

Seizure During Epidural Blood Patch in Spontaneous Intracranial Hypotension Spontan İntrakraniyal Hipotansiyonda Epidural Kan Yaması Uygulanması Sırasında Nöbet

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ABSTRACT

Intracranial hypotension is a syndrome characterized by orthostatic headache and low cerebrospinal fluid pressure. Epidural blood patch is likely to be the most effective therapy method in cases that conservative approaches are unsuccessful. Epidural blood patch seems to be safe since the most commonly encountered complication is only lumbar pain which is temporary. In this report, we aimed to present a 53-year-old woman who was diagnosed as having spontaneous intracranial hypotension and suffered from a seizure during the epidural blood patch application. Seizures following epidural blood patch, which are more common in postpartum period, have been previously reported. Intracranial hypotension was spontaneous in our patient. There was no history of pregnancy or spinal intervention and, she did not have any seizure disorder throughout her life. We may propose that epidural blood patch was the most possible causal factor in our patient and seizures after epidural blood patch might have been related to rapid and abrupt increase in intracranial pressure. However, we may suggest that the procedure is safe providing gradual increase of intracranial pressure. (*Archives of Neuropsychiatry* 2012;49: 160-162)

Key words: Spontaneous intracranial hypotension, epidural blood patch, seizure, intracranial pressure

Conflict of interest:

The authors reported no conflict of interest related to this article.

ÖZET

İntrakraniyal hipotansiyon, ortostatik baş ağrısı ve beyin omurilik sıvısı basıncının düşük olduğu bir sendromdur. Konservatif tedaviler yarar sağlamadığında epidural kan yamasının en etkili tedavi seçeneği olduğu düşünülmektedir. En sık karşılaşılabilecek komplikasyon geçici lomber ağrı olduğu için epidural kan yamasının güvenli olduğu düşünülmektedir. Burada, spontan intrakraniyal hipotansiyon tanısı alan ve epidural kan yaması sırasında nöbet gelişen 53 yaşında kadın hastanın klinik bulguları sunulacaktır. Epidural kan yaması sonrasında nöbet daha önce de bildirilmiştir, fakat bu bildirilerin çoğu postpartum dönemdeki olgulardır. Hastamızda ise intrakraniyal hipotansiyon spontan olarak gelişmişti. Gebelik ve spinal girişim öyküsü yoktu ve tüm yaşamı boyunca nöbet geçirmemişti. Hastamızda epidural kan yamasının en olası neden olduğunu ve nöbetin intrakraniyal basınçtaki hızlı ve ani yükseliş ile ilişkili olduğunu düşünmekteyiz. Bununla beraber, intrakraniyal basıncın kademeli artırılması şartıyla yöntemin güvenli olduğunu söyleyebiliriz. (*Nöropsikiyatri Arşivi* 2012;49: 160-162)

Anahtar kelimeler: Spontan intrakraniyal hipotansiyon, epidural kan yaması, nöbet, intrakraniyal basınç

Çıkar çatışması:

Yazarlar bu makale ile ilgili olarak herhangi bir çıkar çatışması bildirmemişlerdir.

Introduction

Intracranial hypotension is a syndrome characterized by orthostatic headache and low cerebrospinal fluid (CSF) pressure. It can be secondary to spinal/cranial surgery, lumbar puncture or dural damage due to spinal anesthesia; or can occur spontaneously without any trauma history (1). Spinal CSF leakage is the most common reason for spontaneous intracranial hypotension (SIH).

The initial treatment of SIH includes bed rest, oral hydration and oral caffeine applications. Though steroid, intravenous caffeine or theophylline applications seem to be candidates for specific therapies, their efficiencies are limited. Autologous blood injection to the spinal epidural cavity [epidural blood patch (EBP)] is one of the most effective therapy methods in cases that conservative approaches are unsuccessful (2). EBP seems to be safe, since the most commonly encountered complication is only lumbar pain which is temporary. In this report, we aimed to

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discuss a 53-year-old woman who was diagnosed as having SIH and suffered from seizure during the EBP application.

