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Intracranial hypotension is a rare cause of orthostatic headache: a review of the etiology, treatment and prognosis of 13 cases

Ortostatik baş ağrısının çok nadir bir nedeni: İntrakranial hipotansiyonlu 13 olgunun etyoloji, tedavi ve prognozlarının gözden geçirilmesi

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Summary

Objectives: The aim of this investigation is to examine the causes, clinical picture, treatment, and prognosis of spontaneous intracranial hypotension, a rare cause of orthostatic headache, among the cases presenting in our clinic.

Methods: Thirteen cases (5 males and 8 females), diagnosed with spontaneous intracranial hypotension in our clinic between January 1st, 2009 and October 30th, 2011, were included in this study. The presenting symptoms, treatment, findings on cranial magnetic resonance imaging, cerebrospinal fluid pressure measured at lumbar puncture (in available patients), and the healing period of the patients were recorded.

Results: Five patients with orthostatic headache and accompanying symptoms were treated with bed rest, increase in oral fluid intake, intravenous hydration and caffeine, and experienced a complete recovery. Complete recovery was observed in two patients (15.3%) within 10 days, in another two (15.3%) within 15 days and in one patient (7.6%) within 21 days. Headache and other clinical symptoms significantly regressed within 30 days in four patients (37.6%) who received similar treatment, but a mild headache persisted intermittently during follow-up in these individuals. As the headache had not resolved after 30 days, an epidural blood patch was applied in these four cases (37.6%) and the clinical picture completely improved within 10 to 15 days.

Conclusion: Spontaneous intracranial hypotension should primarily be suspected in cases complaining about postural headache and contrast-enhanced cranial imaging should be performed. The presence of cranial nerve paralysis and pyramidal tract signs should b considered. Conservative treatments should be considered initially, however if conservative treatments fail, epidural blood patches must be applied.

Key words: Etiology; intracranial hypotension; prognosis; treatment.

Özet

Amaç: *Ortostatik başağrısının nadir bir deneni olan spontan intrakraniyal hipotansiyonun nedenleri, kliniği, tedavi ve prognozunu kliniğimizde izlenen olgular eşliğinde gözden geçirmek.*

Gereç ve Yöntem: *Ocak 2009-30 Ekim 2011 tarihleri arasında kliniğimizde tanı alan 5 erkek ve 8 kadın olmak üzere 13 tane spontan intrakraniyal hipotansiyon olgusu çalışmaya dahil edildi. Hastalar geliş semtomları, uygulanan tedavi, kraniyal MRG bulguları, uygun hastalarda yapınla BOS basınç değerleri ve klinik iyileşme süreleri değerlendirilerek incelendi.*

Bulgular: *Ortostatik baş ağrısının ve eşlik eden bulguların yatak istirahati, oral sıvı alımının artırılması, intravenöz hidrasyon ve kafein tedavisi uygulanan iki hastada (%15.3) 10 günde iki hastada (%15.3) 15 günde bir hastada (%7.6) 21 günde tamamen düzeldiği görüldü. Benzer tedavi uygulanan dört (%37.6) olguda başağrısı ve diğer klinik özelliklerinin 30 gün içerisinde belirgin olarak gerilediği ancak periyodik takiplerinde hafif şiddette baş ağrılarının aralıklı olarak devam ettiği gözlendi. 30 gün sonrasında ortostatik baş ağrısı gerilemeyen olgulara uygulanan epidural kan yaması sonrasında 10-15 gün içerisinde kliniklerinin tamamen düzeldiği görüldü.*

Sonuç: *Postural baş ağrısı tarifleyen olgularda öncelikle spontan intrakraniyal hipotansiyondan (SIH) mutlaka şüphe edilmeli ve kontrastlı kraniyal görüntüleme yapılmalıdır. Kraniyal sinir paralizileri ve piramidal traktus bulguları olabileceği unutulmamalıdır. Epidural kan yamaları konservatif tedavinin yetersiz kaldığı olgulara uygulanmalıdır.*

Anahtar sözcükler: Etyoloji; intrakraniyal hipotansiyon; prognoz; tedavi.

