RESEARCH

Rebound high-pressure headache after treatment of spontaneous intracranial hypotension

MRV study

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Abstract

Background

Rebound high-pressure headaches may complicate treatment of spontaneous intracranial hypotension (SIH), but no comprehensive study of such patients has been reported and little is known about its frequency and risk factors. We therefore studied patients undergoing treatment for SIH and performed magnetic resonance venography (MRV) to assess for cerebral venous sinus stenosis, a risk factor for idiopathic intracranial hypertension.

Methods

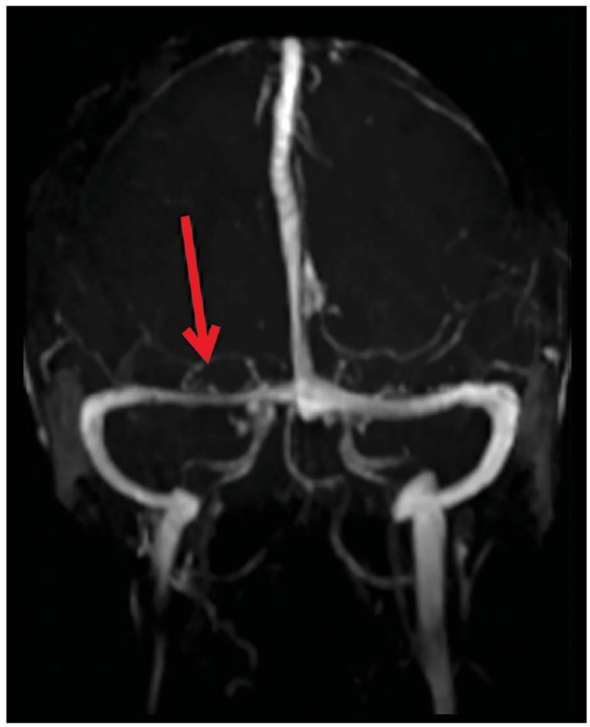
We studied a consecutive group of patients who underwent treatment for SIH. Rebound high-pressure headache was defined as a reverse orthostatic headache responsive to acetazolamide. MRV was obtained in all patients and lateral sinus stenosis was scored according to the system published by Higgins et al., with 0 being normal and 4 signifying bilateral signal gaps.

Results

The mean age of the 46 men and 67 women was 45.9 years (range 13–71 years) at the time of onset of SIH. Rebound high-pressure headache was diagnosed in 31 patients (27.4%); 14% of patients with an MRV score of 0, 24% with a score of 1, and 46% with a score of 2 or 3 ($p = 0.0092$). Also, compared to SIH patients who did not develop rebound high-pressure headaches (n = 82), those with rebound high-pressure headaches were younger, more often female, and more often had an extradural CSF collection on spinal imaging.

Conclusions

Rebound high-pressure headache occurs in about one-fourth of patients following treatment of SIH and is more common in those with restriction of cerebral venous outflow.



Spontaneous intracranial hypotension (SIH) is an important, treatable, and increasingly recognized cause of headaches.[1-3] Although a wide variety of headache types and associated symptomatology have been described in SIH, the vast majority of patients present with classic orthostatic headaches.[1-3] Following treatment, some patients develop a reversal of their orthostatic headaches and their headache is improved in the upright position and worse lying

