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Case Report

Epidural Blood Patch in Subdural Hematoma Due to Spontaneous Intracranial Hypotention: A Case Report and Literature Review

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ABSTRACT

Spontaneous intracranial hypotension (SIH) is an orthostatic headache syndrome with variable symptoms and complications which is often misdiagnosed at initial manifestations. SIH results from spontaneous CSF leakage leading to brain sag. The typical findings on cranial MR imaging consist of subdural fluid collections, enhancement of the pachymeninges, engorgement of venous structures, pituitary hyperemia, and sagging of the brain. Subdural hematoma may occur as a result of tearing of bridging veins and usually develop into chronic subdural hematoma. The majority of patients with SDH due to SIH have chronic DSH and, therefore, rarely present with neurological deficits. Evacuation of SDH may be performed for large SDH with ME (mass effects), or when dilated or asymmetric pupil is present. However in most cases, evacuation of the hematoma is not necessary and may result in worsened outcomes. The epidural blood patch (EBP) is a treatment of choice. Fortunately, most of these subdural hematomas can be handled with treatment directed at the underlying spinal CSF leak without the need for surgery.

We report the case of 42-year-old man with the chief complaint of orthostatic headache. He was admitted to neurology ward and after imaging studies, it was found that he has bilateral subdural hematoma. Due to the lack of history of trauma, underlying disease, and coagulation disorder, and considering the imaging findings, the patient was referred to the pain department to perform an epidural blood patch. After performing the epidural blood patch, the patient's pain was relieved immediately, and during a three-month follow-up period, the epidural hematoma was completely absorbed.

Spontaneous intracranial hypotension (SIH) is a highly misdiagnosed and underdiagnosed disorder and requires a high index of suspicion for diagnosis. During the last decades, a much larger number of spontaneous cases are identified. Literature is a bit confusing, with some authors recommending evacuation of subdural fluid in cases of deteriorating consciousness and few others recommending EBP first even in patients with comatose state but epidural patch is often an important part of treatment.

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Introduction

Spontaneous intracranial hypotension (SIH) is an orthostatic headache syndrome with an estimated annual incidence of 5/100.00. It is pathogenetically separated from that of postdural puncture headache and from postoperative cerebrospinal fluid (CSF) loss. Females gender are more often affected than males (2:1), the peak incidence is around 40 years of age, and SIH is rare but not absent in children [1].

The key symptom is orthostatic headache which generally occurs or worsens within seconds to minutes of taking an upright position (usually within 15 min of assuming the upright position) and which tends to increase in severity over the course of the day but can be delayed by hours. The headache usually improves or relieves after lying down, typically within 30 minutes and is usually holocephalic and diffuse, but may be localized to one region of the head (although most pronounced in the back of the head) or may be asymmetric. It can be explained as the result of the following causal sequence: low CSF volume—sagging of the brain—tension on the cranial nerves and dura mater (The dura mater is especially tension-sensitive in the posterior fossa) [1-2]. Symptoms can lose their positional dependence, or even worsen when the patient reclines [1].

These symptoms usually occur after a strong cough, exercise, trauma or fall, but an association with systemic diseases of the connective tissue such as Marfan and Ehlers Danles has been mentioned [3]. In addition to orthostatic headache, low cerebrospinal fluid (CSF) pressure or volume and radiological evidence of cerebrospinal fluid leakage (diffuse pachymeningeal enhancement on brain MRI) are prominent features of the disease. Although it is benign in most cases, it can be dangerous if left untreated or inadequately treated [4-5]. Spontaneous leakage of CSF leads to brain sag, and occasionally, rupture of bridging veins may cause subdural hematomas (SDHs) [5].

Scahltenbrand initially introduced ‘‘aliquorrhea’’ in 1938 to describe a patient with SIH, and also suggested that there were 3 possible causes of SIH syndrome: increased CSF absorption, reduced CSF production, and CSF leakage. Spontaneous CSF leakage causes CSF hypovolemia which leads to brain sag, downward traction on leptomeninges and neural structures, compensatory venous engorgement and enlargement of the subdural/subarachnoid space [5].