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Introduction

Spontaneous intracranial hypotension (SIH) is a rare condition with an annual incidence of 5/100000. Female to male ratio is 2/1, with a peak incidence in the fourth decade of life. SIH syndrome is caused by the leakage of cerebrospinal fluid (CSF) within the spinal epidural space, resulting in low CSF volume.[1] It is defined as a CSF opening pressure lower than 60 mm H_2O,[2] however the pathogenesis of CSF leakage remains unclear. Some authors reported that CSF leakage could be demonstrated on T2-weighted magnetic resonance (MR) myelography sequences.[3,4] Although clinical signs of SIH may exhibit variations, postural headache is the most characteristic feature. The diagnosis of idiopathic intracranial hypotension is made based on the criteria of International Classification of Headache Disorders (ICDH-2). According to the recent revision of ICDH-2, SIH is defined as a headache that begins or worsens within 15 minutes of moving to an upright position and relieves within 15 to 30 minutes of moving to supine position.[5]

Diagnosis of SIH has been revolutionized by the advance in MRI techniques. Cranial MRI findings include pachymeningeal thickening and contrast uptake, subdural fluid collection, obstruction of venous outflow, dural thickening, pituitary hyperemia, and inferior displacement of the cerebellar tonsils and brain stem.[6,7]

The aim of the present study is to present the presenting symptoms, clinical characteristics, etiology, treatment and prognosis of 13 patients (5 males and 8 females), who were diagnosed with intracranial hypotension and followed up in our clinic between 1 January 2009 and 30 October 2011.

Materials and Methods

Demographic characteristics, presenting complaints, neurological examination findings, imaging findings and prognosis of 13 patients, who were diagnosed with intracranial hypotension according to clinical findings, findings on MRI and lumbar puncture (LP) and were followed up in our clinic for two and a half years between January 1st, 2009 and October 30th 2011, were retrospectively reviewed. In order to identify the etiology of intracranial hypotension, blood biochemistry, complete blood count, erythrocyte sedimentation rate, prothrombin time, and Activated Partial Thromboplastin Time (aPTT) of the patients were obtained. Serological tests included rheumatoid factor, Protein C and S, antithrombin III, VDRL, ANA and TSH. CSF analysis was performed on 7 suitable patients, who did not develop intracranial hypotension after spinal anesthesia or LP. EEG recordings and anticardiolipin antibody values were also obtained. Imaging studies consisted of cranial CT or MRI, MR venography, and angiography. Statistical analysis was not done because of the small sample size and high probability of error.

Results

Of the cases, 5 (38.4%) were male and 8 (61.53%) were female. The mean age of the patients was 40.1±19 years (range, 26-67 years); the mean age of the males was 46, whereas the mean age of the females was 36.5±16 years. The neurological symptoms and signs of the patients are presented in Table 1. Headache that worsened in the upright position and relieved in the supine position was the most common symptom observed in all patients (100%). All the patients complained about throbbing headache that begins in the neck and spreads all over the head. Seven patients (53.8%) had a history of sudden onset headache triggered by coughing or straining. The symptoms and signs of the patients were; fever (n=1; 7.6%), tinnitus (n=7; 53.8), diplopia (n=5; 38.4%), blurred vision (n=8; 61.5%), facial anesthesia (n=6; 46.1%), iron deficiency anemia (n=7; 53.8%), and cranial nerve paralysis (n=2; 15.3%). Three patients (23%) had a history of neurological disease and four (30.7%) patients had a concomitant systemic disease. Vasculitis parameters were positive in two (15.3%) patients. Moreover, six (46.1%) patients had a history of spinal anesthesia and one patient (7.6%) had an additional neurological deficit. None of the patients had papilla edema.

Clinical characteristics of the patients, CSF pressure values taken at lumbar puncture, which was performed in available patients that did not develop SIH following spinal anesthesia, treatment methods and healing periods are summarized in Table 2. Serological, bacteriological, pathological and cytologi-

Table 1. Clinical symptoms and signs

Symptoms and signs	Number of patients	
	n	%
Headache that worsens in the upright position	13	100
Headache that relieves in supine position	1	100
Neck stiffness-discomfort	13	100
Sudden onset headache (triggered with coughing or straining)	7	53.8
Fever	1	7.6
Tinnitus	7	53.8
Aural fullness	8	61.5
Horizontal diplopia	5	38.4
Blurred vision	8	61.5
Facial anesthesia	6	46.1
Iron deficiency anemia	7	53.8
Cranial nerve paralysis	2	15.3
Underlying neurological disease	3	23.0
Positivity of vasculitis parameters	2	15.3
Concomitant systemic disease	4	30.7
History of spinal anesthesia	6	46.1
Presence of papilla edema	0	0
Additional neurological deficit	1	7.6

