Hypoxic Corpus Callosum Lesion after Cardiac Arrest with Good Prognosis

Esra Eruyar, Sule Bilen, Yesim Sucullu Karadag, Neşe Oztekin, Fikri Ak

Abstract

Hypoxic ischemic damage of the corpus callosum after cardiac arrest is a rare condition. Lesions of the splenium of the corpus callosum after hypoxia are bilateral and lead to poor prognosis. Herein, we present a case with good prognosis after cardiac arrest with bilateral lesions of the splenium of corpus callosum.

Key Words: corpus callosum, splenium, cardiac arrest, good prognosis, diffusion weighted tensor imaging

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INTRODUCTION

Global hypoxic-ischemic injury is common after cardiac arrest. Hippocampus, cortex, striatum and cerebellum are most sensitive to hypoxic/ischemic injury[1,2,3]. Although ischemia of the corpus callosum has been reported in up to 9% of ischemic strokes, the majority of these unilateral lesions occurred in conjunction with posterior cerebral artery (PCA) territory infarcts[4].

The corpus callosum (CC) appears to be relatively resistant to ischemic injury[5,6]. In contrast, MRI lesions of the corpus callosum have been reported in situations of trauma, inflammation, malignancy, metabolic disorders, infections and toxic exposure.

The most common clinical presentation of CC lesions (of any cause) is known as altered mental status[7]. Interhemispheric sensorimotor visual information and fusion events are performed by the splenium of corpus callosum (SCC); also interhemisferic verbal information is provided here. All these connections are assumed to be provided through special callosal cord[8,9,10]. SCC lesions after cardiac arrest are usually bilateral and lead to poor prognosis[1]. Here we describe the clinical and MRI findings of a patient with bilateral lesions of SCC with good prognosis after cardiac arrest.

CASE REPORT

A 46 year-old dyspneic female patient was admitted to the emergency room and cardiac arrest developed soon after. Cardiopulmoner ressusitation was performed for about 12 minutes.

She has undergone cardiac arrest again in the emergency room and resuscitated again for about 10 more minutes. Her body temperature was 36°C, blood pressure was 165/78 mmHg, heart rate was 130 beats/min upon her arrival. She was diagnosed as Samter's syndrome previously and had been under inhaler and rarely oral

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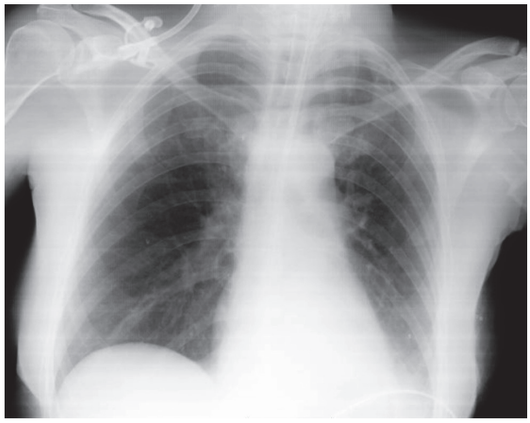

Figure 1. Normal Lung X-ray.

steroid treatment. Her medical history revealed influenza like symptoms 15 days before admission.

Laboratory analyses were as follows: Wbc: 10.46/μl (4.4-11.3), neu: 3.00/μL (2.1-6.1), lym: 7.36/μL (1.3-3.5), hgb: 13.0 g/dL (10,8-14,9), serum glucose: 149 mg/dl, BUN: 20 mg/dL (10-50), creatinine: 0.75 mg/dL (0,5-1,2), AST: 52 U/L (< 35), ALT: 37 U/L (< 35), Na: 137 mmol/L, K: 4.1 mmol/L. Blood, urine and tracheal culture were normal. Her lung X-ray was also normal (Figure 1) but viral pneumonia treatment (oseltamivir phosphate) had been started for flu prophilaxies in emergency room but whenever we learned about her asthma history we stopped it. 1 mg/kg iv steroid and inhaler steroid treatment were administered. In echocardigraphy no cardiac pathology

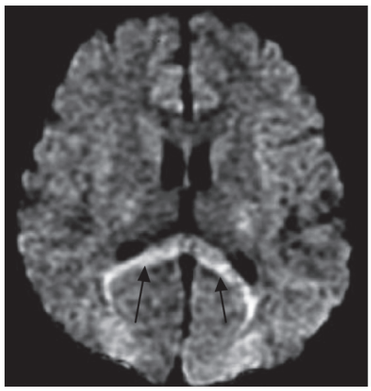

Figure 2. Diffusion restriction in splenium of corpus callosum and in occipital lobes (black arrows).

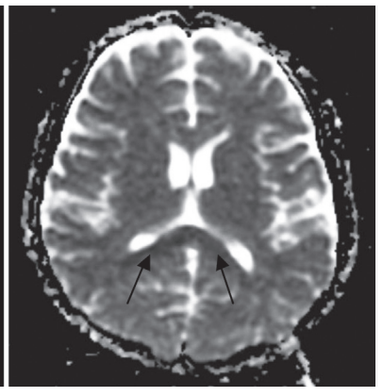

Figure 3. Hypointensity in splenium of corpus callosum bilaterally on ADC image (black arrows).

