

Pontine Hematoma Associated with Eclampsia: Case Report

Eklampsi ile İlişkili Pons Hematomu

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ABSTRACT Intracerebral hemorrhage associated with preeclampsia and eclampsia commonly occurs in the postpartum period. In these cases, factors that predispose to intracranial hemorrhage include hypertension and coagulation abnormalities. In this report, we presented that a 34 year old-woman with eclampsia developed acute coma and quadriplegia at 34(+) weeks of gestation. On computed cranial tomography, bilateral posterior basal-partly tegmental pontine hematoma was detected. Over a period of six months, she improved markedly and was independent on activities of daily life. The underlying mechanisms of the hematoma in our case may include hyperperfusion and blood-brain barrier break down due to eclampsia. Our case suggest the importance of the close blood pressure monitoring in the puerperal patient with hypertension.

Key Words: Eclampsia; hematoma; brain stem; hypertension

ÖZET Preeklampsi ve eklampsi ile ilişkili olan intrakraniyal kanamalar, postpartum dönemde sık olarak görülür. Bu olgularda, hipertansiyon ve pıhtılaşma bozuklukları gibi faktörler intrakraniyal kanamalara yatkınlık oluşturur. Bu yazıda, akut koma ve kuadripleji tablosundaki 34(+) haftalık gebe olan otuz dört yaşındaki eklampstik bir kadın hasta sunuldu. Kraniyal tomografide iki yanlı posterior bazal-kısmen tegmental uzanım gösteren pons hematomu saptandı. Ortalama altı ayda hasta günlük yaşam aktivitelerinde bağımsız hale gelerek belirgin bir iyileşme gösterdi. Hematomun altında yatan mekanizmalar arasında hiperperfüzyon ve kan-beyin bariyerinin eklampsiye bağlı olarak bozulması sayılabilir. Hipertansiyonu olan gebelerde kan basıncının yakından takibi önemlidir.

Anahtar Kelimeler: Eklampsi; hematom; beyin sapı; hipertansiyon

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Pregnancy is a known risk factor for stroke, and the postpartum period is the most vulnerable time.¹ In this period, the large decrease in blood volume at childbirth, rapid changes in hormone status that alter hemodynamics and coagulation may predispose to a stroke.¹ Intracerebral hematoma associated with pregnancy commonly occurs in the postpartum period in the setting of preeclampsia and eclampsia.² Eclampsia is main cause of both nonhemorrhagic stroke and intraparenchymal hemorrhage.² In preeclamptic and eclamptic cases, factors that predispose to hemorrhage include hypertension or coagulation abnormalities.² Putaminal, posterior lobar and cortical hematomas account for the majority of hemorrhages associated with preeclampsia and eclampsia whereas primary pontine

hematoma accounts for about 10% of intracranial hemorrhages.^{2,3} We present a case with eclampsia in which a pontine hemorrhage was detected at 34 (+) weeks of gestation.

CASE REPORT

A 34-year old-woman presented with nausea, vomiting, severe headache and blurred vision at 34(+) weeks of gestation was admitted to the Emergency Department of Neurology. She had recent history of hypertensive episodes since 16(+) weeks of gestation. She was morbidly obese. Her blood pressure was measured 300/120 mmHg. During questioning, the patient developed a generalised tonic-clonic seizure. She was intubated because of respiratory distress. Cesarean section was performed since fetal distress was evident. The newborn child was healthy, but the mother did not awake up after the operation. She required mechanical ventilation. Neurological examination revealed coma, left central facial weakness, quadriplegia, diminished deep tendon reflexes and unresponsive bilateral plantar reflexes. The oculocephalic reflex and cornea reflex were absent. Pupils were barely reactive to

light. She had a normal platelet count on admission. Erythrocyte sedimentation rate (ESR) was 60 mm/h and C-reactive protein (CRP) level was 16 mg/L. The diagnosis was eclampsia based on the findings of very high initial blood pressure, 4(+) proteinuria without edema and seizure. She did not fulfill the criteria for hemolysis, elevated liver enzymes, low platelet count (HELLP) syndrome. Emergent computed tomography (CT) revealed 20x10 mm bilateral posterior basal-partly tegmental pontine hematoma with breaking into the fourth ventricle (Figure 1 A,B). The lesion was more prominent on the left side. Brain magnetic resonance imaging (MR) examination revealed large (2x1 cm in diameter), hyperintense lesion on T1, T2 and FLAIR weighted images in the bilateral basal-tegmental pontine area, which was not contrast-enhancing (Figure 2). The brain stem is surrounded with edema. Intracranial MR angiography was normal. She was treated with 16 mg of dexamethasone daily for ten days and anti-hypertensive drugs. Her consciousness improved on the 20th hospital day. Respiratory distress improved and she was extubed. The oculocephalic reflex and cornea