Table 2. Clinical characteristics, CSF findings, treatment methods and healing periods prognosis of the patients

No	Gender	Clinical signs	CSF pressure	Treatment	Healing period
1	Female	OH+D+FA	6 cmHg	AH	10 days
2	Male	OH+D+BV+FA+B6C	No LP	AH	15 days
3	Female	OH+BV+AF	No LP	AH	10 days
4	Female	OH+T+BV+AF	5 cmHg	AH+P	35 days
5	Female	OH+D+BV+FA	No LP	AH	21 days
6	Female	OH+T+D+BV+FA+AF	6 cmHg	AH	21 days
7	Female	OH+T+BV+AF	6 cmHg	AH	30 days
8	Male	OH+T+BV+AF	5 cm Hg	AH+P	40 days
9	Female	OH+T+FA+AF	4 cm Hg	AH+P	45 days
10	Male	OH+T+D+BV+AF	No LP	AH	30 days
11	Male	OH	No LP	AH+P	40 days
12	Male	OH+BV+FA+L7C+PIH	No LP	AH	30 days
13	Female	OH+FA+AND	No LP	AH	15 days

CSF: Cerebrospinal fluid; OH: Orthostatic headache; D: Diplopia; FA: Facial anesthesia; B6C: Bilateral 6th cranial nerve paralysis; AH: Abundant hydration; BV: Blurred vision; AF: Aural fullness; T: Tinnitus, P: Patch; L7C: Left facial nerve paralysis; PIH: Posture-independent headache; AND: Additional neurological deficit.

cal examinations of CSF, which were performed for differential diagnosis due to dural enhancement revealed no abnormal finding.

All patients had orthostatic headache. The headache of the patient who developed cerebral venous thrombosis (CVT), gained a resistant nature inde-

