

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

Acta Neurol Taiwan 2016;25:21-26

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 interhemispheric 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. Cardiopulmonary resuscitation 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

From the Ankara Numune Education and Research Hospital, Neurology Clinic, Ankara-Turkey.
Received July 14, 2015. Revised November 16, 2015
Accepted January 20, 2016.

Corresponding Author: Esra Eruyar, MD. Ankara Numune Education and Research Hospital, Neurology Clinic, Ankara-Turkey.
E-mail: dr.esrayetkin@gmail.com

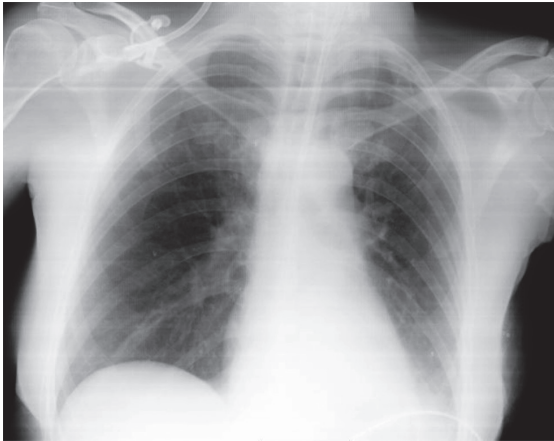


Figure 1. Normal Lung X-ray.

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

Laboratory analyses were as follows: Wbc: 10.46/ μ l (4.4-11.3), neu: 3.00/ μ L (2.1-6.1), lym: 7.36/ μ L (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 prophylaxies 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 echocardiography no cardiac pathology

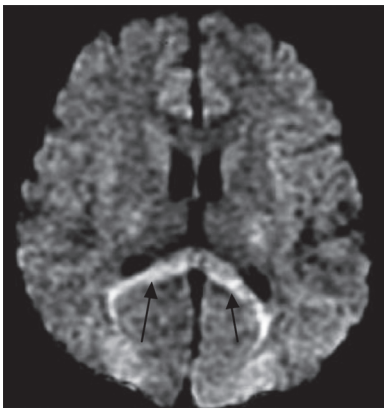


Figure 2. Diffusion restriction in splenium of corpus callosum and in occipital lobes (black arrows).

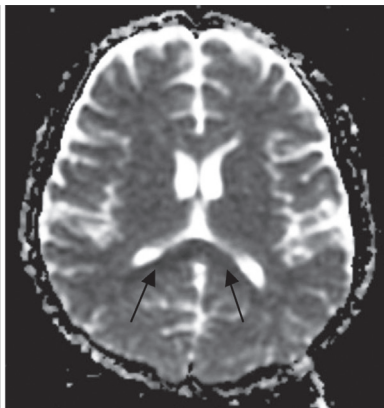


Figure 3. Hypointensity in splenium of corpus callosum bilaterally on ADC image (black arrows).

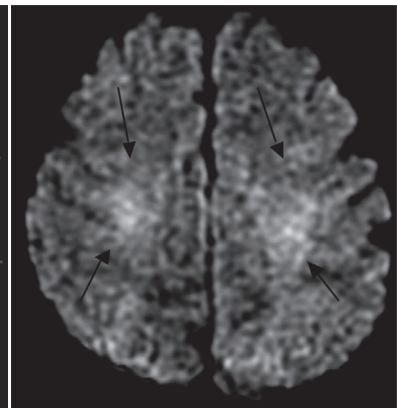


Figure 4. Hyperintensity of bilateral vertex on diffusion weighted images (black arrows).

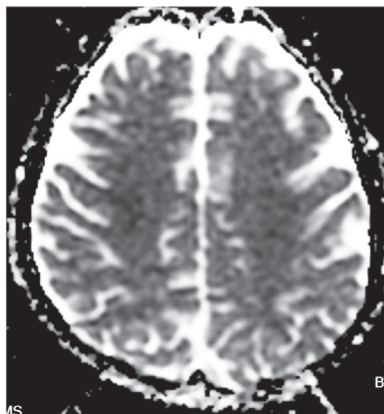


Figure 5. Hypointensity in bilateral vertex on ADC image (black arrows).