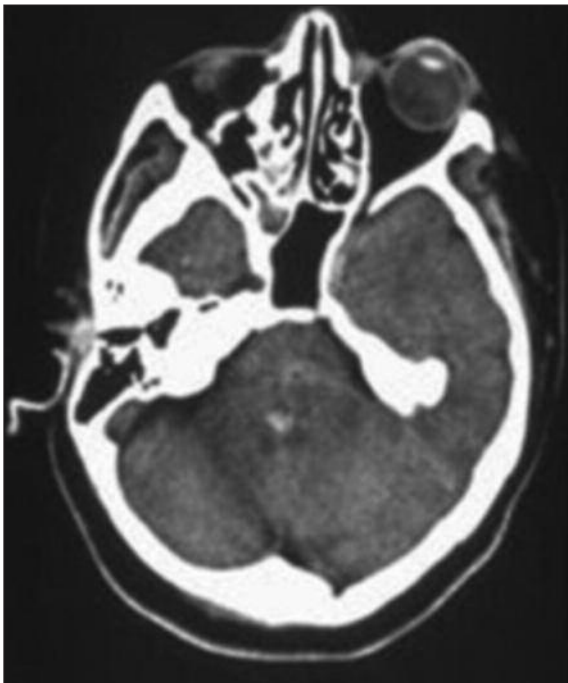


FIGURE 1A,B: Diagnostic images of patient. Cranial CT images show hyperdense lesion in the bilateral basal-tegmental pontine area with breaking into a fourth ventricle.

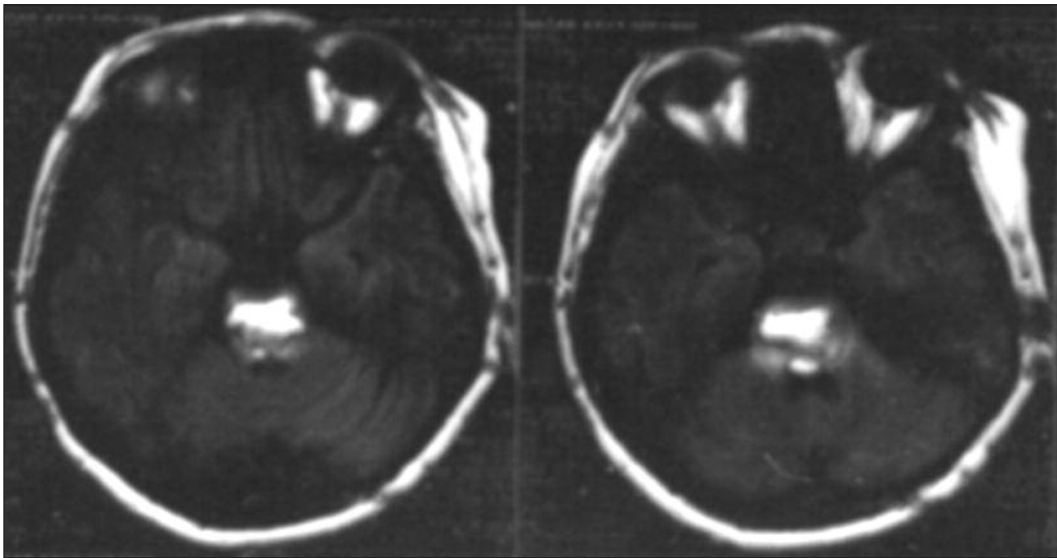


FIGURE 2: Diagnostic images of patient. Cranial FLAIR weighted MRI images show large (2x1 cm in diameter), hyperintense lesion in the bilateral basal-ganglia pontine area.

reflex returned. There was mild skew deviation with left hypotropia and bilateral horizontal gaze paresis but vertical eye movements were normal. Speech was dysarthric. A program of physical therapy was instituted and mild quadriparesis (muscle strength -4/5) persisted. At one month after the admission, her CT showed absorption of the hematoma and she was discharged on the second hospital month. Over a period of six months, neurological examination was nearly normal except bilateral oculomotor disturbances, mild dysarthria, bilateral appendicular and truncal ataxia. Her blood pressure was stable on drugs. She was independent on activities of daily living. The patient gave an informed consent.

DISCUSSION

Eclampsia is defined by occurrence of proteinuria, hypertension, convulsions and/or coma in the intrapartum or immediate postpartum period.⁴ Eclampsia occurs in 0.1% to 0.2% of all pregnancies.⁴ The pathophysiology of eclampsia remains incompletely understood.

We report a case with eclampsia and pontine hemorrhage. Hypertensive hemorrhages, coagulation abnormalities, vascular malformations were taken into consideration in differential diagnosis. There were not any predisposing risk factors to in-

tracerebral hemorrhage except hypertension due to pregnancy. CT delineated pontine hemorrhage. Diagnosis is based on findings in CT and magnetic resonance imaging (MRI) in combination with an angiography presenting a normal vascular situation.