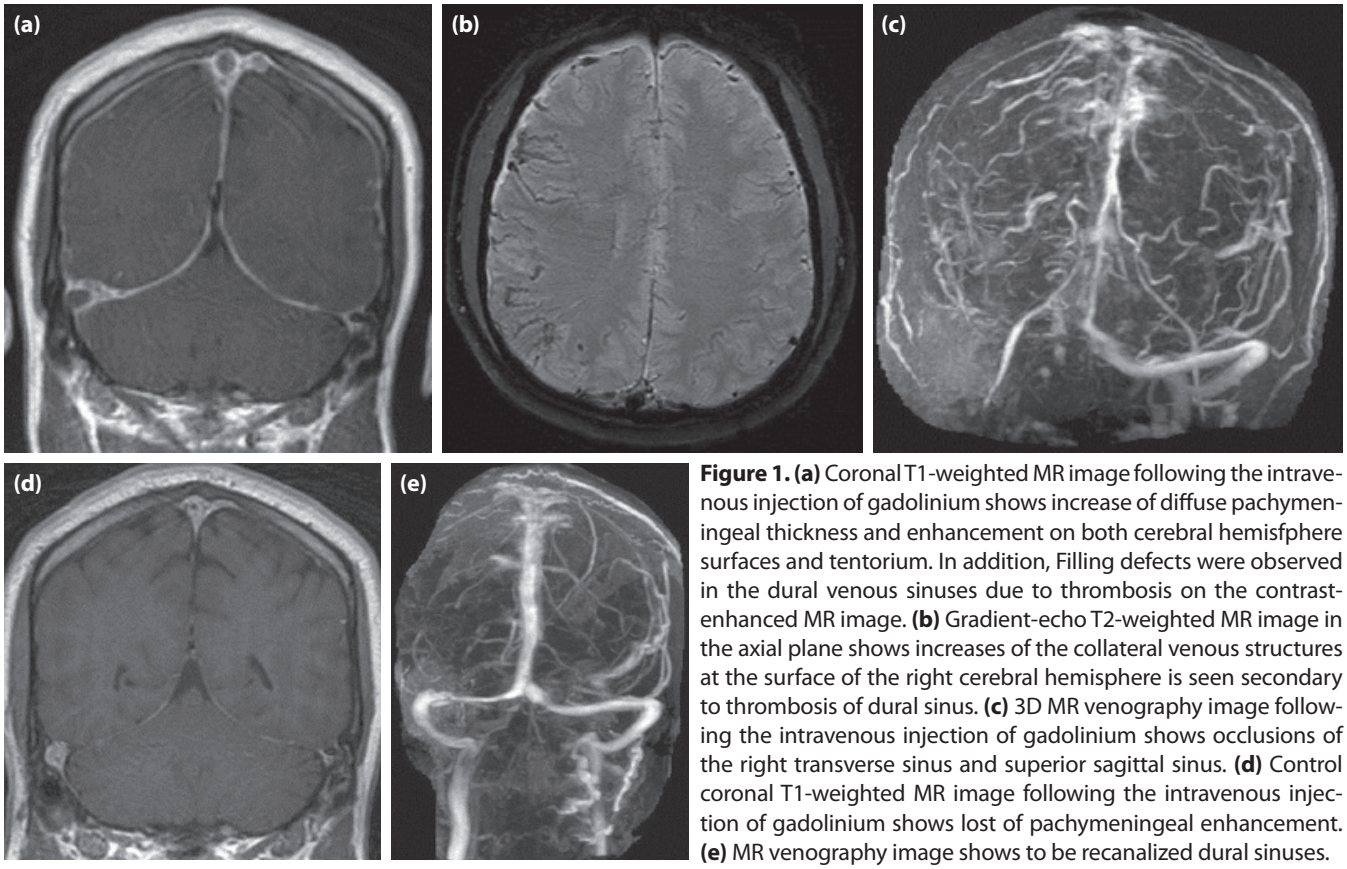

Figure 1. (a) Coronal T1-weighted MR image following the intravenous injection of gadolinium shows increase of diffuse pachymeningeal thickness and enhancement on both cerebral hemisfphere surfaces and tentorium. In addition, Filling defects were observed in the dural venous sinuses due to thrombosis on the contrast-enhanced MR image. **(b)** Gradient-echo T2-weighted MR image in the axial plane shows increases of the collateral venous structures at the surface of the right cerebral hemisphere is seen secondary to thrombosis of dural sinus. **(c)** 3D MR venography image following the intravenous injection of gadolinium shows occlusions of the right transverse sinus and superior sagittal sinus. **(d)** Control coronal T1-weighted MR image following the intravenous injection of gadolinium shows lost of pachymeningeal enhancement. **(e)** MR venography image shows to be recanalized dural sinuses.

pendent of posture. Four patients (30.7%), who had persistent headache despite 30 days of symptomatic medical therapy, underwent epidural blood patches. Headache was completely relieved in these patients within 10 to 15 days after blood patch application. Cranial MRI of the 12th case revealed coronal T1-weighted MR image following the intravenous injection of gadolinium shows increase of diffuse pachymeningeal thickness and enhancement on both cerebral hemisfphere surfaces and tentorium. In addition, filling defects were observed in the dural venous sinuses due to thrombosis on the contrast-enhanced MR image (Figure 1a-c). Complete regression was observed on the control MRI of this patient, which was obtained one month later. Control coronal T1-weighted MR image following the intravenous injection of gadolinium shows lost of pachymeningeal enhancement, and MR venography image shows to be recanalized dural sinuses (Figure 1d, e). Cervical MRI of the 9th case revealed MRI myelography image shows ectasias and diverticular along the spinal nerve roots (Figure 2). Axial T1-weighted of the 5th case revealed MR image following the intravenous injection of gadolinium shows widespread increase of pachymeningeal enhancement (Figure 3a) and) in the contrast-enhanced sagittal MR venography source image shows engorgement of dural sinuses (Figure 3b). Axial FLAIR images of the 4th case revealaed at the level of posterior fossa and supratentorial shows A) Along the internal auditory canal (Figure 4a), in the tentorium (Figure 4b), and dural thickening and increase of intensity is seen along the surface of cerebral hemispheres (Figure 4c). Sagittal T2-weighted brain MR image shows sagging at the brain stem

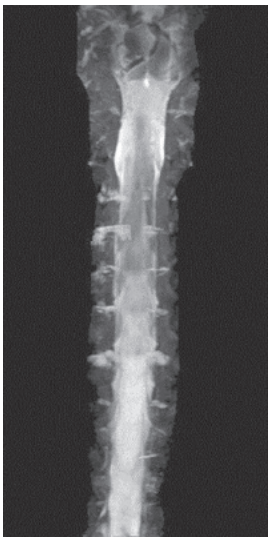

Figure 2. Cervical MRI myelography image shows ectasias and diverticular along the spinal nerve roots.

