Atypical Presentation of Posterior Reversible Encephalopathy Syndrome in a Patient Diagnosed with Postpartum Gestational Hypertension

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Hypertensive disorders of pregnancy are a major cause of maternal mortality and morbidity. Preeclampsia and eclampsia are among the most common causes of posterior reversible encephalopathy syndrome (PRES). Clinically, PRES is characterised by visual disturbances, headache, nausea, change in mental status and seizure. Although treatment of the underlying cause provides clinical and radiological healing, a delay in the diagnosis and treatment can result in permanent brain damage and death. The exact incidence of new-onset postpartum hypertension is difficult to ascertain. Women with mild hypertension are usually asymptomatic; therefore, patients either can not be diagnosed or are diagnosed with late complications. With this case, we would like to present a patient who had no diagnosis of maternal hypertension or preeclampsia-eclampsia during pregnancy but, after a postpartum seizure, was diagnosed with PRES.

Keywords: Postpartum, gestational hypertension, posterior reversible encephalopathy syndrome

Introduction

Posterior reversible encephalopathy syndrome (PRES) is a clinical and radiological condition characterized by various combinations of headache, seizure, visual abnormalities, cognition disorders, nausea/vomiting or focal neurological deficits (1). Seizure type is normally generalized, but focal onset can be observed. Visual impairments can vary from complaints of blurred vision to cortical blindness (2). Signs can develop within hours and can last for weeks depending on the severity and delay in the starting of proper treatment. PRES is almost always observed with preeclampsia, eclampsia, systemic infection, sepsis and shock, some autoimmune diseases, various cancers, chemotherapy, transplantation and accompanying immunosuppression (particularly with calcineurin inhibitors) and a facilitating clinical condition such as sudden hypertension (1, 3).

In the literature, PRES is generally reported to be correlated with the obstetric patient group that developed preeclampsia or eclampsia. However, it is difficult to ascertain the precise incidence of hypertension that develops in the postpartum period. In this case, we present a patient who had convulsions in the post-caesarean period and who was diagnosed with mild gestational hypertension and PRES with common atypical presentation. This case was written-up as an article upon informing the patient herself and obtaining her written consent.

Case Presentation

A 22-year-old, primigravida patient who had spontaneous twin pregnancy at week 36 and who had no features or complaints in her history or pregnancy controls underwent caesarean intervention with spinal anaesthesia. As a routine monitorization, electrocardiogram, pulse oximetry and non-invasive blood pressure monitorization were performed when the patient arrived at the operation room. Spinal anaesthesia was administered to the patient in the sitting position using the 25-gauge spinal needle from L₄₋₅ level, with 2 mL hyperbaric bupivacaine (0.5%). Haemodynamic and respiratory parameters were recorded at 3-min intervals throughout the entire process. Surgical operation was started when the sensory block level examined with the needle tip reached the T₅₋₆ level. The patient's blood pressure was normal in the preoperative period; it remained normal in the perioperative period. The patient was sent to the service room after an uneventful caesarean. However, she was admitted to the intensive care unit in the third postpartum hour because of a generalized tonic-clonic convulsion. Her

convulsions were stopped with 10 mg diazepam IV while she was still in the service, and it was learned that she had severe headache complaints prior to the convulsions. In the initial laboratory examinations of the patient, abnormal values were not detected apart from lactate dehydrogenase (LDH), 803 U L⁻¹; alanine transaminase (ALT), 72 IU L⁻¹ and aspartate transaminase (AST), 86 IU L⁻¹. Proteinuria was not detected in the urine tests. In her intensive care follow-ups, her blood pressure values peaked at 145/95 mm Hg. To prevent convulsions from developing, 2 gr hr⁻¹ IV infusion was started following a 4 gr MgSO₄ loading dose. Methyldopa 3X (250 mg) was administered to the patient as an antihypertensive treatment. The patient's neurological examination was evaluated as normal. Cranial magnetic resonance imaging was performed. In addition to the classical posterior areas, hyperintensity was observed in the bilateral basal ganglia, frontal lobes, cerebellum, cerebellar vermis and left temporal area in T2A and fluid-attenuated inversion recovery (FLAIR) images (Figure 1). Hyperintensity that supports vasogenic oedema was observed in the same areas in diffusion-weighted apparent diffusion coefficient (ADC) images. Pathological findings were not detected in electroencephalography (EEG) tests. The patient, whose general condition was good and whose vital findings were stable, was discharged to the service on the third day following her admittance to the intensive care unit. After 2 weeks, cranial magnetic resonance imaging (MRI) findings were observed as normal (Figure 1).

Discussion

Postpartum hypertension can be correlated with persistent gestational hypertension, preeclampsia or pre-existing chronic hypertension or it can be postpartum-onset secondary to other causes (4). However, ascertaining the precise incidence of postpartum hypertension is difficult.

