

Neuroimaging Findings in Hemifacial Spasm: A Single-Center Experience

● Tuğba Eyigürbüz¹, ● Zerrin Yıldırım¹, ● Elif Korkut¹, ● Ece Akalın Akkaş¹, ● Mehmet Turgut Adatepe²,
● Nilüfer Kale¹

¹University of Health Sciences Turkey, İstanbul Bağcılar Training and Research Hospital, Clinic of Neurology, İstanbul, Turkey

²University of Health Sciences Turkey, İstanbul Bağcılar Training and Research Hospital, Electrophysiology Laboratory, İstanbul, Turkey

ABSTRACT

Introduction: In this study, we documented the demographic, etiological, clinical and radiological features of our patients with primary hemifacial spasm (HFS). We also wanted to emphasize that there may be an association between idiopathic intracranial hypertension (IIH) and HFS.

Methods: Fifty-five patients diagnosed with HFS (28 women) who were followed up in the Movement Disorders Outpatient Clinics of the Department of Neurology University of Health Sciences Turkey, İstanbul Bağcılar Training and Research Hospital between January 2017 and January 2022 were included in this study. Demographic, clinical, and radiological findings were retrospectively reviewed. Depending on radiological findings, patients were divided into three groups: a) Normal findings, b) Incidental findings that did not appear to be related to clinical findings, and c) vascular abnormalities at the level of the brainstem.

Results: Only 23 patients had no atherosclerotic risk factors. While magnetic resonance imaging of the brain was normal in 23 patients, 19 patients had ischemic white matter changes, 5 patients had partial empty sella, 7 patients had dolichoectatic basilar artery, and 1 patient had a compression of the anterior segment of the left superior cerebellar artery to the 7th cranial nerve. Based on the history and clinical findings, lumbar puncture was performed in 4 patients, and 3 of them were diagnosed with idiopathic IIH with HFS, and they were treated with acetazolamide. Fifty-one patients were treated with botulinum toxin injections only.

Conclusion: Vascular compression is often noted on imaging of patients with primary HFS, but as in our case series, an empty sella finding in patients with chronic headache may be a sign of IIH and should not be overlooked. Also, HFS may be an uncommon presentation of IIH, and symptoms of HFS may improve with treatment of IIH. Additionally, the presence and history of Coronavirus disease-2019 infection should be questioned in newly admitted cases.

Keywords: Hemifacial spasm, brain magnetic resonance imaging, empty sella, intracranial hypertension

Introduction

Hemifacial spasm (HFS) is a hyperkinetic movement disorder characterized by involuntary, arrhythmic, painless, clonic, or tonic intermittent spasms on one side of the face that negatively affects the patient's daily life (1). There are two forms of HFS, primary and secondary. Primary HFS is more common in women than in men (1.5:1 ratio), and the mean age at onset is about 45-52 years, although the range is wide. 14.5 in 100,000 women and 7.4 in 100,000 men have HFS. Most cases occur sporadically, but there are some familial cases (2,3). Secondary HFS often occurs after peripheral facial paralysis, and less commonly because of facial nerve or brainstem damage after tumors, demyelinating diseases, trauma, and infections (3).

There are two theories regarding this pathogenesis. The first theory is that a phase or false synapse forms in the area of demyelination caused by compression. Other theory states that the abnormal signals originate

from the nucleus of the facial nerve, which has rearranged itself due to the disorganized afferent information (2). Primary HFS is thought to be caused by vascular compression of the facial nerve at the level of the nucleus and in the regions where it exits the nucleus (4). Magnetic resonance imaging (MRI) is an important tool to exclude secondary etiologies and to demonstrate vascular compression in HFS (5).

Information on HFS comes from clinical, neurosurgical observations, and electrophysiological studies, although the exact cause is still unknown (6). The generally accepted view of the development of HFS is that compression of a vascular structure adjacent to the entry point of the facial nerve root and the focal demyelination resulting from this compression causes spasms the ephaptic transition. Such structures, thought to cause compression, can sometimes be visualized radiologically, but they are also sometimes not visible during surgical procedures or even autopsy (7-9).



