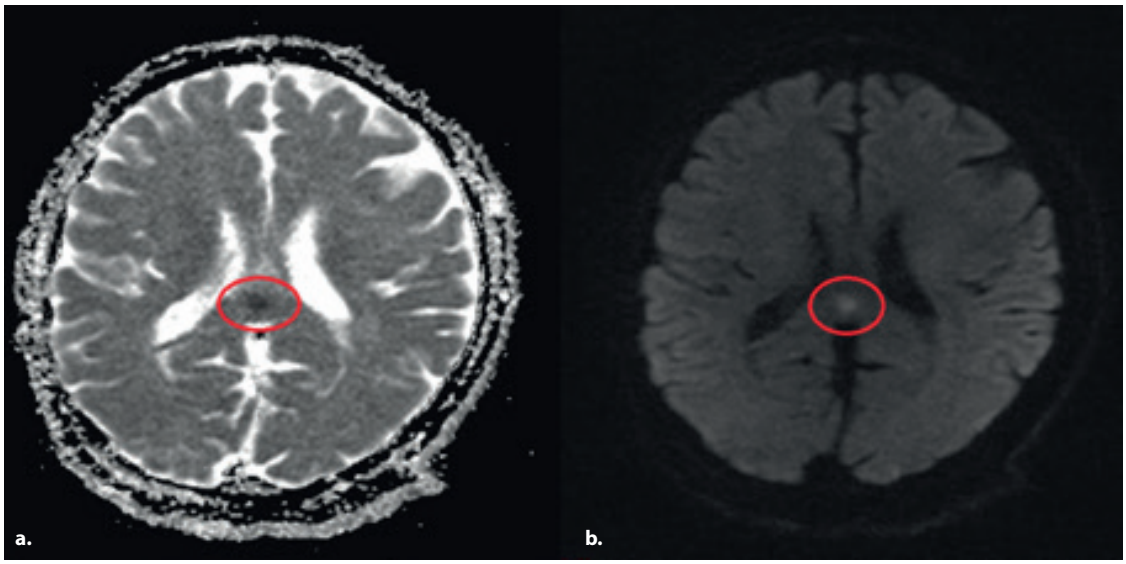

Figure 4a-b. Cranial diffusion MRI, an acute diffusion restriction 5mm in diameter was observed in splenium CC.

or use of antiepileptic drug, neurological pathology was not considered present. It was recommended to investigate metabolic and infectious causes. As whole abdominal USG requested for liver function test disorder was normal, the Department of Gastroenterology was consulted: a Magnetic Resonance Cholangiopancreatography (MRCP) was planned and outpatient clinical control was recommended. After the Department of Pulmonology consultation, patient was admitted to the pulmonology clinic for the treatment of pneumonia.

Discussion

Especially in patients with mixed and nonspecific symptoms that are difficult to diagnose, when CLOCCs is seen one should be alert and underlying primary reasons should be investigated. As Eren et al suggested in their study, CLOCCs might be associated with epilepsy and antiepileptic drugs (43.36%) (7). Although it is the most common cause, our patient had no history of epileptic seizure and no use of antiepileptic drugs. CLOCCs might also be associated with other neurological disorders such as multiple sclerosis (MS), hydrocephalus, subarachnoid bleeding, and ischemic stroke. However, in our patient Brain CT was normal, and history or clinic of MS was not present. In addition, in diffusion MRI there was no other ischemic area. The second most common cause is infective with 33.63% (5). As stated in the study of Şimşek et al, it might be related to central nervous system infections such as meningitis, and encephalitis or immune deficiency (10). Physical examination of our patient did not reveal any finding like fever, neck stiffness or headache. Apart from central nervous system, viral or bacterial infections (influenza virus, rotavirus, measles virus, adenovirus, human parvovirus B19, cytomegalovirus and Mycoplasma pneumonia, Legionella pneumophila, Streptococcus pneumonia and malaria parasites) also might trigger CLOCCs as in our case. Thoracal images of our patient were compatible with bacterial lobar pneumonia. And also, other less common causes (10.62%), metabolism related conditions (5.31%) and high altitude (7.80%) might also lead to CLOCCs (5). Despite further investigations for our patient's liver function test disorder, no liver pathology was detected; MRCP taken after his admission was reported as normal.

Conclusion

Corpus Callosum Cytotoxic Lesion (CLOCCs) once rarely seen in the literature has been more often diagnosed in emergency services nowadays with widespread use of cranial magnetic resonance imaging (MRI). Emergency physician should diagnose this lesion and start the investigation of primary causes and make appropriate consultations for exact diagnosis related to patient's clinic without delay.

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