A small tear in the nerve root sleeves or spinal meningeal diverticula has been identified as a source of leakage, which may be anywhere within the spinal canal, but most commonly in the cervicothoracic region. The most important intracranial complications of SIH are downward displacement of the cerebellar tonsils and subdural hematoma [6]. tearing of bridging veins or bleeding from engorged veins in the subdural space causes subdural hematomas (SDHs). Subacute to chronic SDHs have been reported in 16–57% of SIH patients [5]. SDH is mainly seen in men, while SIH without SDH is predominant in women [7].

Nonpositional and persistent headache also occurs in patients with associated subdural effusion in

the chronic stage of the illness. Subdural hematoma which is limited in volume will usually resolve spontaneously after normal CSF volume has been restored However, a significant volume of hematoma may require surgical treatment [6].

Subdural hematomas with varying degrees of mass effect also are not uncommon in spontaneous intracranial hypotension and are about half as frequent as subdural hygromas. Fortunately, most of these subdural hematomas can be managed with treatment directed at the underlying spinal CSF leak without the need for craniotomy. Only rarely do the subdural hematomas require evacuation, and if the underlying CSF leak is left untreated, the risk of a recurrent subdural hematoma is high [8].

Caudal displacement of the cerebral tonsils may be misdiagnosed as tonsillar herniation with intracranial hypertension in the acute stage of the illness, or idiopathic Chiari type I malformation in the chronic stage of the illness. An important difference is the panpachymeningeal enhancement on MR imaging [6].

Other relatively common symptoms of SIH include posterior neck pain or stiffness, nausea and vomiting, and photophobia and phonophobia. These may be secondary to meningeal irritation. Some patients have changes in hearing (echoing, under water sensation), tinnitus, and a disturbed sense of balance, secondary to traction of cranial nerves. Rare additional symptoms include visual blurring, visual field defects, diplopia, facial numbness or facial pain, facial weakness or spasm,parkinsonism, ataxia, and dementia. The most common cranial nerve that is paralyzed is the 6th pair. Distortion of the pituitary stalk may lead to hyperprolactinemia, galactorrhea and severe brain displacement may lead to diencephalic herniation with stupor or coma.Spinal manifestations of SIH are uncommon, with interscapular pain the most frequent. Rarely, local back pain at the site of the leak, quadriparesis and radicular symptoms can occur [2-3,5].

Characteristic MRI findings of SIH can be classified into two categories. The first category includes compensatory changes in reduced intracranial volume explained by the Monro-Kellie hypothesis. Pachymeningeal enhancement, subdural fluid collections, prominence of cerebral venous sinuses, and pituitary hyperemia are included in this category. The other category is the structural alteration caused by loss of CSF buoyancy. Downward displacement of brain structures is representative for this phenomenon. Recently, alteration of intracranial angles including

mamillopontine distance, pontomesencephalic angle, and vG/SS angle has been reported as a new marker of structural alteration in patients with SIH [4].

Several case series have demonstrated that the majority, but not all patients with SIH have brain MRI abnormalities. These can be remembered by the acronym SEEPS1:

S= subdural fluid collections (mostly hygromas, occasionally hematomas)

E= enhancement of the pachymeninges (uniform, smooth and diffuse)

E= engorgement of venous structures

P= pituitary hyperemia

S= sagging of the brain (descent of the cerebellar tonsils, effacement of the basal cisterns, bowing of the optic chiasm, flattening of the pons) [2,6].

Sagging was defined as a downward displacement of the brain because of CSF leakage. The better responses to autologous EBP treatments in patients with sagging of the brain were considered dependent on the fact that it is helpful to raises the CSF pressure by preventing CSF leakage sites [9]. In any case, diffuse dural enhancement in imaging is the most prominent and valuable finding for diagnosis. Dural enhancement is presumably the result of either tearing or vasodilation of the meningeal vasculature, which lacks a tight junction [6].