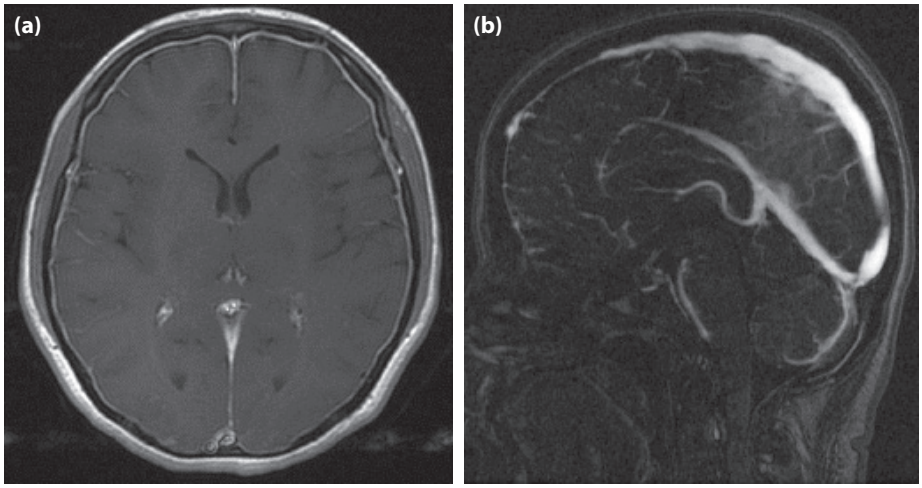

Figure 3. (a) Axial T1-weighted MR image following the intravenous injection of gadolinium shows widespread increase of pachymeningeal enhancement. **(b)** In the contrast-enhanced sagittal MR venography source image shows engorgement of dural sinuses.

and caudal displacement of optical chiasm (Figure 4d) and post-treatment control FLAIR image shows disappearance at findings (Figure 4e, f). Coronal T1-weighted MR image of the 6th case revealaed following the intravenous injection of gadolinium shows increase diffuse uniform pachymeningeal thickness and enhancement on cerebral hemisfpheres surfaces and tentorium (Figure 5a). Control coronal T1-weighted MR image shows findings seems to vanish completely (Figure 5b). A coronal T2W and Coronal T1-weighted MR image of the 7 th case revealaed following the intravenous injection of gadolinium shows caudal displacement of optical chiasm and increases of enhancement and expansion