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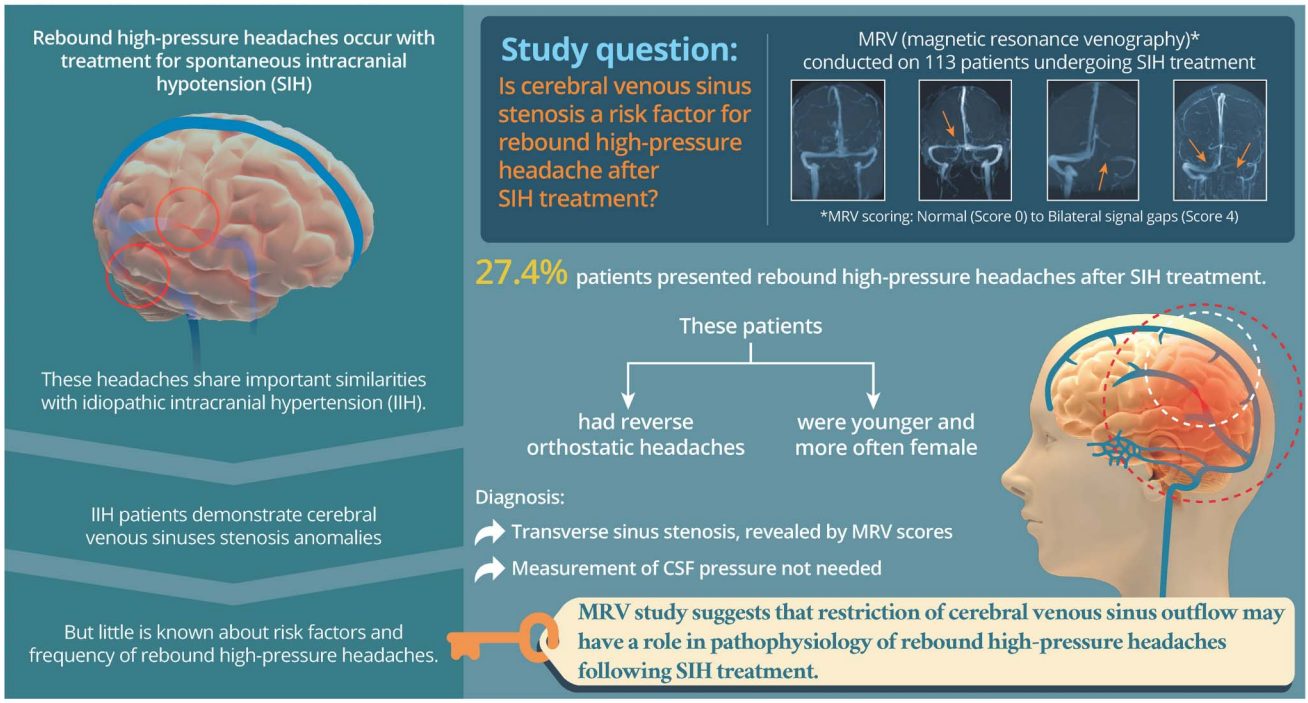

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down. A diagnosis of rebound high-pressure headache is then made and this is generally responsive to a course of oral acetazolamide. Although rebound intracranial hypertension following treatment of SIH has been recognized since the 1990s, much uncertainty remains regarding this complication of SIH treatments.[4-11] The frequency of rebound high-pressure headache is unknown, but has been reported to occur in 0%–20% of patients following treatment of SIH.[4,8,10]

Significant anomalies of the cerebral venous sinuses have been detected using magnetic resonance venography (MRV) in patients with idiopathic intracranial hypertension (IIH), with transverse sinus stenosis being the most frequently reported.[12-17] IIH has some important similarities with rebound intracranial hypertension following SIH treatment.[18,19] We therefore studied MRV in a group of patients undergoing treatment for SIH.

Methods

Standard protocol approvals, registrations, and patient consents

This study was approved by our medical center's institutional review board.

We studied a group of consecutive patients who underwent percutaneous or microsurgical treatment of SIH. The diagnosis of SIH was based on ICHD-III criteria,[20] with minor modifications. These criteria require objective evidence of SIH, consisting of brain MRI showing stigmata of SIH (i.e., pachymeningeal enhancement or brain sagging), spinal imaging showing a CSF leak (i.e., the presence of extradural CSF), or low CSF opening pressure (i.e., <6 cm H2O). The modification consists of also including patients who do not have orthostatic headaches but whose symptoms are best explained by SIH. The type of underlying spinal CSF leak was classified according to a previously published classification system.[21]

