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A TRANSIENT LESION IN THE SPLENIUM OF THE CORPUS CALLOSUM CAUSED BY INHALATION OF SODIUM HYPOCHLORITE AND MANIFESTED AS A PSYCHOTIC EPISODE

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INTRODUCTION

Transient lesions in the splenium of the corpus callosum (TLCCs) have been defined as reversible isolated lesions that involve the central portions of the corpus callosum splenium, without any other abnormality on initial Magnetic Resonance (MR) examination, and which disappear on follow up studies. The splenium of the corpus callosum (SCC) is a location where transient lesions are rarely observed, but may occur in a number of different situations (Da Rocha et al. 2006). Due to their wide variety of causes, but similar pathophysiological mechanism, these lesions are now called Cytotoxic lesions of the corpus callosum (CLOCCs) (Doherty et al. 2005). Whether the cause is infection, drug therapy, intoxication, subarachnoid hemorrhage, or metabolic disorders etc., they all lead to cytotoxic edema of the splenium (Doherty et al. 2005, Starkey et al. 2017).

We present a patient diagnosed with a Psychotic disorder with delusions due to a known physiological condition, following inhalation of sodium hypochlorite (NaOCl), which led to a TLCC that was detected on MR.

CASE REPORT

A 21 year old female patient, with a previously unremarkable medical history, was admitted to our Clinic for Infectious Diseases, presenting with agitation, confusion, sleeping disorder, loss of appetite and speech disorder. Her family reported that she has been cleaning the house for 5 days, using a bleacher containing NaOCl. The next day she was agitated, had pressured speech, did not want to take food nor did she sleep, had bizarre thoughts and vomited. In the following days, her psychological and mental status worsened, after which she was admitted to the hospital.

During the physical examination the patient was febrile (39°C), agitated, and did not respond to questions. The neurological exam also reportedpointless and repetitive speech, and a lack of cooperation with the examiner. Meningeal signs were negative.The most remarkable finding in her laboratory report was that she had elevated creatine kinases levels-1009 U/L (normal range 22-198 U/L), elevated C-reactive protein 66 mg/l (normal range 0.0-3.3 mg/L), aspartate aminotransferase 95 U/L (normal range 8-48 U/L), alanine aminotransferase 62 U/L (normal range 7-55 U/L), and lactate dehydrogenase 700 U/L (normal range 140-280 U/L).

She had no history of drug use. The urine drug test was negative. Lumbar puncture, brain CT, CT angiography and chest X-ray were all unremarkable. Serological tests for viruses (including SARS-CoV-2) were all negative. Brain MRI performed about 7 days after the onset of symptoms showed an oval well–defined T2-weighted and fluid-attenuated inversion-recovery (FLAIR) hyperintense lesion in the central part of the SCC, with restricted diffusion, and without contrast enhancement (Figure 1). These findings were suggestive of a CLOCC. The control MRI, performed a month after the first one, showed complete regression of the lesion.

The patient was later diagnosed with “Psychotic disorder with delusions due to a known physiological condition”. During her time at the hospital her mental status improved, and after two weeks she was discharged from the hospital. She took the therapy prescribed by a psychiatrist for about two years, after which she did not take any more medication, and is now about to successfully graduate from college.

DISCUSSION

Despite the large number of possible etiologies, the exact pathogenesis of reversible splenial lesions is not well understood (Da Rocha et al. 2006). Some etiological hypotheses are the presence of elevated levels of interleukin-6 and cross-reaction to viral antigens. Vasogenic or cytogenic edema has been proposed as a possible reason for infective, post-infectious, and post-seizure involvement (Udaya et al. 2015).

The most significant component of almost every bleaching substance is NaOCl, which is safe for topical