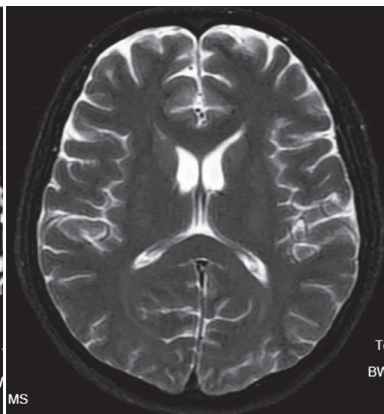
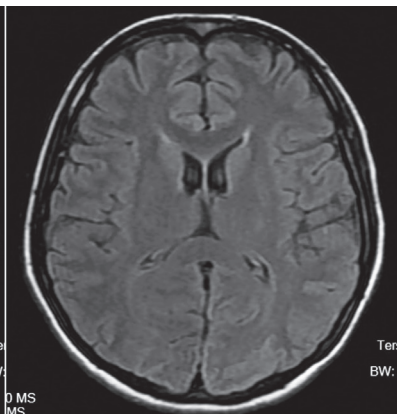


Figure 6,7. Isointens appearance of splenium on T2 image and T2 Flair image.



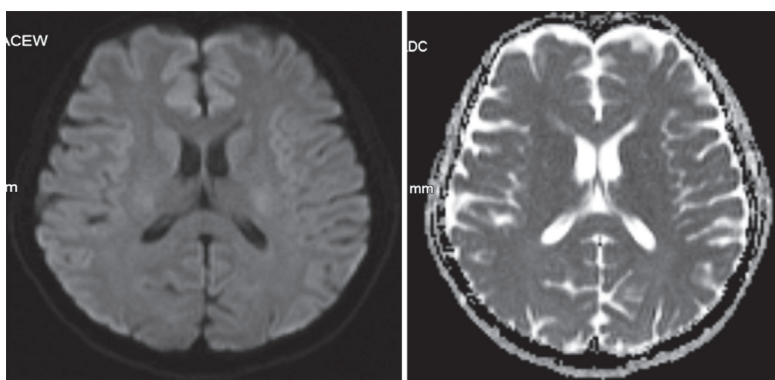


Figure 8,9. Control diffusion MRI and ADC imaging were normal after 3 months.

was observed.

The patient was intubated and connected to the mechanical ventilator. In initial neurological examination she was unconscious and unresponsive to verbal and painful 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 distinguish 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 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 reciprocal and connect the hemispheres homotopically, while others link various cortical

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 occurring 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 vascular areas; 2- the delayed appearance of the splenium lesions in early and late MRIs were available lack of involvement of neighboring vascular 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 sheaths 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 algorithm, 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.