CT myelography has historically been the test of choice for localization of CSF fistula in patients with spontaneous intracranial hypotension [10]. More recent techniques have been described, including dynamic CTM, digital subtraction myelography, heavily T2-weighted spinal MR imaging, and intrathecal gadolinium MR myelography (MRM) [10]. MRM with intrathecal gadolinium has been shown to have a high rate of leak detection and appears safe in small doses used for myelography [10]. Diagnostic criteria include a CSF pressure < 60 mm H2O and/or evidence of a CSF leak on imaging [1].

Several primary and secondary headaches should be considered in the differential diagnosis of this disease, including subarachnoid hemorrhage, subdural hematoma, vertebral and carotid artery dissection, cerebral venous sinus thrombosis, benign intracranial hypotension, post traumatic headache, meningitis, postural orthostatic tachycardia syndrome [3].

Treatment for spinal CSF leaks can be divided into conservative therapy, epidural patching, and surgery [11]. SIH treatment is usually conservative (bed rest and overhydration), but autologous epidural blood patch (EBP) has emerged as the most important non-surgical management However, cases that required hematoma removal after EBP have been reported [5]. Conservative treatment, which includes bed rest, caffeine, and oral hydration and abdominal banding, is often recommended as first-line treatment for patients with SIH. Caffeine is the most commonly used agent and can be administered in the form of caffeinated drinks or caffeinated tablets. Improvement of symptoms has also been reported with other drugs, including corticosteroids, indomethacin, and theophylline, although these agents are usually not curative and may carry a risk of side effects with long-term use [9,11].

The epidural blood patch (EBP) is the mainstay of treatment. Targeted EBP treatment has been reported to be superior to blind EBP. However, the reported success rate of EBP is low, ranging from 36% to 70%(4). Whether to perform EBP or SDH surgery as the initial procedure remains controversial [5].

The volume of blood that can be injected is mainly limited by local back pain or the development of radiculopathy. Sometimes it is recommended to place the blood patch in 2 separate places, first in the thoracolumbar area and then in the lower lumbar area [8]. Trendelenburg position, either supine, prone, and/or lateral for 30 to 60 minutes after EBP, depending on the location of the CSF leak is recommended. Intrathecal infusion of saline or artificial CSF should not be expected to seal a CSF leak but may be required as an effective temporizing measure to restore CSF volume until the leak can be permanently repaired [8].

Relief of symptoms often is instantaneous thereby also serving a diagnostic purpose, and this is likely due to replacement of lost CSF volume with blood volume within the spinal canal. Initially, about 10 to 20 mL of blood is used, and this is effective in relieving symptoms in about one third of patients, presumably by forming a dural tamponade, thereby sealing the leak. The second mechanism is limiting the flow of CSF in the spinal epidural space and as a result, disrupting the absorption of cerebrospinal fluid, which has a delayed effect [8-9].

Epidural patching can be done in two ways: either as a “blind” (also referred to as a “non-targeted”) patch, where autologous blood is injected into the lower thoracic or lumbar epidural space, or as a targeted blood patch, where the patching agent is directed toward the area suspected to be leaking based on spinal imaging studies [11]. Estimates for success of non-targeted EBP vary widely, with success rates of 30–70% reported for the initial epidural patch [9,11].

There is no consensus how to perform a blood patch (“loss of resistance,” fluoroscopy-guided, CT guided, blood, or fibrin glue) [5]. If the epidural patch is unsuccessful, it can be repeated and a large volume (20 to 100 ml) epidural blood patch should be considered. Due to the potentially high volume of blood injected, a minimum interval of 5 days between consecutive injections is recommended. recurrence of a spinal CSF leak is seen in approximately 10% of patients [8]. A recurrence of headache following successful treatment of spontaneous intracranial hypotension may indicate a recurrent CSF leak, but if the pattern of headache has

changed, rebound transient intracranial hypertension or dural venous sinus thrombosis should be considered [8].

Two recent studies addressed the management of subdural hematomas, an occasional complication of SIH. Both studies found that most subdural hemorrhages recurred if the hematoma was drained prior to performing an EBP. These studies reinforce the concept that these hematomas form passively as the result of decreased intracranial pressure, and that they are therefore likely to recur if the underlying CSF leak is not treated first [11]. However, others note that subdural fluid collections can be managed safely by directing treatment at the underlying CSF leak, skipping the hematoma evacuation, and some authors emphasize that craniotomy might increase the risk of brain herniation [5]. SDH which is limited in volume usually resolves spontaneously after normal CSF volume has been restored. However, a significant volume of hematoma may require surgical treatment [5].