Case Presentation

A 53-year-old woman admitted to our outpatient clinic with severe orthostatic headache which occurred with standing and disappeared when lying down. She also had tinnitus and neck pain. Neurological examination was normal but mild neck stiffness. She had no history of seizures or any other medical diseases, drug use, any operation, as well as spinal surgery or anesthesia. Routine blood chemistry was normal. Gadolinium-enhanced cranial magnetic resonance imaging (MRI) revealed thin subdural effusion, bilateral dural thickening and slight brain sagging (Figure 1). Opening pressure on lumbar puncture (LP) was 10 mm H₂O. Despite the opening pressure was low, we succeeded to get limited amount of sample. However, we were able to evaluate protein, glucose and electrolyte levels as well as cellular content which were all normal. Intrathecal MR cisternography showed diffuse dural leakage into the epidural space at the level of dorso-lumbar region. Low LP opening pressure and findings observed on both cranial MRI and MR cisternography examination were concordant with the SIH syndrome. The patient was recommended to rest in bed and to increase fluid and caffeine intake. After 10 days of follow-up, since the symptoms did not improve, EBP application was decided. On the epidurography performed before the application, it was seen that contrast substance which was given at the D12-L1 level rapidly rose to the foramen magnum level (Figure 2). Under fluoroscopy, 20 ml autologous peripheral venous blood was injected in the epidural space at the level of D12-L1 with Tuohy 18 G needle. During the procedure, there appeared sudden loss of consciousness and tonic followed by clonic contractions of all four extremities and facial muscles with head and eye deviation. The whole event lasted about 60 seconds and at this time evident tachycardia was observed. At that moment, blood injection was stopped, the blood injected was taken back as much as possible and 5 mg diazepam was administered via intravenous route. The consciousness of the patient returned to normal in one minute after the seizure and she gave meaningful answers. Therefore, blood was given slowly with the cooperation of the patient 20 to 30 minutes after the first injection. Another 20 cc of blood was injected in 10 minutes without any problem. Neurological examination performed after the procedure revealed no abnormality. The electroencephalography performed in the following days of the procedure showed normal background rhythm without any epileptiform abnormalities. The following week all the symptoms of SIH disappeared and after 2 months MRI was normal.

Discussion

Our patient had dull headache that worsened within a short time after standing accompanied by tinnitus and neck pain which improved by EBP. She did not have any history of spinal trauma, surgery or puncture. Low CSF pressure was shown indirectly by MRI and directly by LP. Therefore, she met all the criteria for SIH in the 2004 version International Headache Society criteria for

headache (3). When initial conservative treatment is insufficient or ineffective, EBP is the preferred choice of treatment. As EBP provides sudden improvement in SIH symptoms, it has the opportunity to be used for diagnostic purposes. The improvement in symptoms can be described by a few mechanisms: (2,3,4) 1-EBP provides clot formation in the area close to the leakage and therefore can prevent more CSF leakage by working as a dural tamponade, 2-Injected blood prevents the passage of CSF to the epidural cavity by increasing epidural pressure and results in reduced absorption of CSF. So the CSF balance in the brain is restored. As a result; after blood patch, the headache and neurological complaints secondary to stretching of pain sensitive structures and cranial nerves dramatically improve.

The complications of EBP can be listed as lumbar pain, paresthesia, neck pain, loss of strength in the legs, temporary bradycardia, dizziness, and pneumocephalus. Theoretically, epidural infections, chemical or infectious meningitis and/or arachnoiditis can develop (5,6). There is a limited body of reports in the literature covering seizures after EBP in the treatment of SIH and the common point of those reports is the occurrence in postpartum cases (5,7,8).