Asymptomatic women with mild hypertension are generally not reported in the clinical practice. Furthermore, in emergency services, hypertension is commonly observed in postpartum women along with signs such as headache or blurred vision, but they are not reported as hypertensive unless they are admitted to the hospital. Despite the limitations, the frequency of new-onset postpartum hypertension or preeclampsia is reported to be in the range of 0.3%–27.5% (5).

Normal pregnancy is characterized by an increase in plasma volume relating to sodium and water involvement in the interstitial tissue. This is more pronounced in women with multiple pregnancies. Additionally, many women are administered vast amounts of intravenous fluids during regional anaesthesia when birth or caesarean surgery is performed and also in the postpartum period. In some women, particularly relating to the suboptimal kidney function, acute or delayed mobilization of vast amounts of intravenous fluids to the intravascular gap may cause a volume overload that leads to hypertension (6). Use of non-steroid anti-inflammatory medicines such as ibuprofen and indomethacin in pain

treatment and of ergot alkaloids (ergometrine or methylergonovine) in uterine atony can cause hypertension. These drugs can cause similar symptoms in patients with severe gestational hypertension (GH)-preeclampsia, and they are also associated with nausea, vomiting and headache (5). Potentially life-threatening complications are cerebral infarction or haemorrhage, congestive heart failure, pulmonary edema, liver failure or death. Hypertensive women with headache, visual impairments or neurological deficits should be evaluated for possible cerebrovascular complications. Diagnostic cranial imaging must be performed for these women, and neurology and/or neurosurgery departments must be consulted. Maternal complications can be prevented by a constant follow-up, record keeping and evaluation of symptoms in the postpartum period. In our case, it was found out that there was a complaint of severe headache that started in the postoperative period and that a seizure ensued 3 h later. Vasogenic oedema compatible with PRES was observed in the cranial MRI performed for aetiology.

The most common finding of patients in neuroradiological images is the cerebral oedema that specifically includes parieto-occipital regions in the posterior regions of the cerebral hemispheres. Lesions are generally symmetrically located, but they can be asymmetric as well (1, 7). The frontal lobe, temporal lobe and cerebellum are the other brain regions that are commonly affected (7). Combined lesions can develop depending on the size of the increasing oedema. Diffusion-weighted MRI imaging is helpful in determining and consistently displaying abnormal areas that represent vasogenic oedema (8). Patients generally recover with early diagnosis and treatment, and oedema completely disappears in control imaging.

Generally, there is no correlation between the clinical features of the patients and the prevalence of lesions (7, 9). Although a general difference was not found in lesion distribution depending on PRES etiology, there are studies that report that the number of brain regions that is affected with basal ganglia involvement in preeclampsia and eclampsia patients is more numerous and that more vasogenic oedemas are observed in normotensive patients (7, 9, 10). In our study, despite the presence of common atypical lesions, there was no presence of a clinical feature, except for headache and a single history of seizure, and her clinic was not severe.

Its pathophysiology is not properly understood and there are various problems with the hypertension/hyperperfusion theory, although it is widely accepted. It does not occur in 25% of hypertension patients, and when it does occur, it generally does not reach the autoregulation limit (mean arterial pressure 150–160 mm Hg) (1). In the follow-ups of our patients, mild blood pressure elevation was observed and these values were not at the autoregulation limit.

Recent data reported that in some women, preeclampsia and even eclampsia can develop in the absence of hyperten-

Figure 1. In the FLAIR examination performed to the patient, hyperintense lesions (B1) compatible with vasogenic oedema were observed in the bilateral parieto-occipital region and cerebellum, cerebellar vermis (A1), bilateral basal ganglia and frontal lobes, left temporal lobe. In the T2A examination, hyperintense lesions (C1) compatible with vasogenic oedema were observed in the bilateral basal ganglia and left temporoparietal junction. In the control T2A and FLAIR examination performed 2 weeks later, the lesions were observed to regress and disappear (A2, B2, C2)

sion or proteinuria as well. Majority of these women display other signs of preeclampsia such as the existence of various symptoms or laboratory abnormalities. When GH occurs in the absence of proteinuria and in the presence of intractable symptoms or abnormal laboratory tests, preeclampsia should be considered. Approximately 25%–50% of mild gestational hypertension cases generally progress to preeclampsia. Awareness and a correct diagnosis and treatment approach are important in these atypical preeclampsia-eclampsia cases (11). Severe headache complaints and elevation in liver enzymes were observed in our case. An elevation in blood pressure measurements was not detected in the pre-seizure period, and proteinuria was not observed. When we evaluate our patient with these data, we maintain that she could be a case of atypical preeclampsia-eclampsia.

Conclusion

Preeclampsia and eclampsia are well-known causal factors for PRES. One must be aware of the neurological signs and find-

ings given that clinical and imaging findings completely recover as well as permanent brain damage can be prevented with early treatment. This case is an example that PRES syndrome can be observed in pregnant women that are not diagnosed with typical preeclampsia or eclampsia, despite having postpartum mild gestational hypertension and a good clinical prognosis. PRES syndrome should be considered for early and differential diagnosis.

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