Address for Correspondence: Zerrin Yıldırım MD, University of Health Sciences Turkey, İstanbul Bağcılar Training and Research Hospital, Clinic of Neurology, İstanbul, Turkey

Phone: +90 212 414 40 00 **E-mail:** yildirimzerrin@gmail.com **ORCID ID:** orcid.org/0000-0002-5128-1784

Cite this article as: Eyigürbüz T, Yıldırım Z, Korkut E, Akalın Akkaş E, Adatepe MT, Kale N. Neuroimaging Findings in Hemifacial Spasm: A Single-Center Experience. *Istanbul Med J* 2022; 23(3): 229-35.

Received: 20.06.2022

Accepted: 07.08.2022

Although in many patients brain imaging is unremarkable, some patients have non-specific findings, and in some patients, lesions that appear to be the cause of HFS can be detected by imaging. These lesions are vascular abnormalities in the vicinity of the facial nerve, as well as less common tumors and structural changes in this area. In some cases, mostly in middle and old age, incidental lesions far from the facial nerve region may be detected, such as cortical infarcts (7,9-14). Surgical microvascular decompression and botulinum toxin injections as symptomatic treatment are the mainstay of treatment for primary HFS (4).

This cross-sectional study documented the demographic, clinical, and radiological findings of patients with HFS. Additionally, this article discusses the very rare HFSs associated with intracranial hypertension (IIH).

Methods

The records of patients who were followed up in the Movement Disorders Outpatient Clinics of the Department of Neurology of University of Health Sciences Turkey, İstanbul Bağırcilar Training and Research Hospital between January 2017 and January 2022 were retrospectively reviewed. Fifty-five patients diagnosed with HFS were included in this study.

Demographic data such as age, sex, and comorbidities, including atherosclerotic risk factors (RF), as well as clinical data such as medications for HFS, MRI of the brain, and MRI angiography (MRA) of the brain or computed tomography angiography (CTA), and cerebrospinal fluid (CSF) results, when appropriate, were collected and recorded.

Hypertension, diabetes mellitus (DM), atrial fibrillation, coronary artery disease, heart failure, previous stroke, smoking, obesity, and dyslipidemia were the atherosclerotic RFs.

We classified RFs into 1 RF, 2 RFs, and >2 RFs. Because DM may result in cranial nerve involvement, the presence of DM was reported separately. Depending on radiological findings, patients were divided into three groups: a) Normal findings, b) Incidental findings that did not appear to be related to clinical findings, and c) Vascular abnormalities at the level of the brainstem.

According to a protocol approved by the Local Ethics Committee of University of Health Sciences Turkey, İstanbul Training and Research Hospital (approval number: 199, date: 17.06.2022), all participants gave written informed consent according to the Declaration of Helsinki.

Statistical Analysis

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 25.0 software (Armonk, NY: IBM Corp.). Normality of variables was analyzed using the Shapiro-Wilk test. The homogeneity of group variances was tested with Levene's test. The Mann-Whitney U test was used to compare continuous data that did not have a normal distribution. Pearson chi-square tests and Fisher's Exact tests were used to compare categorical data. Analysis results were expressed as the number of observations (n), percentage, minimum, maximum, mean, and standard deviation. A p-value < 0.05 was considered statistically significant.

Results

Fifty-five patients with HFS were included in this study. 50.9% were female (n=28). The mean age was 58.16±9.7 years. 65.5% had left-sided HFS (n=36) and the remaining 34.5% (n=19) had right-sided HFS. The mean age was 57.5±9.3 and 59.5±10.6 years in the left HFS and right HFS groups, respectively. While men predominated in the left HFS group (n=19, 52.8%), women predominated in the right HFS group (n=11, 57.9%). There were no significant differences between the left and right HFS groups to age, sex, RFs, concomitant DM, history of chronic headache, disease duration, medications for HFS, and neuroimaging findings. Table 1 shows the findings of our patients.

