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Case Report

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Posterior reversible encephalopathy syndrome developing after eclampsia: a case report

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Abstract

Objective: We aimed to discuss a posterior reversible encephalopathy syndrome (PRES) case, in which the findings such as headache, confusion, seizure, impairment of the visual field and acuity, hemiparesis, and speech and consciousness disorder are observed along with hypertensive disorders of the pregnancy.

Case: In this report, we have presented a PRES diagnosis in a severe preeclamptic case at 36 weeks of gestation. She represented with retrograde amnesia, confusion, and postpartum headache.

Conclusion: With appropriate treatment, PRES is a curable condition without sequel. However, late intervention has high permanent neurological sequel risk. It should not be forgotten that early intervention to etiological reason is the most important factor for favorable prognosis.

Keywords: Posterior reversible encephalopathy syndrome, eclampsia.

Özet: Eklampsi sonrası gelişen posterior reversibl ensefalopati sendromu: Olgu sunumu

Amaç: Gebeliğin hipertansif hastalıkları ile birlikte izlenebilen, baş ağrısı, konfüzyon, nöbet, görme alanı ve keskinliğinde azalma, hemiparezi, konuşma güçlüğü ve bilinç durumunda değişiklikleri de içeren birtakım bulgularla ortaya çıkan posterior reversibl endefalopati sendromu (PRES) olgusunu sunmayı amaçladık.

Olgu: Gebeliğinin 36. haftasında eklampsi tanısıyla tedavi altına alınan ve doğum sonrası baş ağrısı, bilinç bulanıklığı ve retrograd amnezi tarifleyen ağır preeklampsi hastasında ortaya çıkan bir PRES olgusunu ele aldık.

Sonuç: Uygun tedavi sonucu pek çok PRES olgusu sekelsiz iyileşmektedir. Fakat tedavideki gecikmeler kalıcı nörolojik sekel bırakma potansiyeline sahiptir. Etiyolojik nedene yönelik hızlı girişimin prognozun olumlu seyretmesindeki en önemli etken olduğu unutulmamalıdır.

Anahtar sözcükler: Posterior reversibl ensefalopati sendromu, eklampsi.

Introduction

Posterior reversible encephalopathy syndrome is a clinical and radiological condition in which seizure, consciousness disorder, sudden headache, impairment of the visual field, vomiting, nausea and focal neurological findings are observed in varying rates. Its incidence is not known clearly. Retrospective epidemiological studies show that 7–20% of the cases are associated with preeclampsia, eclampsia and other gestational hypertensive diseases.[1] Hypertensive encephalopathy, renal failure, immunosuppressive and chemotherapeutics, autoimmune and ligament diseases may also cause this condition.[2] Although its pathophysiology is still controversial, decreased cerebral autoregulation causing increase in the cerebral blood flow or association of endothelial dysfunction with cerebral hypoperfusion are the leading hypotheses.[3] Injury of blood-brain barrier after sudden hypertensive attack is thought to trigger the syndrome.[4] Displaying temporary edema of cortical area or sub-cortical white matter in basal ganglia, frontal lobes, cerebellum or brain stem, but mainly in parieto-occipital lobes, with magnetic resonance imaging confirms the diagnosis. The diagnosis is usually established retrospectively. Among all the etiological factors, it has been expressed

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that the cases developing after eclampsia were highly retrospective and their lesions were recovering; however, late intervention might cause permanent sequel.[5] In our report, we evaluated a case found to have retrograde amnesia and blurred vision after eclampsia attack and established with PRES diagnosis by clinical and radiological findings after delivery.

Case Report

Twenty-eight-year-old patient (gravida 2, parity 1) referred to the emergency service at 36 weeks of gestation with the complaints of unconsciousness and seizure two times, and it was found in her examination that her blood pressure was 170/110, fetal biometry was consistent with 32–33 weeks and she had oligohydramnios. In her physical examination, no significant finding was observed except pretibial edema. In her laboratory findings, AST was 50 (U/L), ALT was 55 (U/L), creatinine was 0.53 (mg/dl), LDH was 487 (U/L), Hgb was 14.6 (g/dl), BUN was 25.68 (mg/dl), thrombocyte was 151,000 (K/ul) and there was 1+ proteinuria in the urine. There was no significant finding in the pattern of fetal heart beat. The patient was administered 2 g/h magnesium sulphate (MgSO4). Due to the eclampsia indication, the patient delivered 2230 g male baby by cesarean section. During the postoperative period, the patient was followed up for 2 days in the intense care unit. Laboratory values rapidly improved; despite no significant finding was detected in the neurological examination and electro-encephalography (EEG) made upon the complaints of headache, confusion, blurred vision and retrograde amnesia on the postoperative third day, an increase was observed in the signal intensity in *fluid attenuated inversion recovery* (FLAIR) images (**Figs. 1a** and **1c**) and T2 weighted images (**Figs. 1b** and **1d**) in the sub-