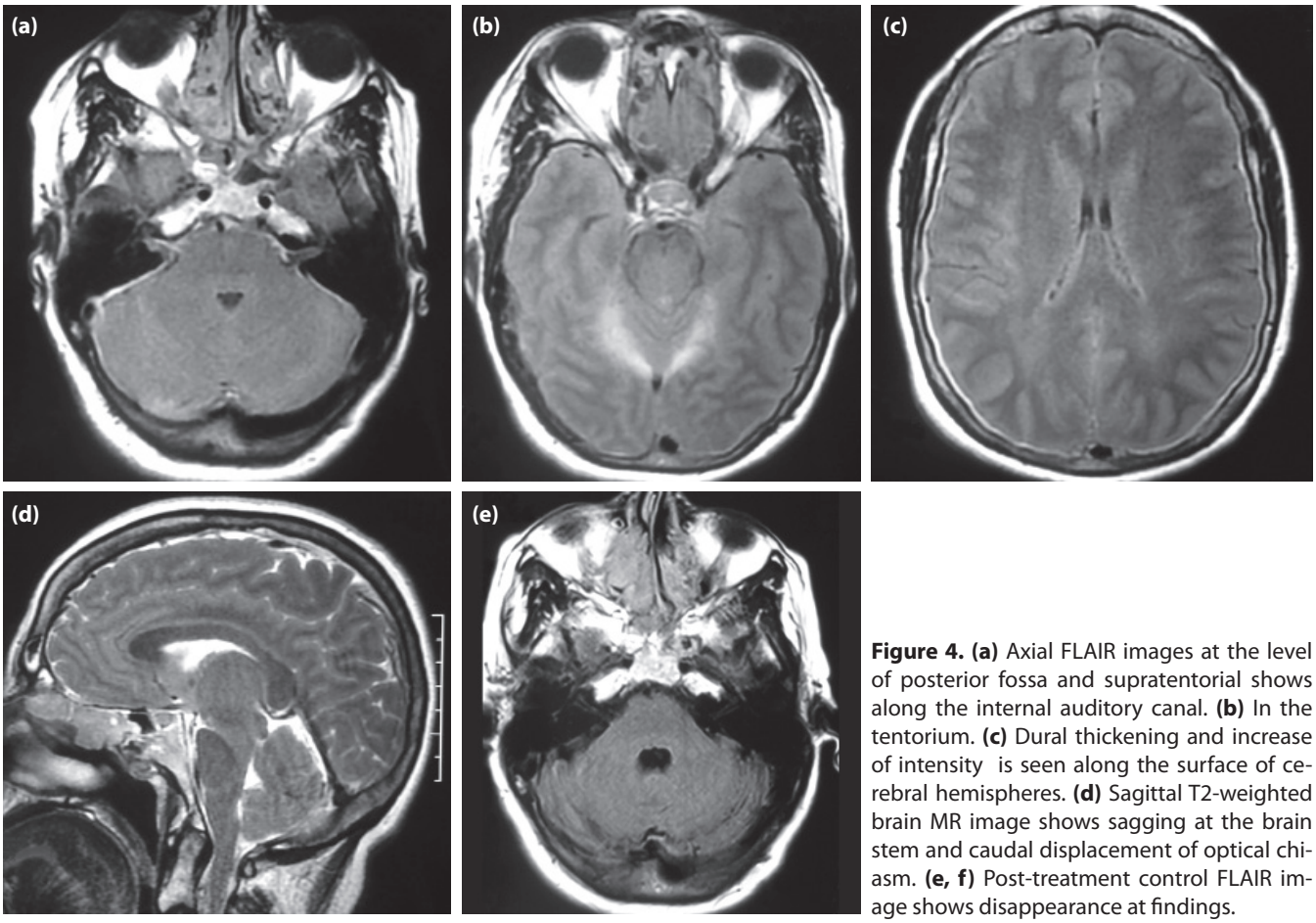

Figure 4. (a) Axial FLAIR images at the level of posterior fossa and supratentorial shows along the internal auditory canal. **(b)** In the tentorium. **(c)** Dural thickening and increase of intensity is seen along the surface of cerebral hemispheres. **(d)** Sagittal T2-weighted brain MR image shows sagging at the brain stem and caudal displacement of optical chiasm. **(e, f)** Post-treatment control FLAIR image shows disappearance at findings.

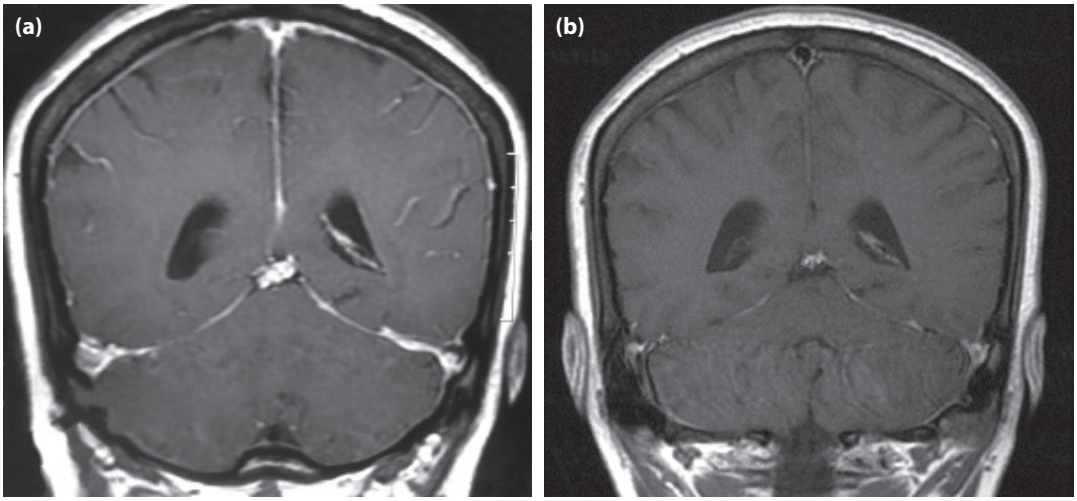

Figure 5. (a) Coronal T1-weighted MR image following the intravenous injection of gadolinium shows increase diffuse uniform pachymeningeal thickness and enhancement on cerebral hemisfpheres surfaces and tentorium. **(b)** Control coronal T1-weighted MR image shows findings seems to vanish completely.

due to hyperaemia at cavernous sinuses and pituitary gland (Figure 6a, b). İn addition to other findings, contrast-enhanced sagittal T1-weighted MR image shows sagging at the upper brain stem and expansion of the dural venous sinuses (Figure 6c).