MRV studies were performed prior to treatment of SIH. MRV was performed by using both phase-contrast (PC) and time-of-flight (TOF) techniques on 1.5T or 3T Siemens (Munich, Germany) scanners. 2D TOF parameters for 1.5T were as follows: time of repetition (TR) 26 seconds, time of echo (TE) 6.5 seconds, slice thickness 3 mm, resolution 256. On 3T, the measures were TR 21 seconds, TE 5 seconds, slice thickness 2.5 mm, resolution 256. 3D PC measures for 1.5T were as follows: TR 28 seconds, TE 11 seconds, velocity encoding 5 cm/s in one direction, resolution 256. On 3T, the measures were TR 69 seconds, TE 9 seconds, velocity encoding 5 cm/s in 3 directions, resolution 256. Source images and maximum intensity projection views were reviewed.

The MRV appearance of the transverse sinuses was scored for each patient according to the system reported by Higgins et al.[14] where normal is 0, the presence of one or more areas

We compared patients with SIH and rebound high-pressure headache to those without such a headache following treatment.

of focal narrowing is 1, and the presence of one or more signal gaps is 2 (figure). The transverse sinus scores were then summated in each patient to give values ranging from 0 (normal) to 4 (bilateral signal gaps). This scoring system was applied by 2 neuroradiologists, who were blinded to the occurrence of rebound high-pressure headaches.

Rebound high-pressure headache was diagnosed when the following criteria were met: (1) reverse orthostatic headache different from the original SIH headache; (2) resolution of headache following administration of oral acetazolamide; and (3) not better accounted for by another cause of headache.

We compared patients with SIH and rebound high-pressure headache to those without such a headache following treatment. The following variables were studied: sex, age at onset of symptoms, body mass index, duration of symptoms, type of spinal CSF leak, CSF opening pressure, presence of extradural CSF, need for surgical treatment, results of brain MRI, and MRV score.

Statistical analysis

Differences between SIH patients with rebound high-pressure headache and those without such headache were assessed with the χ^2 test for categorical variables and with the Student t test or Mann-Whitney U test as appropriate for continuous variables. All statistical tests were 2-tailed, and p values of less than 0.05 were considered to indicate statistical significance. Analyses were performed with SAS software, version 9.4 (SAS Institute, Cary, NC).

For comparisons between continuous variables, we applied Hedges g; for 2-category variables, we calculated relative risk; and for categorical variables with more than 2 categories, we calculated odds ratio. For all estimates of effect size, we provide 95% confidence intervals. Regarding the concern of multiple comparisons and appropriate adjustments, we include estimates of false discovery rates, as this was the guide utilized to plan the multiple comparisons.

Data availability

Anonymized data not published within this article will be made available by request from any qualified investigator.

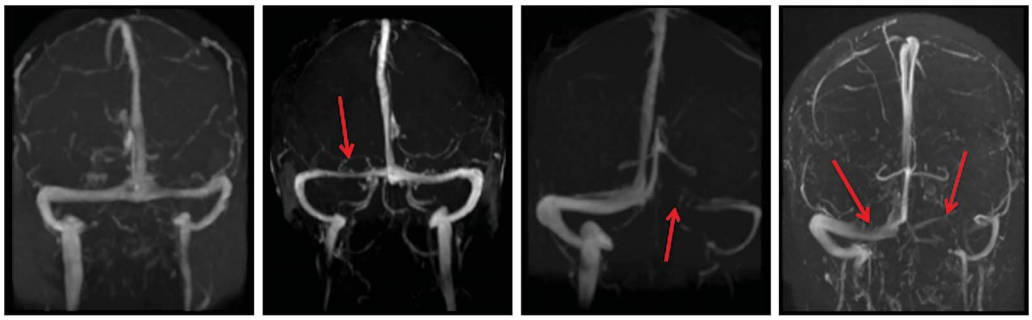
Results

A total of 113 patients (46 men [41%] and 67 women [59%]) with SIH were included in this study. The mean age at the onset of SIH was 45.9 years (range 13–71 years). Clinical and radiographic characteristics of the patient population are shown in table 1.

A total of 109 patients presented with orthostatic headaches, 3 with nonpositional headaches, and 1 with a reverse orthostatic headache.