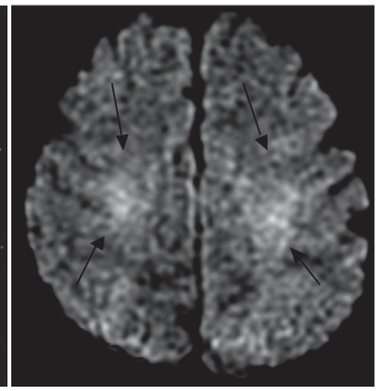

Figure 4. Hyperintensity of bilateral vertex on diffusion weighted images (black arrows).

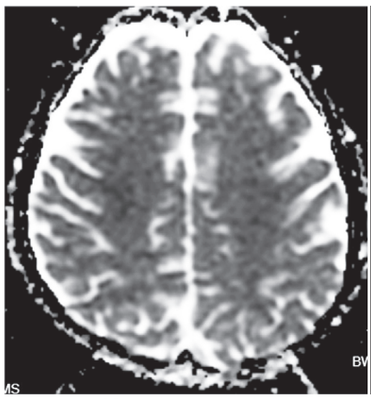

Figure 5. Hypointensity in bilateral vertex on ADC image (black arrows).

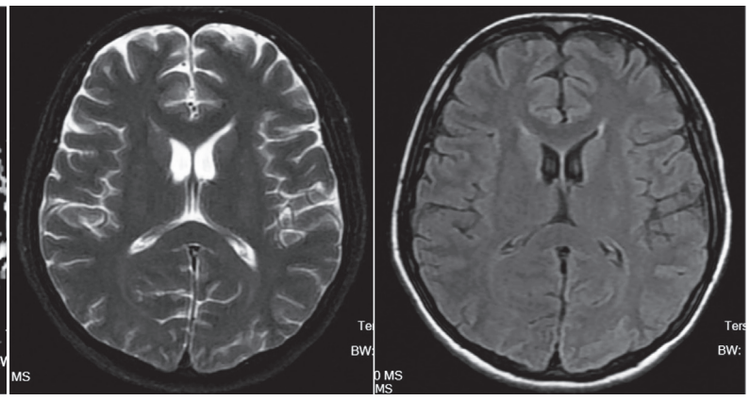

Figure 6,7. Isointens appearance of splenium on T2 image and T2 Flair image.

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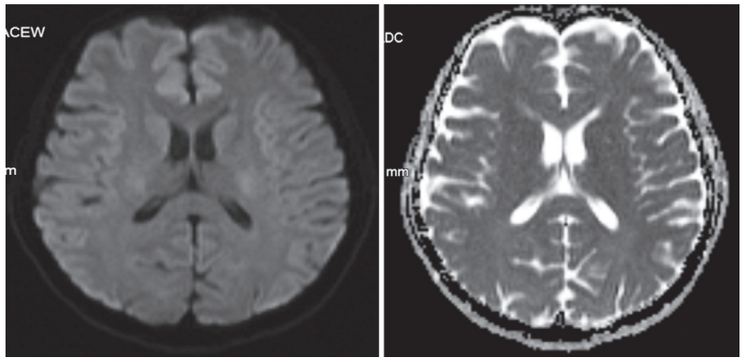

Figure 8,9. Control diffusion MRI and ADC imaging were normal after 3 months.

was observed.

The patient was intubated and connected to the mechanical ventilator. In initial neurological examination she was unconscious and unresponsive to verbal and painfull stimuli with intact brainstem reflexes.

After 72 hours, she was still unresponsive to verbal stimuli but gave response to painful stimuli and Glasgow Coma Scale Score (GCSS) was 6. Brain tomography was normal. The patient was diagnosed as allergic asthma and anaphylaxis led to respiratory and cardiac arrest. She was extubated upon improvement of respiration spontaneously and the recovery of consciousness on the 7th day of hospitalization. She was dysphasic and had blurred vision. In repeated neurological examinations the patient was conscious and cooperative with visual acuity of finger counting at 1 meter but could not distinguish colors and objects. The distal upper extremity muscle strength was 4/5, proximal was 5-/5, lower extremity muscle strength and other neurological examination findings were normal. MRI which was performed 13 days after the cardiac arrest revealed diffuse diffusion restriction in the splenium of corpus callosi bilaterally, in occipital lobes and in cortical gray matter on vertex level (Figure 2, 3, 4, 5). Splenial lesions were isointense in the T2 and Flair sequence (Figure 6, 7). The present lesions were thought to be associated with hypoxia.

The patient's vision almost completely recovered in her control neurological examination after 3 months. She could distunguish colors and objects. Dysphasia and muscle weakness had improved. Lesions had completely disappeared on control imaging after 3 months (Figure 8, 9).