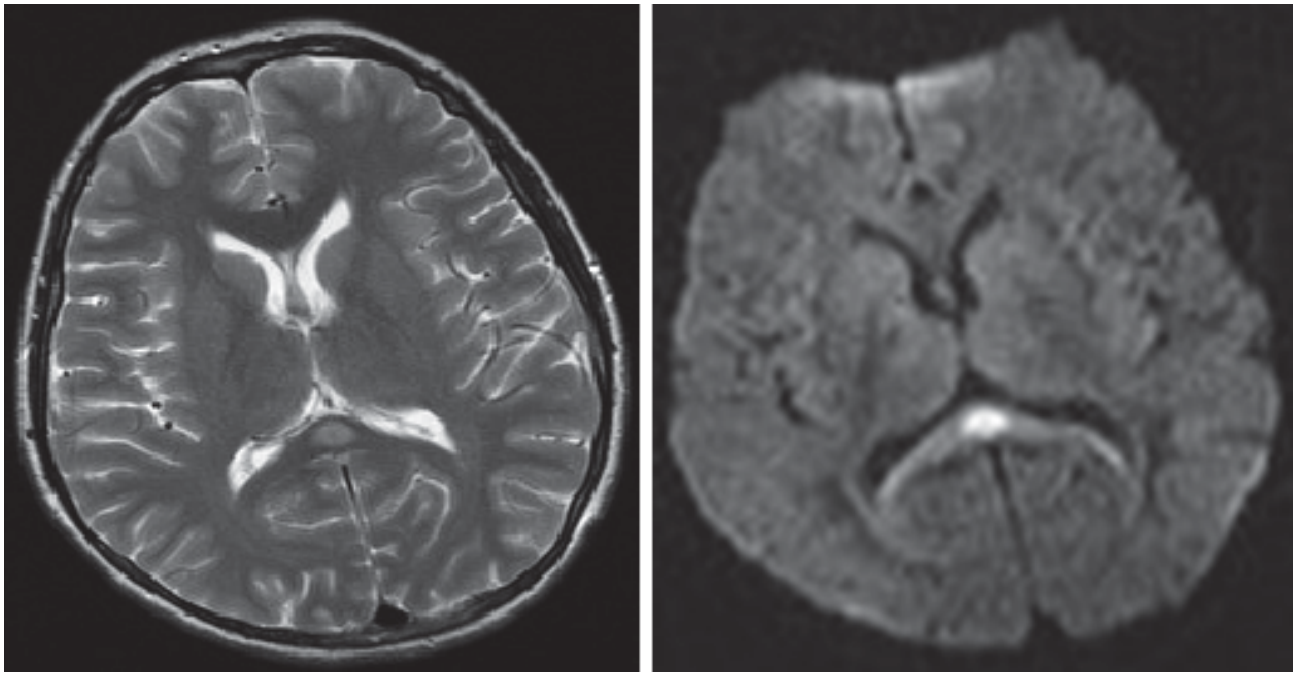

Figure 1. The axial T2-weighted magnetic resonance image (A) show a focal hyperintense lesion in the central part of the splenium of the corpus callosum, with hyperintense signal on the axial diffusion-weighted image (B) suggestive of cytotoxic edema

use, however it has been proven that exposure to NaOCl beyond its topical use, whether it is by ingestion, inhalation, deposition in tissue or injection into the bloodstream, is associated with significant health risks due to its strong oxidizing properties. Mixed with water, NaOCl forms highly reactive HOCl (hypochlorus acid), that generates superoxide radicals that cause oxidative injury and cell death (Peck et al. 2011). HOCl causes adenosine triphosphate (ATP) depletion (Whiteman et al. 2002), and ATP is crucial for keeping cell homeostasis. Without sufficient amounts of ATP, the Na/K pump can not maintain the continuous extrusion of Na from the intracellular compartment. The influx of Cl^- follows via the chloride channels, which leads to increased intracellular osmolarity. This increased osmolarity causes an influx of water into the cell through the aquaporine channels (AQP) (Liang et al. 2007). The larger density of AQP1 and AQP4 channels and the higher density of glutamate receptors could make the SCC very vulnerable to the influx of water into the glial cells, and are therefore a vulnerable region for the development of cytotoxic edema (Blaauw & Meriners 2020). It is now known that the corpus callosum, and particularly the splenium, are vulnerable to cytokinopathy, due to its high density cytokine receptors, glutamate and other excitatory amino acid receptors, toxin receptors, and drug receptors. This higher density leads to a tendency for cytotoxic edema of the corpus callosum to develop when cytokinopathy occurs (Starkey et al. 2017).

In a study of 22 patients receiving AED therapy, Prilipko et al. also suggested that cytotoxic edema could be the reason for the formation of TLCCs (Prilipko et al. 2005). NaOCl poisoning also affects other tissues, which can be appreciated in our case by the elevated liver enzymes, elevated CK and LDH levels, as well as electrolyte disbalance (Peck et al. 2011).

Our patient did not present with the more common symptoms of a splenium lesion (Park et al. 2014) but symptoms which would later be recognized by a psychiatrist as psychotic disorder with delusions due to a known physiological condition (F06.2). As far as we know a CLOCC lesion with such symptoms has not been presented before.

CONCLUSION

The exact function of the splenium is not yet known, but for a number of neurological and systemic pathological processes, and also in our case, it appears to play a role in consciousness. Presenting patients with new and uncommon symptoms with reliable radiological confirmation, is a way towards understanding the functions and connections in our brain.

Contribution of individual authors:

Svjetlana Mujagic: conceptualization, literature survey, diagnosis and follow-up of the patient, writing manuscript, supervision.

Mirza Halilcevic: literature survey, writing manuscript, data acquisition.

All authors approved the final version.

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