Treatment of SIH was complicated by rebound high-pressure headaches in 31 patients (27.4%). Headache characteristics in these 31 patients are summarized in table 2. The most common scenario was that of a (sub)occipital SIH headache transforming to a frontal high-pressure headache. The rebound high-pressure headache developed within 72 hours of treatment in 23 patients, between 3 and 7 days in 7 patients, and between 1 week and 1 month in 2 patients. All 31 patients received oral acetazolamide, but eventually 9 did not tolerate acetazolamide and diuretics or topiramate were prescribed. Overall, rebound high-pressure headaches

Figure Magnetic resonance venography (MRV) scoring system (Higgins et al.[25]) for patients with rebound high-pressure headaches following treatment for SIH



Normal is 0; one or more areas of focal narrowing is 1; one or more signal gaps is 2. The scores are summated to give values ranging from 0 to 4.

Table 1 Clinical and radiographic characteristics of patients with spontaneous intracranial hypotension (SIH) and rebound high-pressure headache

		Rebound high-pressure headache				
	All patients	Yes	No	*p* Value	FDR	Effect size (95% CI)
No. of patients (%)	113	31 (27.4)	82 (72.6)			
Age at symptom onset, y				0.0135	0.0675	−0.5 (−0.9, −0.1)
Mean (SD)	45.9 (12.6)	41.6 (11.9)	47.6 (12.5)			
Median (IQR)	48 (39–53)	41 (37–48)	50 (39–54)			
Range, min–max	13–71	13–61	16–71			
Sex				0.0474	0.1185	1.3 (1.0–1.6)
Male	46 (40.7)	8 (25.8)	38 (46.3)			
Female	67 (59.3)	23 (74.2)	44 (53.7)			
Body mass index				0.2106	0.3343	−0.3 (−0.7, 0.1)
Mean (SD)	25.3 (4.0)	24.5 (4.2)	25.6 (3.9)			
Median (IQR)	25 [22–28]	24 [21–27]	25 [23–28]			
Range, min–max	17–39	18–33	17–39			
Opening pressure, cm H_2O[a]				0.0715	0.143	−0.3 (−0.7, −0.1)
Mean (SD)	9.6 (7.4)	7.9 (7.9)	10.3 (7.1)			
Median (IQR)	9 [3–15]	7 [0–11]	10 [5–16]			
Range, min–max	0–21	0–21	0–26			
Symptom duration, mo[b]				0.5377	0.5377	0.1 (−0.3, 0.5)
Mean (SD)	41.4 (60.9)	45.5 (78.4)	39.9 (53.3)			
Median (IQR)	19 [4–58]	16 [3–49]	22 [5–60]			
Range, min–max	0–344	1–344	0–334			
CSF leak type (ref 21)				0.4607	0.5119	
1A	29 (25.7)	11 (35.5)	18 (22.0)			1.9 (0.6–6.3)
1B	4 (3.5)	0 (0)	4 (4.9)			<0.001
2A	48 (42.5)	14 (45.2)	34 (41.5)			1.3 (0.4–3.9)
2B	3 (2.7)	0 (0)	3 (3.7)			<0.001
3	4 (3.5)	0 (0)	4 (4.9)			<0.001
4	25 (22.1)	6 (19.4)	19 (23.2)			1.0 (reference)
Extradural CSF				0.0286	0.0953	1.3 (1.0–1.6)
Positive	54 (47.8)	20 (64.5)	34 (41.5)			
Negative	59 (52.2)	11 (35.5)	48 (58.5)			
Surgery, n (%)				0.4116	0.5119	1.1 (0.9–1.4)
Yes	85 (75.2)	25 (80.7)	60 (73.2)			
No	28 (24.8)	6 (19.3)	22 (26.8)			
Brain MRI, n (%)				0.2340	0.3343	1.2 (0.9–1.5)
Stigmata of SIH	86 (76.1)	26 (83.9)	60 (73.2)			
Normal	27 (23.9)	5 (16.1)	22 (26.8)			

