Case Report

The neurological monitoring and treatment of a case diagnosed with postpartum cerebellar infarction

Refah Sayin[1], Mansur Kamaci[2], Hanim Guler Sahin[3], Zehra Kurdoglu[4], Serif Aksin[5]

ABSTRACT

A 32-year-old female patient with a previous history of cesarean section, who was referred to our clinic due to pre-eclampsia, was administered a cesarean section due to fetal distress. Consultation with the Neurologic Department was performed upon the development of complaints including headache, dizziness and blurred vision. Upon detection of acute infarction in the left cerebellar region on cerebral diffusion magnetic resonance imaging (MRI), she was referred to the Neurology Clinic. To investigate the etiology of stroke occurring at an early age, various biochemicals, hematological analyses, vasculitis, infection markers and radiological imaging methods (carotis, vertebral artery color Doppler ultrasonography, cerebral arteriography-venography MR angiography, and cervical MR angiography) were used. Treatment included anti-lipid and anti-aggregant therapies for hypertriglyceridemia and infarction respectively. The patient was discharged upon improvement in her complaints.

KEY WORDS: Pregnancy, Cerebellar infarction, Antiaggregant therapy, Cerebral arteriography.

Pak J Med Sci January - March 2012 Vol. 28 No. 1 217-219

How to cite this article:

Sayin R, Kamaci M, Sahin HG, Kurdoglu Z, Aksin S. The neurological monitoring and treatment of a case diagnosed with postpartum cerebellar infarction. Pak J Med Sci 2012;28(1):217-219

INTRODUCTION

The cerebellar infarction observed during early pregnancy nearly 50 years ago were reported to occur secondarily to venous thrombosis.[1] Soh et al suggested that cerebellar infarction developing within 10 hours after the haemolysis, elevated liver enzymes, low platelet (HELLP) syndrome was not directly attributed to the HELLP syndrome.[2] Usually, the etiology is arterial occlusion in cases of cerebellar infarctions associated with stroke in young people, and the studies report that it results from 40% dissection in the intracranial vertebral artery and primarily from the involvement of the posterior inferior cerebellar artery (PICA).[3] Hypercoagulation is suggested to represent a significant risk factor in the development of pre-eclampsia during pregnancy.[4] The reports suggest that the cerebrovascular risk increases due to the increased predisposition to hypercoagulation caused by pregnancy and that cerebellar infarctions develop, even if rarely. Magnetic Resonance Imaging (MRI) can explicitly determine the localization of the cerebellar infarction.

This case report presents the neurologic monitoring and treatment of a case of postpartum cerebellar infarction accompanying pre-eclampsia together with the literature data.

1. Refah Sayin, MD,
Department of Neurology,
2. Mansur Kamaci, MD,
3. Hanim Guler Sahin, MD,
4. Zehra Kurdoglu, MD,
5. Serif Aksin, MD,
2-5: Department of Obstetrics and Gynecology.
1-5: Faculty of Medicine,
Yuzuncu Yil University,
Van, Turkey.

Correspondence:

Dr. Refah Sayin,
Assistant Professor,
Department of Neurology,
Faculty of Medicine,
Yuzuncu Yil University,
65200, Van,
Turkey.
E-mail: refahsayin@yyu.edu.tr
refahsayin@yahoo.com.tr

* Received for Publication: July 15, 2011
* Accepted: November 20, 2011

CASE REPORT

A 32-year-old, Gravida two, Para one female with a previous history of cesarean section was referred to our clinic from the center, to which she presented with the complaints of headache, dizziness, blurred vision and edema, under the preliminary diagnosis of pre-eclampsia. An obstetric ultrasonography revealed positive fetal cardiac activity, breech presentation; BPD and FL found the case was 30 weeks pregnant; and an umbilical arterial Doppler ultrasound exam revealed S/D.4.5, RI.0.78, PI.1.4, and a biophysical profile of 6. Upon detection of fetal distress on the non-stress test (NST), she underwent cesarean operation to give birth to a live female baby with a weight of 1090 grams, 1st minute apgar score of 4 and 5th minute apgar score of 6; the newborn was put under monitoring in the intensive care unit.

The patient was referred to the Neurology Clinic due to persistence of the headache, dizziness and blurred vision during the postpartum period. The cerebral diffusion MRI revealed a 10-mm field that was of heterogeneous intensity on T1-weighted series and of hyperintensity on T2-FLAIR-weighted series (Fig-1), and the patient was referred to the Neurology Clinic. The blood pressure was 140/90 mmHg and the pulse was 80/min, the body temperature was 36.5^0C, and the physical examination was considered normal (excluding hands, face and the pretibial edema). On neurologic examination, she was conscious, cooperated, and oriented with normal speech. No neck stiffness, meninks irritation findings, motor deficit or sensory deficits were detected. Deep tendon reflexes (DTR) were normoactive; the left cerebellar tests showed slight incapacity. Romberg and tandem walk tests revealed impairment. At the neurology clinic, various biochemical-hematological analysis and radiological imaging methods were used to investigate the etiology of this early-age stroke. Bilateral carotis artery and bilateral vertebral artery color Doppler ultrasonography revealed normal results. Cerebral artery MR angiography, bilateral carotis arteries, anterior cerebral artery, pericallosal artery and mid cerebral artery, Willis Polygon arterial structures, basilar artery, and arteria cerebri posterior branches were normal; no aneurysm, malformation or displacement were detected; venous MR angiography revealed a slightly weakened right transverse sinus calibration (variation) and normal sagittal sinus, straight sinus, left transverse and sigmoid sinus alignments and calibrations. The echocardiographic assessment revealed normal results.