Cerebral autoregulation maintains a constant blood flow to the brain, despite systemic blood pressure alterations, by means of arteriolar constriction and dilatation in the nonpregnant individual normally.⁵ During severe preeclampsia and eclampsia, cerebral autoregulation of cerebral blood flow may be altered.⁶ The upper limit of autoregulation may be reduced in such patients, breached by rapid and systolic pressure rises with consequent forced overdistention of the cerebral vasculature.⁶ When cerebral autoregulation is disrupted, the blood-brain barrier can be affected and increased permeability and extravasation of proteins and fluid may lead to edema in some vascular beds, whereas rupture and hemorrhage might occur in others.⁶ Similar events occur in hypertensive encephalopathy, cyclosporin A neurotoxicity, renal diseases (i.e. uremic encephalopathy), after treatment of high-grade carotid stenosis or large intracranial arteriovenous malformation or in high altitude mountain sickness and had been named as hyperperfusion syndrome.⁷⁻⁹ Neuroimaging features of hyperperfusion syndromes include diffuse or

focal hyperintensity on T2-weighted images without restricted diffusion on diffusion weighted MR, predominately in the supratentorial white matter, especially in occipital lobes.^{8,10} Brain stem involvement has also been reported.¹⁰ These changes are reflecting vasogenic edema and resolve completely in most cases although rarely small infarcts and hemorrhages may occur.⁸ Thus the etiologic mechanism of the hemorrhage in our case may be associated with hyperperfusion due to eclampsia.

Pontine hematomas were classified according to CT findings: type 1, small unilateral tegmental; type 2, basal-tegmental; type 3, bilateral tegmental; and type 4, massive type.³ In our case a bilateral posterior basal-partly tegmental located pontine hematoma was presented. Pontine hematomas frequently lead to death or severe disability and they have a poor prognosis.¹¹ In previously studies, the mortality rate was reported as 40-60%.^{3,11}

Dziewas et al. reported that mortality and morbidity of pontine hematoma might be estimated best by CT parameters and the clinical features.¹² According to them coma on admission, large paramedian type of hematoma and the transverse diameter ≥ 20 mm predicted a poor prognosis.¹² Jung et al. detected that long term outcome of survivors is influenced by the initial level of consciousness, diameter and volume of the hematoma.¹³

In another study, coma and bad clinical status on admission was found the only significant prognostic factor for mortality.¹⁴ In different study, a higher blood pressure was found to be significantly related to high mortality.¹⁵ Jang et al. investigated predictors of 30-day death and 90-day functional recovery after pontine hematoma.¹⁶ They obtained that a systolic blood pressure < 100 mmHg at admission was associated with a high risk of mortality within 30 days.¹⁶ Otherwise, Dziewas et al. found that an excessive high systolic blood pressure at admission was strongly associated with mortality in pontine hematoma.¹²

In our case, hypertension was aggressively and early treated with anti-hypertensive drugs. Despite the initial presentation and a large hematoma, our patient had marked clinical and radiological improvement. Since the hyperperfusion could have a role in pathogenesis of the hemorrhage, blood pressure should be carefully monitored in these cases.

Pontine hematoma associated with eclampsia has not been reported except a case with pontine hematoma was associated with HELLP syndrome.⁴ HELLP syndrome is a severe complication in late pregnancy.¹⁷ It is more common than eclampsia.¹⁸ It syndrome has been considered a variant of preeclampsia, but it can occur as an separate or in association with preeclampsia.¹⁸ Preeclampsia and HELLP syndrome are related and significant overlap in their both clinical presentation and placent features.¹⁸ Approximately 4 to 12 percent of patients with preeclampsia or eclampsia develop superimposed HELLP syndrome.¹⁸ Cerebral manifestations of the HELLP syndrome only rarely occur.¹⁷ Brain stem involvement is exceptionally associated with HELLP syndrome with a variable prognosis.¹⁹ In general, location of the intracranial hemorrhage was extrapontine areas (lobar or basal ganglia).^{20,21} Our case did not fulfill the criteria for HELLP.

The several studies were reported about the relationship between obesity and incidence of spontan intracranial hematoma. In a recently published study demonstrate that body mass index level might have an impress on the location of intracranial hematoma, that is, obesity was associated with pontine hematoma.²² Compatible with this study, pontine hematoma of our patient may be associated with obesity.

In conclusion, we have presented a case of eclampsia and pontine hematoma, with a unexpectedly good outcome. Our main intention is to point out the importance of the close blood pressure monitoring in the puerperal patient with hypertension.