Continued

Table 1 Clinical and radiographic characteristics of patients with spontaneous intracranial hypotension (SIH) and rebound high-pressure headache *(continued)*

		Rebound high-pressure headache				
	All patients	Yes	No	*p* Value	FDR	Effect size (95% CI)
MR venography score[c]				0.0092	0.0210	
0	42 (37.2)	6 (19.4)	36 (43.9)			1.0 (reference)
1	34 (30.1)	8 (25.8)	26 (31.7)			1.8 (0.6–6.0)
2	34 (30.1)	15 (48.4)	19 (23.2)			4.7 (1.6–14.2)
3	3 (2.7)	2 (6.4)	1 (1.2)			12.0 (1.0–153.8)
MR venography score[c]				0.0021	0.0210	0.3 (0.1–0.6)
0/1	76 (67.3)	14 (45.2)	62 (75.6)			
2/3	37 (32.7)	17 (54.8)	20 (24.4)			

Abbreviations: CI = confidence interval; FDR = false discovery rate; IQR = interquartile range; MR = magnetic resonance.
Effect size calculated for continuous (Hedges g) and categorical data (relative risk, odds ratio–CSF leak type).
[a] Opening pressure (data available for 92 patients), cm H_2O.
[b] Time between date of symptoms and date of treatment.
[c] Higgins et al. scoring system.[14]

resolved with medical treatment within 6 weeks in 25 patients and within 3 months in a further 4 patients. One patient elected to undergo ventriculo-peritoneal shunt placement 14 months following SIH treatment and one patient was not able to wean off acetazolamide at last follow-up 15 months following SIH treatment.

Funduscopic examination during the period of rebound high-pressure headaches was performed in 25 patients and showed papilledema in 2 patients.

Postprocedure brain imaging was performed in all 31 patients who developed rebound high-pressure headaches and showed complete resolution of the stigmata of SIH in all 27 patients who had those findings prior to treatment. Cerebral venous sinus thrombosis was not detected in any patient. Postprocedure brain imaging was performed within 48 hours of treatment in 21 patients and within 1 month in all 31 patients.

Postprocedure spine imaging was performed in all 20 patients with an extradural CSF collection who developed rebound high-pressure headaches and showed complete resolution of the extradural CSF collection in all 20 patients. Postprocedure spine imaging was performed within 48 hours of treatment in 17 patients and within 1 month in all 20 patients.

Rebound high-pressure headache was diagnosed in 14% of patients with an MRV score of 0, in 24% of patients with a score of 1, in 44% of patients with a score of 2, and in 67% of patients with a score of 3 ($p = 0.0092$). There were no patients with a score of 4.

Compared to SIH patients without rebound high-pressure headache (n = 82), those with rebound high-pressure headache were younger ($p = 0.0135$), more often female ($p = 0.0474$), and more often had an extradural CSF collection on spinal imaging ($p = 0.0286$) (table 1).

Following the onset of rebound high-pressure headache, a lumbar puncture was eventually performed in 4 patients and the opening pressures were elevated in 1 patient (30 cm H_2O compared to 16 cm H_2O pretreatment), borderline elevated in 2 patients (22 cm H_2O compared to less than 10 cm H_2O and 21 cm H_2O compared to 12 cm H_2O), and normal in 1 patient (17 cm H_2O compared to 8 cm H_2O). Following lumbar puncture, low CSF pressure headache requiring epidural blood patching occurred in 3 of these 4

Table 2 Headache location pretreatment and posttreatment in patients with rebound high-pressure headaches following treatment for spontaneous intracranial hypotension

Pretreatment	Posttreatment	No. of patients
(Sub)occipital	Frontal	12
Generalized	Frontal	5
(Sub)occipital	Generalized	3
Frontal–(temporal)	Frontal	3
Vertex	Frontal	2
Temporal–parietal	Generalized	2
Generalized	Generalized	2
Temporal–parietal	Frontal	1
(Sub)occipital	(sub)occipital	1

In this study, we found that about one-fourth of patients undergoing treatment for SIH developed rebound high-pressure headaches.

patients. The lumbar punctures in these 3 patients were performed with a 22-G or 24-G Gertie-Marx spinal needle (International Medical Development, Park City, UT) in 2 patients and a 22-G BD Quincke type spinal needle (Becton, Dickinson and Company, Franklin Lakes, NJ) in one patient.