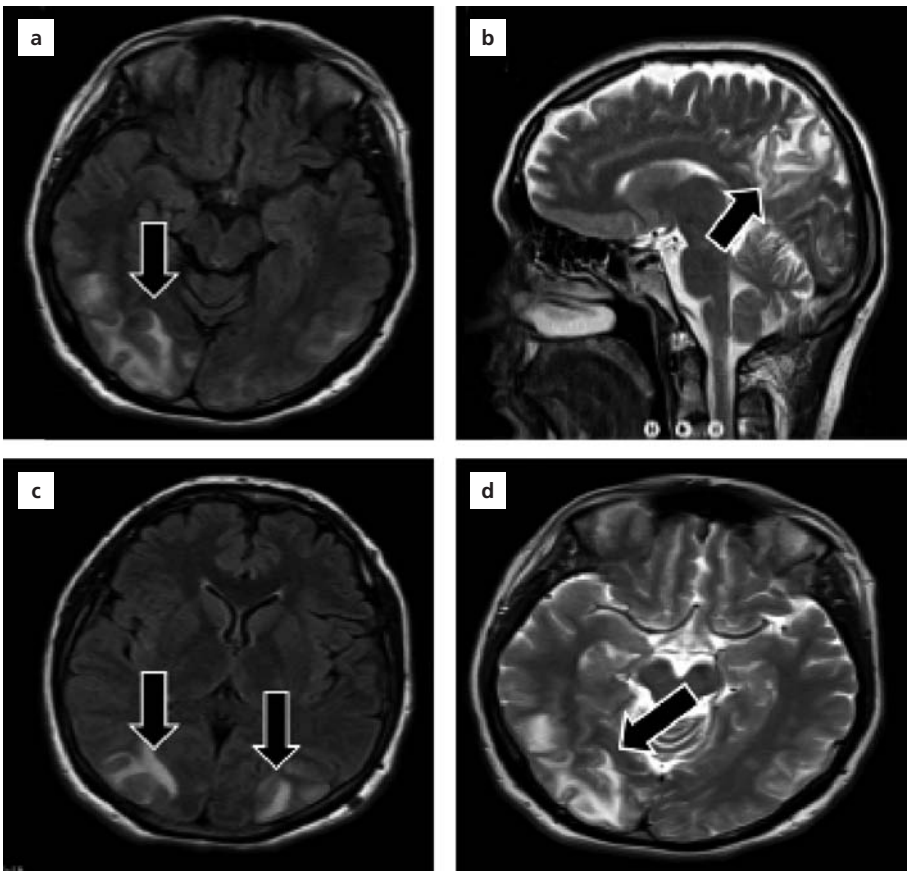

Fig. 1. An increase is observed in the signal intensity together with cortical and sub-cortical edema in the areas shown by black arrows on FLAIR (**a** and **c**) and T2 weighted (**b** and **d**) images.

cortical white matter of both occipital lobes, more particularly in right parieto-occipital region via magnetic resonance imaging (MRI). The patient was established the diagnosis of posterior reversible encephalopathy syndrome by current findings and differential diagnosis. No further treatment was required except taking blood pressure under control and following up vital signs. The patient was discharged on postoperative seventh day with the recommendation of the follow-up of neurological findings and EEG monitoring.

Discussion

The differential diagnosis of acute focal neurological findings and mental state changes occurring in preeclamptic patients is difficult. This may worry clinicians. Thrombosis, palsy and intracranial bleeding associated with hypertension are among the expected complications in eclamptic patients. Computed tomography (CT) and MRI are useful tools to identify such cases.