One patient with left-sided HFS had otosclerosis and one patient with right-sided HFS had systemic amyloidosis. When asked about concomitant symptoms, one patient with right-sided HFS was found to have tinnitus and one patient with left-sided HFS was found to have cervical dystonia concomitant with HFS, whereas the other patients had isolated HFS symptoms.

We had a patient with HFS on the left side whose symptoms occurred after severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) infection. This patient, who had SARS-CoV-2 infection about two weeks before the onset of symptoms compatible with HFS and had not been vaccinated, also had pulmonary involvement but did not require hospitalization.

Brain MRI was normal in 41.8% patients (n=23). 34.5% of patients (n=19) had non-specific ischemic white matter changes, 9.1% of patients (n=5) had partial empty sella, and 12.7% of patients (n=7) had a dolichoectatic basilar artery. A patient had a compression of the anterior segment of the left superior cerebellar artery on the 7th cranial nerve, accompanied by partial empty sella. MRA and CTA were used only to confirm vascular abnormalities when necessary. MRA and CTA were used only to confirm vascular abnormalities when necessary. The results of these examinations were consistent with MRI findings of the brain. There was no significant difference between the left and right HFS groups in neuroimaging findings (Table 1).

Radiologically, the study population was divided into three groups: Normal, incidental findings, and vascular abnormalities (one patient with compression of the anterior segment of the left superior cerebellar artery on the 7th cranial nerve associated with partial empty sella was included in this group). The mean ages of the three groups were 54.7 10.3, 62.3 7.1, and 55.6 11.2 years, respectively (p=0.014). Pairwise comparisons showed that the mean age of patients with incidental findings was significantly higher than that of patients with normal MRI findings (p=0.012). While male predominance was observed in patients with normal MRI findings and patients with vascular findings (n=13, 56.5% and n=6, 75%, respectively), females predominated in patients with incidental findings (n=16, 66.7%). There were no significant differences in the other pairwise comparisons (Table 2).

RF were significantly different among the three groups. Patients without RFs were predominant in the group with normal MRI findings and group with vascular findings, while patients with 1 RF and 2 RFs were predominant in the group with incidental findings (Table 2).

Table 1. Demographics and clinical data of the left and right HFS groups

(n=55)	Left HFS (n=36)	Right HFS (n=19)	p
Age mean \pm SD	57.5 \pm 9.3	59.5 \pm 10.6	0.32*
Sex, female:male, n (%)	17:19 (47.2%:52.8%)	11:8 (57.9%:42.1%)	0.32**
Risk factors, n (%)			
None	17 (47.2%)	6 (31.6%)	0.29***
1 RF	11 (30.6%)	4 (21.1%)	-
2 RFs	6 (16.7%)	7 (36.8%)	-
>2 RFs	2 (5.6%)	2 (10.5%)	-
Diabetes mellitus, n (%)	5 (13.9%)	2 (10.5%)	1.0**
Chronic headache, n (%)	7 (19.4%)	2 (10.5%)	0.47**
Disease duration, mean \pm SD (months)	53.8 \pm 59.3	75.4 \pm 59.9	0.29*
Medication against HFS, n (%)			
None	28 (77.8%)	18 (94.7%)	0.14**
Carbamazepine/oxcarbazepine	8 (22.2%)	1 (5.3%)	-
Neuroimaging, n (%)			
Normal	17 (47.2%)	6 (31.6%)	0.33***
Non-specific ischemic white matter changes	9 (25%)	10 (52.6%)	-
Dolichoectatic basilar artery	5 (13.9%)	2 (10.5%)	-
Partial empty sella	4 (11.1%)	1 (5.3%)	-
Vascular abnormalities and empty products	1 (2.8%)	0	-
Treatment, n (%)			
Botulinum toxin	32 (88.9%)	19 (100%)	0.52***
Acetazolamide + botulinum toxin	2 (2.8%)	0	-
Acetazolamide	1 (5.6%)	0	-
No treatment	1 (2.8%)	0	-

n: Number, SD: Standard deviation, HFS: Hemifacial spasm, RF: Risk factor, *Mann-Whitney U test, **Fisher's exact test, ***Pearson chi-square test