REFERENCES

- Jeng JS, Tang SC, Yip PK. Incidence and etiologies of stroke during pregnancy and puerperium as evidenced in Taiwanese women. *Cerebrovasc Dis* 2004;18(4):290-5.
- Geocadin RG, Razumovsky AY, Wityk RJ, Bhardwaj A, Ulatowski JA. Intracerebral hemorrhage and postpartum cerebral vasculopathy. *J Neurol Sci* 2002;205(1):29-34.
- Chung CS, Park CH. Primary pontine hemorrhage: a new CT classification. *Neurology* 1992;42(4):830-4.
- Zeidman LA, Videnovic A, Bernstein LP, Pelletier CA. Lethal pontine hemorrhage in postpartum syndrome of hemolysis, elevated liver enzyme levels, and low platelet count. *Arch Neurol* 2005;62(7):1150-3.
- Paulson OB, Strandgaard S, Edvinsson L. Cerebral autoregulation. *Cerebrovasc Brain Metab Rev* 1990;2(2):161-92.
- Martin JN Jr, Thigpen BD, Moore RC, Rose CH, Cushman J, May W. Stroke and severe preeclampsia and eclampsia: a paradigm shift focusing on systolic blood pressure. *Obstet Gynecol* 2005;105(2):246-54.
- Kitaguchi H, Tomimoto H, Miki Y, Yamamoto A, Terada K, Satoi H, et al. A brainstem variant of reversible posterior leukoencephalopathy syndrome. *Neuroradiology* 2005;47(9):652-6.
- Schwartz RB. Hyperperfusion encephalopathies: hypertensive encephalopathy and related conditions. *Neurologist* 2002;8(1):22-34.
- Tarım E, Giray S. [Posterior reversible encephalopathy syndrome (PRES) in the differential diagnosis of eclampsia: case report]. *Turkiye Klinikleri J Gynecol Obst* 2011;21(3):213-5.
- de Seze J, Mastain B, Stojkovic T, Ferriby D, Pruvo JP, Destée A, et al. Unusual MR findings of the brain stem in arterial hypertension. *AJNR Am J Neuroradiol* 2000;21(2):391-4.
- Murata Y, Yamaguchi S, Kajikawa H, Yamamura K, Sumioka S, Nakamura S. Relationship between the clinical manifestations, computed tomographic findings and the outcome in 80 patients with primary pontine hemorrhage. *J Neurol Sci* 1999;167(2):107-11.
- Dziewas R, Kremer M, Lüdemann P, Nabavi DG, Dräger B, Ringelstein B. The prognostic impact of clinical and CT parameters in patients with pontine hemorrhage. *Cerebrovasc Dis* 2003;16(3):224-9.
- Jung DS, Jeon BC, Park YS, Oh HS, Chun TS, Kim NK. The predictors of survival and functional outcome in patients with pontine hemorrhage. *J Korean Neurosurg Soc* 2007;41(2):82-7.
- Balci K, Asil T, Kerimoglu M, Celik Y, Utku U. Clinical and neuroradiological predictors of mortality in patients with primary pontine hemorrhage. *Clin Neurol Neurosurg* 2005;108(1):36-9.
- Seo W, Oh H. Acute physiologic predictors of mortality and functional and cognitive recovery in hemorrhagic stroke: 1-, 3-, and 6-month assessments. *J Stroke Cerebrovasc Dis* 2007;16(2):57-63.
- Jang JH, Song YG, Kim YZ. Predictors of 30-day mortality and 90-day functional recovery after primary pontine hemorrhage. *J Korean Med Sci* 2011;26(1):100-7.
- Knopp U, Kehler U, Rickmann H, Arnold H, Gliemroth J. Cerebral haemodynamic pathologies in HELLP syndrome. *Clin Neurol Neurosurg* 2003;105(4):256-61.
- Wolf JL. Liver disease in pregnancy. *Med Clin North Am* 1996;80(5):1167-87.
- Gutiérrez-García JM, Carreres A. [Brain stem involvement in eclampsia and HELLP syndrome]. *Rev Neurol* 1999;28(12):1162-6.
- Gliemroth J, Knopp U, Kehler U, Felberbaum R, Nowak G. HELLP syndrome with haemoglobin vasospasm. *J Clin Neurosci* 2000; 7(1):59-62.
- Hashiguchi K, Inamura T, Irita K, Abe M, Noda E, Yanai S, et al. Late occurrence of diffuse cerebral swelling after intracerebral hemorrhage in a patient with the HELLP syndrome-Case report. *Neurol Med Chir (Tokyo)* 2001;41(3):144-8.
- Matsukawa H, Shinoda M, Fujii M, Takahashi O, Yamamoto D, Murakata A, et al. Impact of body mass index on the location of spontaneous intracerebral hemorrhage. *World Neurosurg* 2011 Nov 1. doi:10.1016/j.wneu.2011.10.038