Around 1/5 of patients treated with either epidural blood patches or surgery develop symptoms of rebound hypertension (rebound intracranial hypertension/ RIH). Patients are usually managed with acetazolamide for some weeks. Risk is higher when treatment is delayed for more than 10 weeks and when patients are obese [1]. Although often self-limited and minor, RIH symptoms can at times be severe or persist well beyond the immediate posttreatment period [12]. There are several diagnostic guidelines for suspecting RIH. First, patients who have RIH typically describe a change in headache phenotype. In contrast to the headaches of intracranial hypotension, which are commonly occipital in location, headaches in RIH are most commonly frontal or peri-orbital in location. Additionally, headaches in intracranial hypotension are exacerbated with upright positioning, whereas those associated with RIH are typically worse when recumbent [12]. The headaches associated with RIH are worse in the morning. These symptoms mirror features of those patients with raised intracranial pressure caused by other etiologies. Jugular venous outflow is impaired in the recumbent position, which probably accounts for the worsening of symptoms in some patients with RIH while lying down. Prolonged recumbent positioning at night may contribute to morning headaches, though other proposed mechanisms such as a rise in pCO2 at night, decreased CSF reabsorption, and increased CSF production modulated by chemical factors such as melatonin, for example, may also play a role [12].

Severe or continuous nausea, vomiting, and blurred vision are not common with low CSF pressure., patients with acute RIH frequently have nausea, vomiting, and blurred vision, which are symptoms commonly found with elevated intracranial pressure. Finally, the timing of the symptoms with the time of the patch is helpful. These findings begin within the first 24 to 48 hours after treatment, and for this reason, patients should be carefully monitored during this period of time [12].

patients with abnormal brain MRI findings and a focal spinal CSF leak have an excellent prognosis while those with normal initial MRI findings and a diffuse multilevel spinal CSF leak have a poor prognosis. Some patients have persistent symptoms following treatment, in spite of documented resolution of CSF leakage. Such patients may have residual altered CSF dynamics or small residual CSF leaks below the level of detection of current imaging technique [8].

Improvement of MRI abnormalities can be seen within hours to weeks of successful treatment of the CSF leak. Clinical improvement usually occurs first, before changes on MRI are seen. Larger subdural hematomas can take up to a few months to improve [2]. Surgery should be considered when the following criteria are present: high severity of symptoms, identification of leak site, failure to respond to other treatment methods [1-2]. Leaking meningeal diverticula can be ligated with suture or a metal aneurysm clip, while dural rents, holes, or other defects are repaired either directly with suture or, more commonly by placement of a muscle pledget along with gelfoam and fibrin sealant [8].

For growing SDHs caused by SIH, some authors recommend urgent neurosurgical treatment for patients with clinical deterioration and consciousness impairment [2,5]. Surgery is often, but not always, successful in relieving symptoms due to a localized CSF leak [2]. In some cases, in which SDH becomes symptomatic, urgent irrigation of the hematoma should be considered before EBP. In these cases, we suggest early lumbar EBP after SDH evacuation before the patient gets up to prevent frequent recurrences of SDH caused by underlying CSF leakage and promote an early recovery [5]. In a case series, SDH evacuation not followed by EBP was associated with a high SDH recurrence rate (two-thirds of patients) [5].

Case Report

A 42-year-old man with no medical history was admitted to the neurology department of Imam Khomeini Hospital on 2022/7/3 due to headache. He was suffering from Sudden headache since two months ago, which has gradually improved, but has intensified since six days before hospitalization. The history of trauma, accident, and intense sports activity is not mentioned. The headache is positional (especially when standing) and radiates to the back of the eyes and neck. The patient's pain was immediately caused by changing the position from lying to sitting and standing and lasted for 10 to 20 seconds and was accompanied by tinnitus. He did not have nausea and vomiting, blurred vision and diplopia, convulsions and loss of consciousness. Fatigue and sleepiness and loss of daily activities are mentioned by the patient. Pupils were mid-sized and similar and

reactive to light. Muscle force on both sides was similar and 5/5, reflexes were similar and +2. In ophthalmoscopy, the optic disc was sharp and normal on both sides and there was no papillary edema. Mini mental score was 30/30. Other neurological examinations were reported as normal in the patient's record.