TAKE-HOME POINTS

- Rebound high-pressure headaches occur in about one-fourth of patients following treatment for spontaneous intracranial hypotension.
- Rebound high-pressure headache is characterized by reverse orthostatic headache.
- Measurement of CSF pressure is not necessary to make the diagnosis of rebound high-pressure headache.
- Transverse sinus stenosis is a risk factor for the development of rebound high-pressure headache.

Discussion

In this study, we found that about one-fourth of patients undergoing treatment for SIH developed rebound high-pressure headaches. This is similar to a recent study of pediatric SIH, where we noted rebound high-pressure headaches in about one-fifth of patients.[10] This indicates that rebound high-pressure headaches are a more frequently encountered occurrence following SIH treatment than previously appreciated.

The main finding of our study was the relationship between transverse sinus stenosis, as demonstrated on MRV, and the frequency of rebound high-pressure headaches. Rebound high-pressure headache following treatment for SIH occurred in only 14% of patients with normal MRV signal, in about one-fourth of those with focal narrowing in one transverse sinus, and in half of those with complete signal gap in one transverse sinus or any involvement of both transverse sinuses. This suggests that restriction of cerebral venous outflow plays an important role in the pathophysiology of rebound high-pressure headaches in patients following treatment of SIH. The areas of venous sinus stenosis we observed, however, are normal anatomic variants.

Similar to IIH,[18,19] our study found that rebound high-pressure headaches were more common in women and at a younger age. The presence of an extensive extradural CSF collection was also associated with an increased risk of rebound high-pressure headaches, possibly reflecting the severity of the underlying spinal CSF leak and the potential for significant shifts in CSF dynamics following treatment. Indeed, posttreatment imaging revealed complete resolution of the extradural CSF collection in all patients with rebound high-pressure headaches, often within 48 hours of treatment.

Rebound intracranial hypertension following treatment of SIH was first reported in 1996 in a patient who underwent surgical repair of a leaking thoracic meningeal diverticulum.[4] The symptoms of SIH had been present for 9 years and postoperative visual blurring was found to be due to bilateral papilledema and a retinal hemorrhage. Since then, rebound intracranial hypertension has been described in more detail and it has been diagnosed more frequently, also following percutaneous procedures for SIH.[5-11] Yet only case reports and small cases series have been reported. Nowadays, unrecognized rebound intracranial hypertension resulting in papilledema is noted rarely, while CSF pressure is measured infrequently or is only borderline elevated, and the diagnosis of rebound intracranial hypertension has mostly been clinical. We have been reluctant to measure CSF pressure under these circumstances because of the increased likelihood of a postdural puncture headache when CSF pressure has been restored. Indeed, that was the case for most of our patients who underwent a lumbar puncture, including those in whom small-gauge atraumatic spinal needles were used. Also, asymptomatic post–epidural blood patch rebound intracranial hypertension has been reported.[9] Currently, we manage patients based on their symptomatology and only resort to lumbar puncture if symptoms of elevated CSF pressure become intolerable or if there is sufficient doubt regarding the diagnosis of rebound high-pressure headache. Although lumbar puncture showed high CSF pressure in only 1 of the 4 patients with rebound high-pressure headache, a significant increase in CSF pressure compared to pretreatment was noted in all 4 patients. This suggests that the change in CSF pressure may be an important determinant in the development of rebound high-pressure headache. The observation that posttreatment brain MRI showed resolution of the stigmata of SIH in all patients with rebound high-pressure headaches, and often within 48 hours, is consistent with our clinical diagnosis of rebound high-pressure headache.