Discussion

Clinical signs, typical MRI findings and treatment options of SIH have been clearly defined in the literature.[8] Decreased CSF pressure and pachymeningeal and dural thickening on brain MRI are detected in middle-aged subjects with orthostatic headache. The Monroe-Kellie hypothesis states that, the total intracranial volume (intracranial blood, CSF and brain tissue) must be constant in an intact cranium. [9,10] SIH usually occurs due to spontaneous CSF leaks in the inferior cervical and superior thoracic spine. Mechanical stress, meningeal diverticula and connective tissue diseases have been reported as the potential risk factors for the development of SIH.

The annual incidence of SIH has been reported to be 5/100000, being more prevalent in women in the 4th decade of life.[8] In this case series, the mean age was 40.1±19 years and there was a slight female predominance. The disease has a wide clinical spectrum. Patients may present with orthostatic headache, nausea, vomiting, neck stiffness, and cranial nerve paralysis.[11] Although the abducens nerve is the mostly frequently involved cranial nerve in SIH, optic, oculomotor and trigeminal nerve paralysis may also be seen.[12] In the present study, all patients had orthostatic headache and two patients had cra-

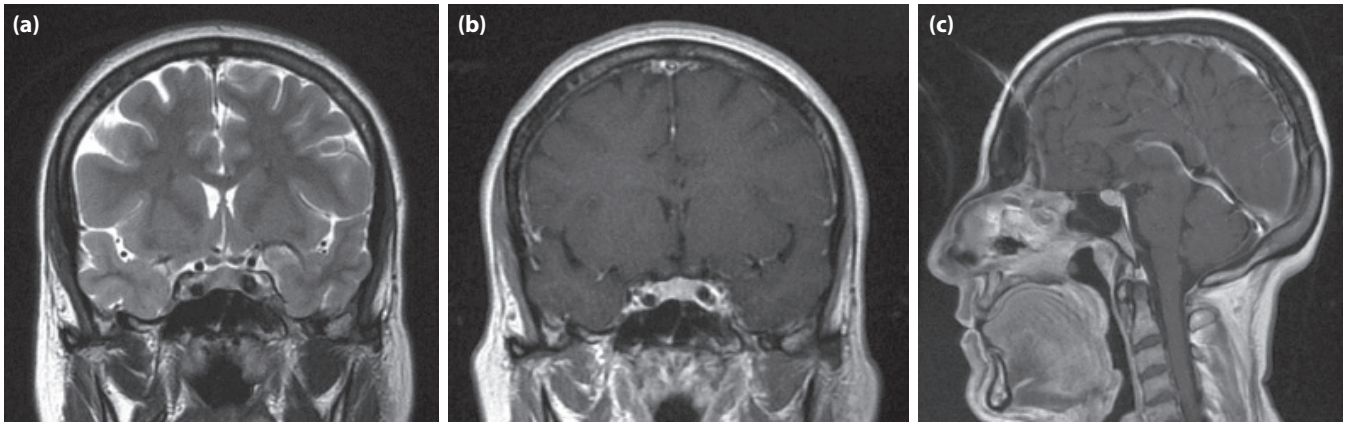

Figure 6. (a, b) A coronal T2W and coronal T1-weighted MR image following the intravenous injection of gadolinium shows caudal displacement of optical chiasm and increases of enhancement and expansion due to hyperaemia at cavernous sinuses and pituitary gland. **(c)** Contrast-enhanced sagittal T1-weighted MR image shows sagging at the upper brain stem and expansion of the dural venous sinuses.

nial nerve palsies (sixth cranial nerve involvement in one patient, and seventh cranial nerve involvement in the other).

Brain MRI shows diffuse pachymeningeal gadolinium enhancement, subdural fluid collection, and downward displacement of the brain. Both supratentorial and infratentorial pachymeninges, including cerebral and cerebellar convexities, and falx and tentorium are involved.[13] Brain MRI findings were consistent with SIH in all our cases.

Spontaneous intracranial hypotension may occur after lumbar puncture or spinal anesthesia. SIH may also develop at any time the dura mater is damaged, such as after craniotomy or spinal trauma.[14] CSF opening pressure is always low in cases with SIH. Six of the present cases had a history of spinal anesthesia, whereas one had a history of lumbar puncture. As the clinical symptoms and MRI findings were consistent with SIH, lumbar puncture was not performed in these patients, and, the diagnosis was based on clinical and radiological findings.