In MRI, subacute bilateral supratentorial hematoma with a thickness of 19 mm and without midline shift and compressive marks and prominent enhancement of leptomeninges and sagging of brain have been reported. In the examination, the patient is alert, alert and obedient. Biochemistry and coagulation tests were normal. The patient reported the intensity of the pain in the most severe state with the VNS (verbal numeric scale no. 9). In the neurology department, he was treated with Novafen, Dexamethasone and Pantoprazole. With the diagnosis of SDH (chronic subdural hematoma) in the context of SIH, a pain consultation was done by a neurologist and it was suggested to perform EBP.

On 2022/7/5 he was admitted to Reyhaneh's operating room in Imam Khomeini Hospital to perform an epidural blood patch. After establishing an intravenous line and standard monitoring in the prone position, intravenous sedation was prescribed. under C-arm guide with a TOUHY 18 G needle approach to the epidural space via L1-L2 interlaminar space was performed using the" loss of resistance" method, and the exact location of the needle was ensured by performing three face, lateral, and oblique views and injecting contrast material. Then, under sterile conditions, 20 cc of the patient's autologous blood was taken from the dorsal vein of the left hand and injected into the epidural space. During the injection, the patient did not have any local pain or radiculopathy. Half an hour before the procedure, the patient was in the supine and Trendelenburg position, and this condition continued during the patient's recovery. After the procedure, the patient mentioned a clear reduction in pain. A written recommendation was made in the patient's file to maintain the Trendelenburg position for 24 hours and to avoid straining and activity during two weeks after the procedure. In the follow-up, the patient had a 100% pain reduction after 24 hours, one week, and two weeks. The size of the subdural hematoma in the MRI two weeks later is reduced by 10%, and in the MRI four months after the procedure, the complete absorption of the hematoma and the normalization of the previous findings were reported.

After studying the contents of the patient's file, medical history and clinical examination were performed. PUBMED, SCOPUS, WEB OF SCIENCE and GOOGLE SCHOLAR databases were searched with the keywords "spontaneous intracranial hypotension, CSF leak, epidural blood patch". The chapters related to SIH were studied and analyzed in the textbooks "Practical of Pain Medicine Management (6th edition 2022)" (3) and" ESSENTIAL OF PAIN MEDICINE (4th edition 2018)" [13]. Among 35 articles, 11 articles including two case series, one observational study, three review articles and 5 original articles related to the years 2006 to 2021 were selected by the authors to conduct the review. The follow-up of the patient's condition was done 24 hours after the epidural patch and one and two weeks and one and three months later. Also, the patient's MRI was done two weeks, one month and three months after discharge.

Discussion

Spontaneous intracranial hypotension (SIH) is a debilitating condition with protean symptoms, which is often misdiagnosed at initial presentation. The most common cause of SIH is a spinal CSF leak. Patients often have an underlying connective tissue disorders, though underproduction or increased absorption of CSF, dural elasticity, and minor trauma, including disk herniation, may all be contributing factors [10]. Although headache is often positional, non-positional and continuous headache also occurs in patients with subdural effusion in the chronic stages of the disease [6].

In an observational study on 568 patients by Schievink et al., the causes of spinal leakage are divided into four types: type one, dural tear (26.6%), type two, meningeal diverticulum (42.3%), type three, liquid fistula. brain to vein (2.5%) and type four, unspecified type (28.7%) [12].

The typical findings on cranial MR imaging in cases of spontaneous intracranial hypotension consist of subdural fluid collections, enhancement of the pachymeninges, engorgement of venous structures, pituitary hyperemia, and sagging of the brain [6].

