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CASE REPORT / *OLGU SUNUMU*

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Posterior Reversible Encephalopathy Syndrome After Cesarean Section: A Case Report

Sezaryen Sonrası Gelişen Posterior Reversible Ensefalopati Sendromu: Olgu Sunumu

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ABSTRACT Posterior reversible encephalopathy syndrome (PRES) is an acute and severe neurological condition characterized by typical clinical and neuroradiological symptoms. Typical clinic symptoms include headache, confusion, nausea and vomiting, mental and visual alterations. Although PRES is not a complication of spinal anesthesia, it can be commonly confused with dural puncture headache that develops after spinal anesthesia. Our patient was diagnosed with PRES following a dural puncture and spinal anesthesia for cesarean section. PRES is a condition that needs multidisciplinary approach and that can heal without any sequelae.
Keywords: PRES, confusion, spinal anesthesia, dural puncture

ÖZ Posterior reversible ensefalopati sendromu (PRES); klinik ve nöroradyolojik bulgularla karakterize akut ve ciddi bir nörolojik durumdur. Karakteristik klinik bulguları baş ağrısı, konfüzyon, kusma, mental ve görsel değişiklikleri içerir. PRES spinal anestezinin bir komplikasyonu olmamasına rağmen spinal anestezi sonrasında gelişen dural ponksiyon baş ağrısı ile karışabilir ve dural ponsiyon PRES'i tetiklemiş olabilir. Bizim olgumuz sezaryen için spinal anestezi yapılan ve dural ponksiyon hikayesi olan, PRES tanılı bir hastadır. PRES multidisipliner yaklaşım gerektiren sekelsiz bir iyileşme ile sonuçlanan bir durumdur.
Anahtar Kelimeler: PRES, konfüzyon, spinal anestezi, dural ponksiyon

Introduction

Posterior reversible encephalopathy syndrome (PRES) is an acute and severe neurological condition and characterized neuroradiological findings (1). The incidence is not known clearly. PRES was first described in 1996 by Hinchey et al. (2) PRES may also be referred to hypertensive encephalopathy, cerebral vasospasm syndrome, and posterior reversible leukoencephalopathy syndrome (3). Characteristic clinical findings are a headache, confusion, nausea, vomiting, mental and visual changes (4). In serious cases, a seizure may be added to the clinical findings but it is not always seen. PRES can be related to acute hypertension, pre-eclampsia-eclampsia, sepsis, and immunosuppressant exposure (5). Although dural puncture is not included in PRES risk factors, symptoms following dural puncture may be similar to PRES symptoms. In this case report; we evaluated the relationship between PRES and dural puncture, the patient was diagnosed with dural puncture due to spinal anesthesia. A written consent of the patient is provided for the publication of this report.

Case Report

A 23-year-old woman at 39 weeks of gestation was evaluated for the emergency cesarean section. In the preoperative evaluation, body mass index was 31 kg/m^2. Spinal anesthesia was planned according to American Society of Anesthesiologists physical status 1 E risk. Preoperative blood pressure was 120/75 mmHg, heart rate was 110/min and oxygen saturation was 98%. Two attempts with a spinal needle of 25G from L3-4 spinal gap failed. In the third attempt, after the cerebrospinal fluid (CSF) flow was seen through the spinal needle; 12 mg of 0.5% hyperbaric bupivacaine was injected into the CSF. Both sensory and motor block did not occur in the following 15th min. General anesthesia was planned. Anesthesia induction was performed with 2 mg/kg of propofol and 0.9 mg/kg rocuronium. For the maintenance of anesthesia 2% sevoflurane and oxygen/air mixture was used. The patient delivered a healthy boy (Apgar score 8) at five minutes after the induction. The mother's vital signs were normal postoperatively and the patient was transferred to ward. The patient was discharged on the first postoperative day.

On the postcesarean second day, the patient was referred to the anesthetist by an obstetrician with headache complaint and patients visual analog scale (VAS) was 6/10.

The patient described a severe throbbing headache in the frontal and occipital region after sitting. This situation was thought to be post-dural puncture headache. We recommended analgesic, bed rest, and fluid intake. In the following two days, the patient described a regression in the frontal region headache; however, headache on the occipital region (VAS: 6/10) continued.