A headache that worsens in the afternoon is not a common condition. However, orthostatic headache, which occurs due to SIH following CSF leakage, worsens in the late morning or in the afternoon.[15] This phenomenon is called as the second half of the day headache.[16] This is corroborated by the intracranial hypotension signs on the cranial MRI of the cases with second half of the day headache secondary to CSF leakage. Moreover, orthostatic characteristics and MRI changes have been found to be associated with dizziness, tinnitus, aural fullness, neck stiffness, and horizontal diplopia.[17] In addition to orthostatic headache, eight of the present cases had tinnitus, nine had aural fullness and five had horizontal diplopia, whereas all of the cases had neck stiffness or discomfort.

CVT is one of the most important complications of SIH. It is a rare complication, occurring in only 2% of the cases. According to the Monroe-Kellie hypothesis, the decrease in intracranial blood volume is compensated by the dilatation of the cerebral veins.[10] Furthermore, CSF loss reduces the CSF absorption into the cerebral venous sinuses leading to an increase in blood viscosity in the cerebral compartment. One of the present cases had CVT associated with a similar mechanism.

There are several options for the treatment of SIH. Bed rest is of great importance and is adequate for the treatment of most cases. Increased oral fluid intake and oral caffeine may provide symptomatic relief. Caffeine is an adenosine receptor antagonist and produces an increase in CSF production secondary to an increase in cerebral blood flow. Sedatives and intravenous hydration therapy may also be applied. Despite unsuccessful treatment with high dose steroids in the previous years, cases that responded to oral steroid therapy have been reported in the recent years.[18] In the present study, all cases benefited to varying degrees from bed rest, increased oral fluid intake, and concurrent intravenous hydration and caffeine therapy.

Epidural blood patch, epidural saline or dextran infusion are applied in persistent cases.[19,20] Epidural blood patch, using 10-15 ml of blood, is the main therapy. It is applied directly to the CSF leakage site if the leak is detected, or to the lumbar epidural region if the leak cannot be detected.[21,22] As CSF leaks usually occur in the thoracic region, high volume blood patches should be applied in the thoracolumbar region, and the patients should be placed in the trendelenburg position for 20-30 minutes and then should be turned to prone position for 20-30 minutes; this positioning allows blood to travel over many spinal segments toward the site of the leak. Continuous lumbar epidural saline infusion is another treatment method.[23] Although the method is efficient at the beginning, recurrence is common.[24] Surgical repair may be considered if conservative therapy proves unsuccessful. In the present case series, epidural blood patch was performed in cases with headaches that did not regress within 30 days despite treatment with analgesics, bed rest and hydration. None of the patients, who underwent blood patches, developed recurrence.

Some authors state that patients with SIH should be evaluated periodically, as headaches that are usually self-limiting and show a favorable response to conservative therapies within a few weeks may sometimes become chronic or recur.[25,26] In fact, mild orthostatic headache of four patients, who received

medical therapy alone, continue to occur intermittently. These patients do not have any radiological signs of recurrence or severe orthostatic headache, and they remain under follow up in our clinic.

Pathophysiological mechanisms underlying epidural lumbar blood patch may be associated with an increase in spinal epidural pressure that leads to a equilibrium in epidural venous pressure. This suggests that the main reason for intracranial hypotension is not the CSF leakage but that the negative epidural pressure within the spinal canal results in aspiration of CSF on dural surface and at the origin of spinal roots. Particularly activities like standing or walking, may contribute to the maintenance of negative pressure within the spinal canal via the epidural venous drainage towards the inferior vena cava.[9,27] Symptoms of headache did not recur in any of the present patients who underwent blood patches. Dural and pachymeningeal thickening signs detected on the cranial MRI were significantly regressed.

In conclusion, SIH is a less known syndrome and may usually be misdiagnosed despite its characteristic clinical picture and typical MRI findings. SIH should be particularly suspected in cases that complain about postural headache and contrast-enhanced brain MRI should performed. Epidural blood patches should be applied when conservative therapy remains inadequate. It should be kept in mind that headaches may become chronic or recur; therefore, these patients should be invited to periodic follow-ups. Moreover, in order to prevent chronic headache syndrome and secondary complications such as CVT and subdural hemorrhage, early diagnosis and treatment of SIH is of great importance.

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