DISCUSSION

The hypoxic ischemic lesions of corpus callosum are rare. MRI lesions of the CC have been seen in trauma, inflammation, malignancy, metabolic disorders, infections and toxic exposure[6]. Antiepileptic drugs, high-altitude disease and encephalitis-encephalopathy cause reversible MRI lesions with transiently restricted diffusion in the SCC. Transient focal lesions of splenium of corpus callosum can be seen as a component of many central nervous system diseases, including antiepileptic drug toxicity. The conventional magnetic resonance (MR) findings of the disease are characteristic and include ovoid lesions with high signal intensity at T2-weighted MRI in these cases. In our patient we excluded the causes above. She did not have the history of antiepileptic drug use. Hypoxic SCC lesions are usually bilateral and have poor neurological prognosis. Matt et al. reported poor prognosis in patients with bilateral SCC lesions after cardiac arrest. Only the prognosis of a patient with unilateral lesion was good[1]. But on the contrary, our case had have good prognosis, lesions were localized in the central part[11,12,13]. For this reason we thought that our patient had stayed as hypoxic for very short time.

Anatomical tracing studies report that the fiber composition of the splenium is heterogeneous. Anterior part of the splenium contains thin late-myelinating fibers from parietal and medial temporal association areas, while the posterior part include thick early-myelinating fibers linking primary/secondary visual areas. Splenium fibers are mostly resiprocal and connect the hemispheres homotopically, while others link various cortical

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areas[15,16]. When higher-order visual areas are inactivated, the suppressive surround of neurons in lower-order areas weakens, suggesting a role for top-down connections in mechanism. The heterotopic splenial fibers especially those between association and primary visual areas, could mediate such feedback[17,18,19]. In our case; vision loss can be explained with fusion events that can not be made by hypoxic changes in the splenium.

During the acute period after diffuse cerebral anoxia, the results of conventional MRI and CT scanning may be normal. Diffusion-weighted images (DWI) showed earlier and more extensive abnormalities than did conventional MR images after global cerebral anoxia. In addition, cerebral abnormalities, as seen on DWI, followed sequential changes, with predominantly gray matter abnormalities during the acute and early subacute periods, white matter abnormalities during the late subacute period, and a return to normal during the chronic stage. The diffusion abnormalities correlate well with known histologic abnormalities and underlying pathologic mechanisms occuring with global cerebral anoxia[20,21]. DWI showed bilateral SCC and occipital lesions in subacute period and returned to normal during chronic period in our case. Conventional MRI was normal.

Imaging characteristics of hyperintensity on DWI and hypointensity on ADC maps suggest ischemia, although the actual mechanism of the splenium lesions is yet to be clarified. Several features argue against ischemia as the lesion etiology in these fatalities: 1- lack of involvement of neighboring vasculer areas; 2- the delayed appearance of the splenium lesions in early and late MRIs were available lack of involvement of neighboring vasculer areas; 3- the relative resistance of the corpus callosum to hypoxic-ischemic injury (relative to cortex, hippocampus, striatum and cerebellar purkinje cells); 4-symmetric midline-spanning lesions do not reflect a single vascular area[1].

Splenium findings can be explained by the early Wallerian degeneration from diffuse cortical neuronal death from global hypoxic-ischemic injury, especially the highly susceptible transcallosal pyramidal neurons in cortical layer three. Wallerian degeneration of central nervous system occurs most commonly on a longer time scale (weeks), although early subacute changes (over days) reflecting Wallerian degeneration have been reported following brain ischemia[22,23]. Another theory is cytotoxic edema. Normally, the SCC exhibits irregular water component and low homogeneity. Whereas myelin sheats in the SCC exhibit a relatively high water component, and the SCC is more susceptible to cytotoxic edema than other brain areas[24,25,26]. Positron emission tomography, single-photon emission tomography, MR spectroscopy and pathological examination may be helpful in the diagnosis of diffuse cerebral anoxia in the future[20]. In our case; the SCC lesion on DWI performed in 13 days after cardiac arrest explain with cytotoxic edema rather than ischemia. Disappearance of lesions in the control images supported this opinion.

Late bilateral splenium DWI lesions after cardiac arrest may be a specific predictor of poor neurological outcome. Recovery from coma after cardiac arrest may involve many factors related to the patient's premorbid condition and the circumstances of the arrest and resuscitation. Prognostication of neurological function after brain injury remains an important clinical challenge. First guideline about neurological prognostication for coma after cardiac arrest was released by the American Academy of Neurology. In this evidence-based guideline, MRI studies had limited supportive literature among the numerous variables reviewed. Thus, MRI findings are not considered in the algoritm, demonstrating that, we can not just decide for prognosis with only MRI[27].

In our case good prognosis might be explained with higher GCS after 72 hours, intact brain stem reflexes, absence of myoclonic seizures, early intervention, absence of premorbid diseases, vital signs which were stable during arrest and being stayed as hypoxic for very short time.

In our case; DWI showed earlier and more extensive abnormalities than did conventional MR images after global cerebral anoxia and MRI studies had limited contribution in estimation of prognosis.

To conclude we deduced that neurological examination repeated after 72 hours of the event along with DWI will have a more prognostic value. Positron emission tomography, single-photon emission tomography, MR spectroscopy and pathological examination may be helpful in the diagnosis of diffuse cerebral anoxia in the future.

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