The patient referred to the emergency department with loss of consciousness and generalized seizure on the 5th day post operatively. The patient who had three generalized tonic-clonic seizures was evaluated by a neurologist in the emergency department. In the neurological evaluation, the patient was unconscious, non cooperative and non orientation. Pupils were considered isochoric and light reflex was weak positive. Glaskow Coma Score (eyes: 2 motor: 4 verbal: 2): was 8, vital signs were stable. Ischemic or hemorrhagic cerebrovascular disease, intracranial hypotension and PRES was considered as preliminary diagnosis. The cranial computed tomography and cranial diffusion magnetic resonance imaging (MRI) of the patient were normal. Cranial MRI showed hyperintense areas in the right posterior parieto-occipital area on fluid attenuated inversion recovery (FLAIR) sections (Figure 1). The patient was admitted to the neurology intensive care unit with the preliminary diagnosis of sinus vein thrombosis. His vital signs were stable during intensive care unit follow up. Biochemical examinations revealed no abnormal findings except lactate dehydrogenase (LDH): 780 U/L (240-480 U/L). The 24-hour urine protein was 14 mg/24 h (0-110 mg/24 h). Cranial MR venography normal, which was taken for differential diagnosis, was found and sinus vein thrombosis was excluded. The patient was diagnosed with PRES according to clinical findings and neuroradiological imaging results. After 24 hours of follow up in intensive care unit, the vital signs were stable and the patient transferred to neurology service without any seizure. Treatment of the patient was organized by a neurologist, an obstetrician, and an anesthesiologist. A headache and other neurological findings were relieved on the 3rd day of treatment. Anti-oedema and anti-convulsive treatments were continued. On the 7th day of admission, the complaints were completely lost. On the 10th day of the hospitalization, the patient was discharged to home with a prescription of 1000 mg/day levetiracetam treatment.

Discussion

PRES is a transient condition with distinctive clinical and neuro-radiological features that develop acutely. PRES etiology includes acute hypertension, pregnancy-induced pre-eclampsia, eclampsia, auto-immune diseases, and immunosuppressive drugs (6). Other reasons are summarized in Table 1. Li et al. (7) investigated PRES's commitment to primary hypertension or secondary causes. Nearly 50% of the cases the cause was hypertension while 39% had immunosuppression, 7% had pre-eclampsia-eclampsia and 4% had marijuana intake 4%. Ng and Manikappa (8) presented a 33 years old case with an epileptic seizure after epidural anesthesia. In the case, they reported dural

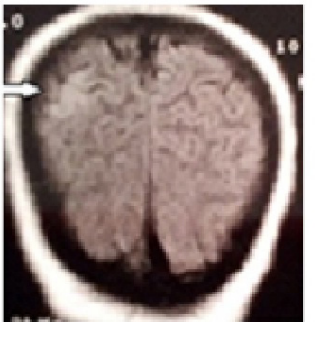

Figure 1. Hyperintense areas in the right posterior parietooksipital area fluid attenuated ınversion recovery sections (white arrows)

puncture developed during epidural anesthesia. The patient was diagnosed with PRES secondary to late normotensive postpartum eclampsia. Pre-eclampsia and eclampsia are triggered by the pregnancy and can be seen from the 20th week of pregnancy until the 48th hour postpartum (9). In our case, the possibility of late postpartum eclampsia was considered in the intensive care unit admission, but the absence of hypertension, organ damage and proteinuria excluded that diagnosis.

Two hypotheses are proposed in the pathophysiology of PRES. The first hypothesis is the vasospasm/hypoperfusion principle; which is explained with vasospasm of cerebral vessels and vasogenic edema secondary to hypoperfusion causes cerebral ischemia due to insufficient perfusion. The second hypothesis, on the other hand, is hypertension/hyperperfusion hypothesis. The theory suggests that enlargement of small arterial vessels after vascular autoregulation disorder triggered by severe hypertension damages the vascular endothelial structure and increased perfusion results with vasogenic edema in brain tissue (10).

Characteristic symptoms of PRES include a headache, vomiting, visual and mental changes, and seizures. A headache related to dural puncture is one of the complications following spinal anesthesia. The holes in the dura mater cause CSF leakage and CSF pressure fall. It is accepted that deprivation of fluid cushion in the brain causes pain-sensitive structures especially the meningeal structures to stretch and cause pain (11).

Table 1. Posterior reversible encephalopathy syndrome related conditions

Hypertension
Chemotherapy (cisplatin, cytarabine)
Treatment of immunosuppressive (cyclosporine ve tacrolimus)
After organ transplantation
Autoimmune disease - Systemic lupus erythematosus - Scleroderma - Wegener's granulomatosis - Polyarteritis nodosa - Hashimoto's disease
Infection (HIV)
Shock (sepsis, injury)
Renal failure
Pregnancy (preeclampsia, eclampsia and late postpartum eclampsia)
Others (hypercalcaemia, Henoch-Schönlein purpura, thrombocytopenia)

There is no specific laboratory parameter to identify PRES. In severe cases, thrombocytopenia, elevated LDH, abnormal red blood cell and endothelium permeability secondary to fluid-electrolyte disturbance and hypo-proteinemia may occur. Red blood cell morphology and LDH can be used to determine endothelial damage. CSF examination is usually normal (12). In our case, there was no abnormality except the elevated LDH level on the first day of intensive care. The diagnosis of PRES is made with MR findings. Reversible cortical/subcortical vasogenic edema, bilaterally symmetrical hyperintense lesions in neurocranial MR T2 weighted images in particular occipital areas are frequent (13). In our case in the cranial MR T2 FLAIR sequences, hyperintense areas were detected on the posterior parieto-occipital area. This view was evaluated as vasogenic edema induced by ischemia.