The association between intracranial hypotension and SDHs has been known since at least the 1950s. SDH may occur as a result of tearing of bridging veins in the layer of the dural border cells and usually develop into chronic SDH. SDH which is limited in volume usually resolves spontaneously after normal CSF volume has been restored. However, a significant volume of hematoma may require surgical treatment. The SDH among SIH patients is predominant in males, whereas SIH without SDH is predominant in females [5].

It is important to note that not all cases of postural headache are due to a CSF leak. Rarer conditions, including postural tachycardia syndrome and increased compliance of the lower spinal CSF space should be considered, especially when all tests are negative [2].

Diffuse dural enhancement is the most striking and valuable finding for the diagnosis. This finding presumably is the result of either tearing or vasodilation of the meningeal vasculature, which lacks a tight junction. The most important intracranial complications of SIH are downward displacement of the cerebellar tonsils and subdural hematoma. Subdural hematoma usually has a chronic pattern [6]. Because of this chronic hematoma pattern, neurological findings are rare [5].

Caudal displacement of the cerebral tonsils may be misdiagnosed as tonsillar herniation with intracranial hypertension in the acute stage of the illness, or idiopathic

Chiari type I malformation in the chronic stage of the illness. An important difference is the panpachymeningeal enhancement on MR imaging [6].

Scientific documents show that patients with SDH have a tendency to recurrence of hematoma, persistence of symptoms and deterioration of clinical condition, therefore, they recommend performing an epidural patch before surgery and basically draining the hematoma except in emergency situations and the presence of a large hematoma with symptoms related to the mass effect (ME) and dilated or non-parallel pupils are not necessary and sometimes even worsen the patient's condition [5].

Persistent non-positional headache after epidural patch is a predictive factor for subdural hematoma and an indication for surgery [5]. Some authors use the following therapeutic strategy to treat SIH patients with SDH: initial EBP and, if SDH is thick, SDH surgery is done immediately after EBP. The volume of the hematoma was defined as “thick” when the maximum hematoma thickness was> 15 mm [5]. Chung et al. reported that the onset age of SIH with SDH was 44.9 years old on average, which is significantly older than SIH without SDH (average: 36.6 years old) [7]. it is reported that many cases have bilateral SDHs (90%). Gd enhancement was useful to distinguish SIH with SDH from SDH unrelated to SIH [7].

Schievink et al have recommended that a large number of patients with SDH with mass-related symptoms can be treated with an epidural patch without hematoma drainage, provided that the patient's symptoms improve after the patch, and Chang et al. also recommend performing an epidural patch before surgery [7].

In the report of Yingfeng Wan et al., there were 15 cases of SIH, eight patients had recurrence of hematoma after surgery, and these were cases in which there was CSF leak in the spinal area and the treating doctors did not pay attention to it [6].

Loya et al. reviewed 29 cases of SIH associated with coma. EBPs were successful in ameliorating the comatose state in 85%. Evacuation of SDHs may be performed for large SDH with mass effect or for when dilated or asymmetric pupil is present. however, Loya et al. concluded that in most cases evacuation of the hematoma is not necessary and may give rise to worsened outcomes [7].

Koichi TaKahashi et al recommend that when faced with a middle-aged patient with bilateral subdural hematoma without a history of trauma, an MRI with gadolinium injection should be performed first to rule out SIH, and if the patient's condition worsens during the subdural hematoma surgery, Let the surgical incision be closed in order to prevent the intertracheal space from being exposed to atmospheric pressure, and an epidural patch will be applied immediately [7]. In our patient, the age of the patient, the absence of underlying disease and coagulation disorder, the absence of history of trauma and intense activity, and the radiological findings and strong clinical suspicion of the treating neurologist led to the timely and correct treatment of the patient without unnecessary surgical intervention. done and the desired clinical improvement should take place immediately and the improvement of radiological findings after three months.

Conclusion

Spontaneous intracranial hypotension (SIH) is a highly misdiagnosed and underdiagnosed disorder and requires a high index of suspicion for diagnosis. During the last decades, a much larger number of spontaneous cases are identified. Literature is a bit confusing, with some authors recommending evacuation of subdural fluid in cases of deteriorating consciousness and few others recommending EBP first even in patients with comatose state but epidural patch is often an important part of treatment.

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