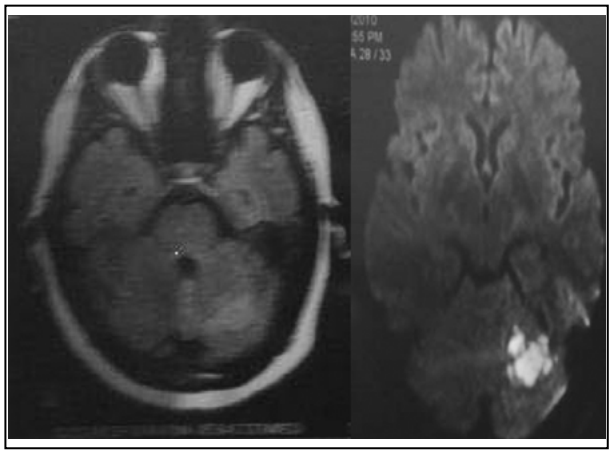

Fig-1: In the cerebral diffusion MRI and FLAIR weighted series revealed a 10-mm field in the left cerebellar hemisphere (acute infarction in the left cerebellar region)

During monitoring at the Neurology Clinic, antilipid and anti-aggregant therapies were initiated for hypertriglyceridemia and infarction respectively. The patient was discharged upon improvement in her complaints.

DISCUSSION

Cerebellar infarction was reported to occur secondarily to venous thrombosis during early pregnancy.[1] Soh et al underlined the fact that cerebellar infarction was rarely described during pregnancy. A 39-year-old Japanese primipara, at term without previous hypertension and vascular disease, developed HELLP syndrome and late deceleration repeating on the NTS, in addition to epigastric and headache symptoms, on the third day of hospitalization. Due to these findings, she had an emergency cesarean section to give birth to a female baby with a weight of 2510 grams and an apgar score of 8-10. Following the operation, she was transferred to a more advanced center upon persistence of the HELLP syndrome together with the high blood pressure and the laboratory results. She was conscious, had a headache and was experiencing dizziness; six hours after the transfer, she developed stupor and coma. An emergency computerized tomography and MRI revealed massive cerebellar infarction and secondary compression of the cerebral system. However, the patient could not undergo cerebral surgery due to the severe shock and the cerebellar infarction exhibited a rapid fatal course. Although the reason for mortality was considered to be HELLP syndrome, Soh Y et al reported that the cerebellar infarction occurred within 10 hours of the onset

of HELLP syndrome, and during this process the platelet count was 36000/mm^3 and therefore did not directly attribute the cerebellar infarction to the HELLP syndrome.[2]

Depending on the degree of the localization level of the cerebellar infarction, mortality is reported to be detected in 2-3% of cases, and this rate is reported to be higher than other localizations of cerebral infarction.[3]

In pregnancy, the cerebral infarction rate is known to be increased 13-fold relative to non-pregnancy status, and to be associated with the physiological changes in blood clotting factors, particularly during the late pregnancy and early postpartum periods. In occlusive cerebrovascular diseases, vasospasm and hypercoagulation with contribution to the development of pre-eclampsia during pregnancy are known to be significant risk factors.[4]

The literature reports that an increase in the platelet count, nystagmus, ataxia towards the right, dysmetria and motor deficit, sensory loss on the right side of the face eight days after cesarean section, cerebral MRI showing right cerebellar and median posterior bulbar infarction, color Doppler ultrasonography of the cerebral veins showing occlusion in the right vertebral artery, coagulation pattern analysis showing MTHTR gene and single prothrombin gene mutation heterozygosis could be various significant risk factors bringing on stroke during pregnancy.[5]

The case referred to our clinic under the preliminary diagnosis of pre-eclampsia underwent an emergency cesarean section on the fourth day of hospitalization upon detection of fetal distress. The cerebral diffusion MRI performed due to the persistent complaints of headache, dizziness and blurred vision revealed acute infarction in the left cerebellar region. Following the treatments performed at the neurology clinic with the details given above, the case was discharged in a state of recovery.

In conclusion, pre-eclamptic pregnant women should be monitored carefully during the postpartum period, and cerebellar infarction should be considered for the differential diagnosis, even if rare, if the neurological symptoms persist.

REFERENCES

1. Stevens H, Ammerman HH. Intracranial venous thrombosis in early pregnancy. Am J Obstet Gynecol. 1959; 78(1):104-8.
2. Soh Y, Yasuhi I, Nakayama D, Ishimaru T. A case of postpartum cerebellar infarction with hemolysis, elevated liver enzymes, low platelets (HELLP) syndrome. Gynecol Obstet Invest. 2002; 53(4):240-242.
3. Barinagarremeteria F, Amaya LE, Cantu C. Causes and mechanisms of cerebellar infarction in young patients. Stroke. 1997; 28(12):2400-2404.
4. Aminoff MJ. ¯Neurological disorders(fourth edition), Creasey RK, Resnik R (Eds), Maternal-Fetal Medicine, Philadelphia, Saunders. pp 1091-1119, 1999.
5. Altamura C, Vasapollo B, Tibuzzi F, Novelli GP, Valensise H, Rossini PM et al. Postpartum cerebellar infarction and haemolysis, elevated liver enzymes, low platelet (HELLP) syndrome. Neurol Sci. 2005; 26(1):40-42.