Posterior reversible encephalopathy syndrome was first defined by Hinchey et al. in 1996 as a case series of 15 patients.[6] It appears together with headache, confusion, seizure, decrease in impairment of the visual field and acuity, hemiparesis, and speech and consciousness disorder.[7] Focal or generalized convulsions may be seen. Unease and agitated mood can be observed in addition to somnolence, lethargy, stupor and coma. Hemianopsia, cortical blindness, blurred vision and decrease in visual acuity were reported.[6] Conditions such as acute hypertension, gestational hypertensive diseases, HIV infection causing immunosupression and cisplatin, tacrolimus, cyclosporine A and steroid drug use, and cases associated with hemolytic uremic syndrome, glomerulonephritis, blood transfusion, porphyria, tumors and hypercalcemia were reported in the literature.[8]

The regions affected in the CT are observed as diffuse hypodense area.[9] In the MRI imaging, they are seen as iso-hypo intense areas in T1 weighted images while they are seen as hyperintense areas in T2 weighted and FLAIR images. Involvement can be observed mostly on posterior cortical, sub-cortical and deep parenchymal areas. All brain structures, especially the parietal and occipital lobes, may display involvement.[9] Increased permeability in blood-brain barrier, injury of cerebral auto-regulatory mechanisms and vascular damage causing blood and fluid extravasation to parenchymal area were suggested in its pathophysiology.[10] The latter mechanism is especially asserted in cases associated with immunosuppressive drugs.[6] There are also studies arguing the opposite mechanisms. Decrease in cerebral blood flow developed due to the acute hypertension, cerebral arterial vasospasm and ischemia-induced cytotoxic edema are also other hypotheses.[11] In their case series including 47 patients, Brewer et al. reported that PRES is one of the key components of eclampsia pathogenesis. It was observed that antepartum eclampsia developed in 23 of the patients while 24 patients had postpartum eclampsia. It was also reported that headache was the most common symptom (87.2%) and there was mental state changes in 51.1% of the patients.[12] On the other hand, Ural et al. published a PRES case developing severe preeclampsia without any convulsion.[13]

With appropriate treatment, PRES is a curable condition without sequel. However, the delayed treatment may cause permanent sequel in the brain tissue.[8] Particularly, the cases with concomitant vasospasm and ischemia are under risk.[14] Monitorization of blood pressure, discontinuing immunosuppressive drugs and anticonvulsant treatment are the basic approaches. In the study of Demirel et al. including 7 pregnant women diagnosed with PRES, it was reported that six patients showed remission within approximately 2 and 5 days, and this period delayed up to 15 days in a patient receiving mechanical ventilation support for 10 days and speech disorder persisted in this patient.[15] In the treatment, mean artery pressure (MAP) is aimed to be 105–125 mmHg. For that purpose, parenteral calcium channel blockers and labetalol or oral nifedipine treatment are recommended.[8] It was reported that nimodipine, which is a cerebral vasoselective calcium channel blocker, is effective against vasospasm. In the studies performed on pregnant women who underwent treatment due to hypertensive leukoencephalopathy, both conventional and MRI angiography images showed the efficiency of nimodipine.[16] MgSO4, propofol, benzodiazepines and phenytoin were recommended for the treatment of cases developing refractory status epilepticus. There are studies showing the success of intravenous valproic acid use in such patients.[17] Especially $MgSO_4$ helps cerebral vasodilatation by inhibiting calcium-dependent vasoconstriction and shows neuroprotective activity by preventing ischemia.[18] In our case, the patient was monitored in the intense care unit without requiring mechanic ventilation, and target blood pressure values were achieved by administrating 30 mg nifedipine (with 12 hours of inter-

val) as anti-hypertensive in addition to $MgSO_4$ for 48 hours (1 g/h) during pre-postoperative period. We believe that the $MgSO_4$ treatment, anti-hypertensive treatment and rapid intervention according to etiological reasons are useful for the prognosis.

Conclusion

PRES is a clinical condition with multifactorial reasons where different symptoms appear together, imaging methods stand out and diagnosed increasingly. Suspicious neurological findings should be warning factors for all cases presenting gestational hypertensive diseases. Providing rapid diagnosis, effective treatment and intense care conditions are the most significant factors for the positive progress of the prognosis.

Conflicts of Interest: No conflicts declared.

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