It is not possible to correlate PRES directly with a dural puncture. Dural puncture is a complication that may occur after spinal anesthesia. As a headache does not occur after every dural puncture, PRES is not a regularly expected result. However, the literature has been examined after this case and similar cases have been found and it is thought that there may be an indirect relationship (14). According to the Monro-Kellie hypothesis, the shrinking of the cerebral ventricles after CSF reduction may mechanically cause stimulation of the cerebral vessels, followed by cerebral vasogenic depression due to impaired autoregulation in the blood-brain barrier (15). Our case had an unsuccessful spinal anesthesia attempt. The reason is likely to be the patient's obesity or lack of dexterity in the application of spinal anesthesia. We think that CSF leakage is a result of dural puncture after these failed attempts. When we look at the etiology of PRES in this patient, there are no factors other than pregnancy. It was thought that the late postpartum eclampsia may cause PRES, but the exact diagnosis could not be made. Although not certain; loss of CSF after dural puncture, can be effective in indirect development PRES. More data is needed to prove the cause of the ventricle vasospasm.

Concisely, PRES is a condition that needs to be diagnosed and treated rapidly. Delay in diagnosis and treatment can result in death. It is important that patients with dural puncture headache have full neurological examination and attention should be paid to the regulation of blood pressure in follow-ups. PRES should also be considered

in the foreground. Early diagnosis and treatment with the multidisciplinary approach are important as it was in this case we presented. Cerebral edema, which was also present in our case, was successfully treated and the patient was discharged with no complications.

Ethics

Informed Consent: Consent form was filled out by all participants.

Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: E.Y., Concept: M.E.T., Design: Ö.Ö., Data Collection or Processing: A.K., Analysis or Interpretation: K.A., Literature Search: E.Y., Writing: E.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

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References

1. Ehtisham S, Hashmi HA. Posterior reversible encephalopathy syndrome. J Coll Physicians Surg Pak 2012;22:398-400.
2. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. N Engl J Med 1996;334:494-500.
3. Bell AJ. Reversible posterior leukoencephalopathy syndrome: a case presentation. Neurodiagn J 2012;52:20-33.
4. Raina S, Mahesh D, Rajendra G, Chauhan NS. Reversible posterior leukoencephalopathy syndrome. J Neurosci Rural Pract 2012;3:222-4.
5. Shah R, Kubisz-Pudelko A, Reid J. Posterior reversible encephalopathy syndrome following an inadvertent dural puncture during an emergency laparotomy for ischemic colitis - a case report. Local Reg Anesth 2014;7:1-4.
6. Liman TG, Bohner G, Heuschmann PU, Scheel M, Endres M, Siebert E. Clinical and radiological differences in posterior reversible encephalopathy syndrome between patients with preeclampsia-eclampsia and other predisposing diseases. Eur J Neurol 2012;19:935-43.
7. Li R, Mitchell P, Dowling R, Yan B. Is hypertension predictive of clinical recurrence in posterior reversible encephalopathy syndrome? J Clin Neurosci 2013;20:248-52.
8. Ng MD, Manikappa S. Postpartum seizure and ischaemic stroke following dural puncture and epidural blood patch. Anaesth Intensive Care 2012;40:347-51.
9. Erkoç SK, Kayacan Ü, Can A, Çöplüoğlu HE, Tosun A. Atypical Presentation of Posterior Reversible Encephalopathy Syndrome in a Patient Diagnosed with Postpartum Gestational Hypertension. Turk J Anaesthesiol Reanim 2015;43:119-22.
10. Feil K, Forbrig R, Thaler FS, Conrad J, Heck S, Dorn F, et al. Reversible cerebral vasoconstriction syndrome and posterior reversible encephalopathy syndrome associated with intracranial hypotension. Neurocrit Care 2017;26:103-8.
11. Doherty H, Hameed S, Ahmed I, Russell IF. Post-dural puncture headache and posterior reversible encephalopathy syndrome: a misdiagnosis or co-presentation? Int J Obstet Anesth 2014;23:279-82.
12. Postma IR, Slager S, Kremer HP, de Groot JC, Zeeman GG. Long-term consequences of the posterior reversible encephalopathy syndrome in eclampsia and preeclampsia: a review of the obstetric and nonobstetric literature. Obstet Gynecol Surv 2014;69:287-300.
13. Siddiqui TS, Irfan-ul-Haq, Rehman B, Kumar M, Iqbal N. Posterior reversible encephalopathy syndrome (PRES). J Coll Physicians Surg Pak 2012;22:168-70.
14. Minai FN, Hasan SF, Sheerani M. Post-dural puncture posterior reversible encephalopathy syndrome. J Coll Physicians Surg Pak 2011;21:37-9.
15. Şahin N, Jale M, Çelik E, Solak A, Genç B, Kalaycıoğlu S, et al. Atypical Presentation of Posterior Reversible Encephalopathy Syndrome after Post-dural Puncture Headache. Turk J Anaesthesiol Reanim 2013;41:142-5.