Table 2. Demographics and clinical data of the groups according to neuroimaging findings

(n=55)	Normal (n=23)	Incidental (n=24)	Vascular (n=8)	p
Age mean \pm SD	54.7 \pm 10.3 ^a	62.3 \pm 7.1 ^b	55.6 \pm 11.2 ^{ab}	0.014*
Sex, female:male, n (%)	10:13 (43.5%:56.5%)	16:8 (66.7%:33.3%)	2:6 (25%:75%)	0.08**
Side, L:R, n (%)	17:6 (73.9%:26.1%)	13:11 (54.2%:45.8%)	6:2 (75%:25%)	0.3**
Risk factors, n (%)				
None	16 (69.6%)	3 (12.5%)	4 (50%)	0.01**
1 RF	3 (13%)	10 (41.7%)	2 (25%)	-
2 RFs	3 (13%)	9 (37.5%)	1 (12.5%)	-
>2 RFs	1 (4.3%)	2 (8.3%)	1 (12.5%)	-
Diabetes mellitus, n (%)	1 (4.3%)	5 (20.8%)	1 (12.5%)	0.24**
Chronic headache, n (%)	2 (8.7%)	4 (16.7%)	3 (37.5%)	0.17**
Disease duration, mean \pm SD (months)	62.4 \pm 53.2	62.9 \pm 69.6	23 \pm 14.7	0.47*
Medication against HFS, n (%)				
None	18 (78.3%)	21 (87.5%)	7 (87.5%)	0.66**
Carbamazepine/oxcarbazepine	5 (21.7%)	3 (12.5%)	1 (12.5%)	-
Treatment, n (%)				
Botulinum toxin	23 (100%)	21 (87.5%)	7 (87.5%)	0.5**
Acetazolamide + botulinum toxin	0	1 (4.2%)	1 (12.5%)	-
Acetazolamide	0	1 (4.2%)	0	-
No treatment	0	1 (4.2%)	0	-

n: Number, SD: Standard deviation, F: Female, M: Male, HFS: Hemifacial spasm, RF: Risk factor, *Kruskal-Wallis test, ^{ab}There is no difference between groups with the same letter, **Pearson chi-square test

There were no significant differences among these three groups on the side of HFS, presence of DM, presence of chronic headache, duration of illness, medication for HFS, and treatment (Table 2).

Electromyography was performed in 5.45% patients (n=3), and short-term, frequently recurrent spasm activity was observed when recorded with simultaneous superficial disc electrodes from the orbicularis oculi and orbicularis oris muscles.

A lumbar puncture was performed in 11.1% of patients with left-sided HFS (n=4) because of a history of chronic headache with partial empty sella. CSF opening pressure, cell count, biochemistry (including sodium, potassium, chloride, glucose, and protein levels), IgG and oligoclonal banding pattern, bacterial culture, and viral polymerase chain reaction panel were evaluated. One patient had normal CSF findings. Three patients had increased opening pressure, whereas the other CSF findings were completely normal, and they were diagnosed with idiopathic IHH.

IHH patients (n=3) were treated with acetazolamide. While one of them improved only with acetazolamide treatment, two of them also required botulinum toxin injection. 92.72% patients (n=51) were treated with botulinum toxin injections only. One patient with left-sided HFS refused botulinum toxin injection and was followed up without treatment. Various MRI examples of some patients are shown in Figures 1-5.

Discussion

The mean age and left-sided predominance of our patients with primary HFS was consistent with the literature (1,2). Although females predominate in the literature in patients with primary HFS, there was no significant difference in the female-to-male ratio in our patient series.