Figure 1. Gadolinium enhanced cranial magnetic resonance imaging (MRI) revealed thin subdural effusion, bilateral dural thickening and slight brain sagging

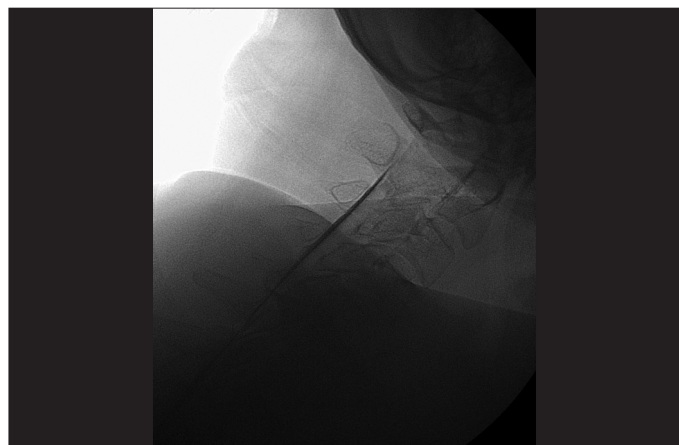


Figure 2. Epidurography which was performed before the epidural blood patch application, it was seen that contrast substance given at the D-12-L1 level rapidly raised to cervical region and foramen magnum level

Kardash and colleagues (8) mentioned about seizures in a postpartum patient after EBP, however, this patient had subdural hematoma which was overlooked before EBP. After the diagnosis of subdural hematoma, the seizures and other neurological symptoms were ascribed to the subdural hematoma, not to EBP in this case. On the other hand, Marfurt and colleagues (5) reported the case of a patient with postdural puncture headache after labor who had recurring seizures after EBP. Seizures emerged hours after the procedure. Although these authors mentioned about caffeine toxicity, direct toxic effects of blood, changes of vascular reactivity, and infections in the etiology of seizures after EBP, they did not point out any factor specifically. In another reported case, postpartum seizure after postdural puncture headache and EBP was linked to application of intravenous caffeine sodium benzoate or coincident eclampsia (7). Although EBP was a possible cause of seizures in those reported postpartum cases, differential diagnosis of preeclampsia/eclampsia or structural brain diseases should be done perfectly to say the reason of seizure is EBP (5). Since patients with preeclampsia/eclampsia have an increased risk of seizures, EBP should be performed with precaution. However, our patient was not in the postpartum period and did not have any seizure throughout her life. EBP was the most possible direct causal factor. During EBP, an epileptic attack developed as a result of blood leakage through the arachnoid space and irritation of brain would require time. But in our case, seizure developed while blood was being injected into the epidural space and it did not recur during the second and slower procedure or thereafter. Therefore, direct blood toxicity mechanism is insufficient to explain our case. We may speculate that the cause of seizure was the rapid increase in intracranial pressure (ICP). In SIH, ICP is low and epidural blood injection causes rapid CSF movement to intracranial space. This event leads to rapid and dramatic increase in ICP in SIH patients which may trigger seizures. Increased ICP in association with clinical seizures has been reported in several small case series (9,10). A single patient reported by Gabor and colleagues (9) was

therapeutically paralyzed and, therefore, had clinically unrecognizable prolonged seizures that resulted in high ICPs. In another case report, a patient with post-traumatic seizures had paroxysmal rise in ICP with subclinical seizures (10). These reports suggest the relationship between the rise in ICP and electrographic seizures. Abrupt improvement of seizure in our case after ceasing the procedure and taking the blood back supports this theory. Furthermore, the observation of slower application of the same amount of blood (within 10 minutes) leading to slower and mild increases in pressure did not lead to seizure further supports above mentioned theory.

In conclusion, without an underlying seizure disorder, in SIH, seizures may be seen during EBP application possibly because of a rapid increase in ICP. Seizures may be prevented by slow application and, therefore, gradual increases in ICP.

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