REFERENCES

1. Bianchi MT, Sims JR. Restricted Diffusion in the Splenium of the Corpus Callosum After cardiac arrest. *The Open Neuroimaging Journal* 2008;2:1-4.
2. Tanaka K, Nogawa S, Ito D, Suzuki S, Dembo T, Kosakai A, Fukuuchi Y. Phosphorylation of cyclic adenosine monophosphate response element binding protein in oligodendrocytes in the corpus callosum after focal cerebral ischemia in the rat. *J Cereb Blood Flow Metab* 2001;21:1177-1188.
3. Moody DM, Bell MA, Challa VR. The corpus callosum, a unique white-matter tract: anatomic features that may explain sparing in Binswanger disease and resistance to flow of fluid masses. *Am J Neuroradiol* 1988;9:1051-1059.
4. Chrysikopoulos H, Andreou J, Roussakis A, Pappas J. Infarction of the corpus callosum: computed tomography and magnetic resonance imaging. *Eur J Radiol* 1997;25:2-8.
5. Kasow DL, Destian S, Braun C, Quintas JC, Kagetsu NJ, Johnson CE. Corpus callosum infarcts with atypical clinical and radiologic presentations. *Am J Neuroradiol* 2000;21:1876-1880.
6. Friese SA, Bitzer M, Freudenstein D, Voigt K, Kuker W. Classification of acquired lesions of the corpus callosum with MRI. *Neuroradiology* 2000;42:795-802.
7. Doherty MJ, Jayadev S, Watson NF, Konchada RS, Hallam DK. Clinical implications of splenium magnetic resonance imaging signal changes. *Arch Neurol* 2005;62:433-437.
8. Apak S. "Korpus Kallozum" Beynin Merkezindeki Gizemli Bölge. *Güncel Pediatri* 2009;7:142-146.
9. Josse G, Seghier ML, Kherif F, Price CJ. Explaining function with anatomy: language lateralization and corpus callosum size. *J Neurosci* 2008;28:14132-14139.
10. Gazzaniga MS. Cerebral specialization and interhemispheric communication. Does the corpus callosum enable the human condition? *Brain* 2000;123:1293-1326.
11. Takanashi J, Barkovich AJ, Shiihara T, Tada H, Kawatani M, Tsukahara H, Kikuchi M, Maeda M. Widening spectrum of a reversible splenial lesion with transiently reduced diffusion. *AJNR Am J Neuroradiol* 2006;27:836-838.
12. Uchino A, Takasa Y, Nomiyama K, Egashira R, Kudo S. Acquired lesions of the corpus callosum: MR imaging. *Eur Radiol* 2006;16:905-914.
13. da Rocha AJ, Reis F, Gama HP, da Silva CJ, Braga FT, Maia AC Jr, Cendes F. Focal transient lesion in the splenium of the corpus callosum in three non-epileptic patients. *Neuroradiology* 2006;48:731-735.
14. Knyazeva MG. Splenium of corpus callosum: patterns of interhemispheric interaction children and adults. *Neural Plast* 2013.
15. Lamantia AS, Rakic P. Cytological and quantitative characteristics of four cerebral commissures in the rhesus monkey. *J Comp Neurol* 1990;291:520-537.
16. Saenz M, Fine I. Topographic organization of V1 projections through the corpus callosum in humans. *NeuroImage* 2010;52:1224-1229.
17. Angelucci A, Bullier J. Reaching beyond the classical receptive field of V1 neurons: horizontal or feedback axons? *J Physiol Paris* 2003;97:141-154.
18. Segraves MA, Innocenti GM. Comparison of the distributions of ipsilaterally and contralaterally projecting corticocortical neurons in cat visual cortex using two fluorescent tracers. *J Neurosci* 1985;5:2107-2118.
19. Innocenti GM. General organization of callosal connections in the cerebral cortex. *Cerebral Cortex* 1986:291-353.
20. Arbelaez A, Castillo M, Mukherji SK. Diffusion-Weighted MR Imaging of Global Cerebral Anoxia. *AJNR Am J Neuroradiol* 1999;20:999-1007.
21. Burdette JH, Ricci RE, Pettiti N, Elster AD. Cerebral infarction. Time course of signal intensity changes on diffusion-weighted MR imaging. *AJR Am J Roentgenol* 1998;171:791-795.
22. Castillo M, Mukherji SK. Early abnormalities related to postinfarction Wallerian degeneration: evaluation with MR diffusionweighted imaging. *J Comput Assist Tomogr* 1999;23:1004-1007.
23. Uchino A, Sawada A, Takase Y, Egashira R, Kudo S. Transient detection of early wallerian degeneration on diffusion-weighted MRI after an acute cerebrovascular accident. *Neuroradiology* 2004;46:183-188.
24. Cho JS, Ha SW, Han YS, Park SE, Hong KM, Han JH, Cho EK, Kim DE, Kim JG. Mild encephalopathy

- with reversible lesion in the splenium of the corpus callosum and bilateral frontal white matter. *Clin Neurol* 2007;3:53-56.
25. Seo HJ, Kim SY, Kim WM, Hong YJ, Sohn JH, Lee SM, et al. A case of encephalitis with a reversible splenial lesion on a diffusion weighted MRI image. *J Korean Neurol Assoc* 2006;24:507-510.
26. Hong JM, Park MS, Jun DC. Transient splenial lesion of the corpus callosum in patients with infectious disease. *J Korean Neurol Assoc* 2005;23:667-669.
27. Wijdicks EF, Hijdra A, Young GB, Bassetti CL, Wiebe S. Practice parameter: prediction of outcome in comatose survivors after cardiopulmonary resuscitation (an evidence-based review): report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurology* 2006;67:203-210.