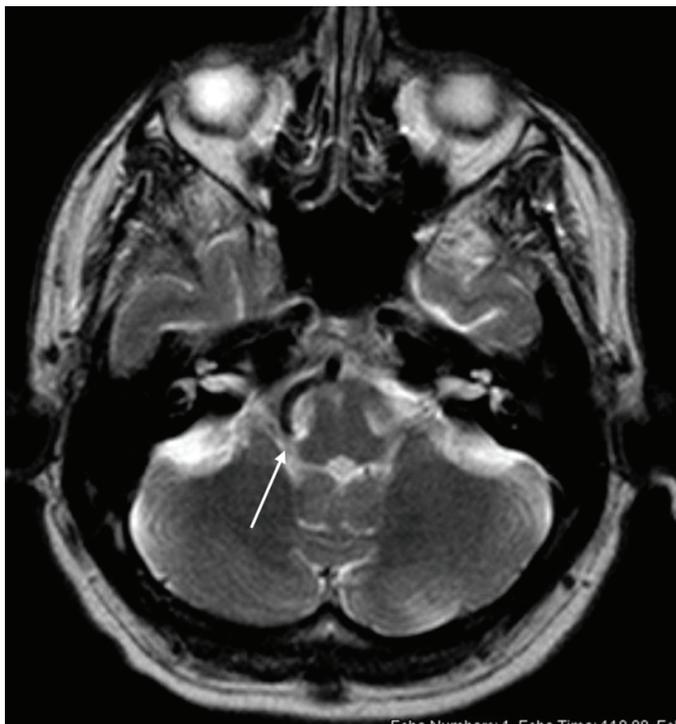


Figure 1. Dolichoectasia at the right vertebral artery and the proximal part of the basilar artery on axial T2 weighted image and compression to the lower cranial nerves on the right



Figure 2. Dolichoectasia at the left vertebral artery on axial T2 weighted image and compression on left 7th and 8th cranial nerves

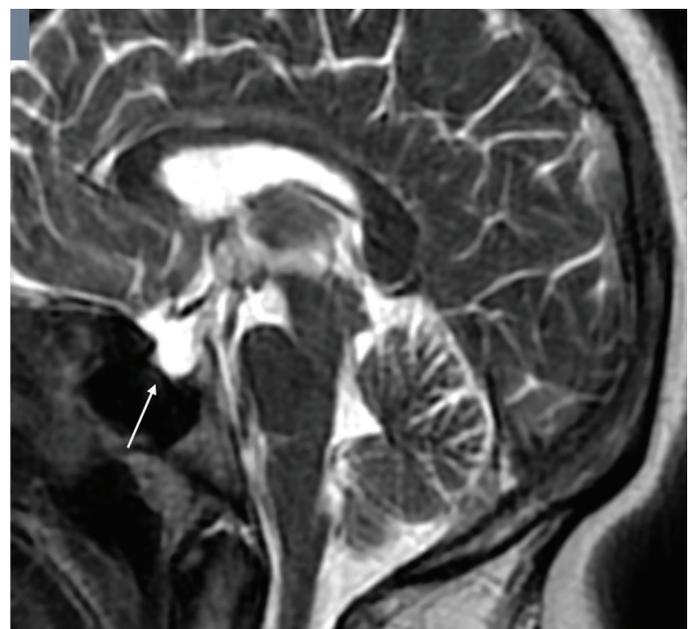


Figure 3. Empty sella on sagittal T2 weighted image

HFS is a clinical picture of unknown causes but is thought to be triggered by arteriosclerosis by many authors. This relationship was first described by Schultze in 1875 during the autopsy of a patient with an aneurysm compressing the facial nerve (15). In the 1960s, Gardner and Sava drew attention to this relationship and pointed out that the symptoms would be relieved by surgically relocating the vessel (9,14). In our series, 7 of