The usual treatment of rebound high-pressure headaches is acetazolamide. Acetazolamide is a carbonic anhydrase inhibitor that decreases production of CSF. It remains the first-line treatment for patients with IIH.[22-24] Although rebound high-pressure headache following treatment of SIH generally is not a serious

complication, e.g., it is not due to cerebral venous sinus thrombosis and papilledema is rare, the headache is bothersome and often interrupts sleep. Therefore, we recommend treatment with acetazolamide if simple means such as elevating the head of the bed or sleeping on an extra pillow are insufficient. We usually start acetazolamide at a dose of 500 mg PO BID or TID, but some of our patients required doses of up to 3,000 mg QD to control symptoms. Rebound high-pressure headaches resolved within 6 weeks in most patients and treatment lasting more than 1 year was rare. Of note, Ferrante et al.[8] premedicated with acetazolamide (500 mg) 42 patients undergoing epidural blood patching for SIH and reported not a single instance of rebound high-pressure headache. However, in an unpublished series of 23 patients with SIH premedicated with acetazolamide (750 mg) who we treated with epidural blood patching, rebound high-pressure headaches developed in 4 patients.

The main side effects of acetazolamide are paresthesias of the fingers, toes, or face, dysgeusia, nausea and vomiting, diarrhea, and fatigue.[23] If acetazolamide is not tolerated or is ineffective, other pharmaceutical treatments may be of benefit, including topiramate (a mild carbonic anhydrase inhibitor) and diuretics, such as furosemide, hydrochlorothiazide, or lisinopril. Ventriculo-peritoneal shunt placement is an effective alternative that we have resorted to only rarely. The findings in our study of lateral sinus stenosis suggest that endovascular treatment could also be considered in patients with recalcitrant rebound high-pressure headaches, at least when conventional catheter-based angiography confirms a significant pressure gradient at the site of stenosis.[25-29]

The diagnosis of rebound high-pressure headache following treatment of SIH is clinical and is mainly based on the reversal of the typical orthostatic headache pattern. This is unlike IIH, where the presence of a recumbent headache is not prototypical. This may be due to the fact that rebound high-pressure headache following treatment of SIH develops over a short time period while the headache of IIH is associated with a more chronic time course.

Finally, it should be noted that the occurrence of rebound high-pressure headaches following SIH treatment is generally considered a good prognostic sign as it would indicate that the CSF leak has been successfully treated. However, high-pressure type headaches will most likely also develop in patients who have headaches from almost any other etiology who undergo epidural blood patching.

Limitations and generalizability

Several limitations of this study should be noted. First, this is a highly selected group of patients referred to a quaternary referral center for SIH and the generalizability of our findings is unknown. For example, it is possible that rebound high-pressure headache following treatment of SIH is less likely to occur in patients who are cured with a single epidural blood patch who do not need referral to a quaternary referral center. Second, only a small minority of the presently reported patients underwent formal measurement of CSF pressure while experiencing rebound high-pressure headache. However, the diagnosis is clinical and we have been reluctant to perform lumbar puncture in these patients because of the risk of postdural puncture headache. Perhaps further developments in noninvasive intracranial pressure monitoring will help in establishing the diagnosis. Third, a spinal CSF leak, i.e., the presence of extradural CSF, could not be detected in all of the presently reported patients and an alternate diagnosis cannot be excluded. However, all patients met the well-established ICHD-III criteria for SIH.

Author contributions

W.I. Schievink: drafting/revising the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data; acquisition of data; study supervision or coordination. M.M. Maya: drafting/revising the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data; acquisition of data; study supervision or coordination. S. Jean-Pierre: drafting/revising the manuscript for content, including medical writing for content; acquisition of data; study coordination. Franklin G. Moser, M.M. Maya: drafting/revising the manuscript for content, including medical writing for content; analysis or interpretation of data; acquisition of data; study supervision or coordination. Miriam Nuño: drafting/revising the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data; acquisition of data; statistical analysis; study supervision or coordination. B.D. Pressman: drafting/revising the manuscript for content, including medical writing for content; study concept or design; analysis or interpretation of data; acquisition of data; study supervision or coordination.

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