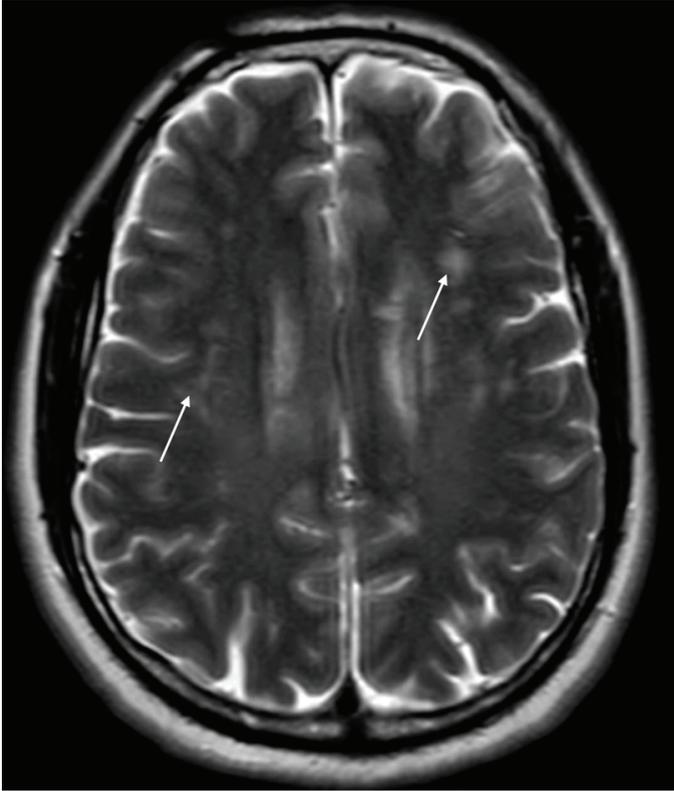


Figure 4. Bilateral hyperintense ischemic-gliotic lesions on axial T2 weighted image



Figure 5. Partial empty sella on sagittal T2 weighted image

55 patients (12.7%) had vascular abnormalities at the brainstem level, revealed radiologically. In the literature, space-occupying lesions in the cerebellopontine corner or congenital causes of bone structure are considered more rarely (9,14). In our series, there were no such lesions. Non-specific ischemic white matter changes were considered

incidental in 19 (34%) cases and partial empty sella in 5 cases (9.1%). Additionally, a case of empty sella was accompanied by brain stem vascular abnormality. In our cases, vascular abnormalities included the dolichoectatic basilar artery. The radiological findings in our study agree with the literature (16).

Neuroradiological examinations with various MRI techniques may show a compression of vascular structures passing through the facial nerve root entry zone (2).

HFS is a movement disorder thought to be caused mainly by compression of the facial nerve by vascular structures in the root exit region (17). Mostly, the posterior inferior cerebellar artery, anterior inferior cerebellar artery, and vertebral arteries are held responsible. Space-occupying lesions such as epidermoid tumors, neuroma, meningioma, astrocytoma, and parotid gland tumors are observed in approximately 5% of HFS patients (2).

Vascular abnormalities at the level of the brain stem are defined by many researchers as the triggering factor for developing HFS (6,8-10,14,18-20). It has been reported that the vessel causing compression may be as large as those found in the vertebral artery or as thin as in the posterior inferior cerebellar artery, anterior inferior cerebellar artery or cochlear artery (9,14). It is stated that in classical HFS, the compressing artery almost always crosses the nerve from front to back at the level of the exit point or the intrapontine section of the facial nerve, and the compressing veins cause a similar picture in many patients with a close relationship (8,10,20). Some cases had more than one vascular abnormality. MRI showed neurovascular compression of the seventh cranial nerve in 43% of patients (21).

The rates of etiological causes vary in the literature, in line with the examination methods or whether the series is clinical or surgical. In a literature review that included 1688 cases, 509 had vascular abnormalities, 19 tumors, 7 bone abnormalities, and 986 cases had an undetermined cause, and in 163 cases, radiologic, surgical, or autopsy studies failed to demonstrate a cause (22). Barker et al. (23) reported that all 703 cases with HFS in which they applied microvascular decompression were due to vascular abnormality and that microvascular decompression is a safe and definite treatment for HFS with proven long-term efficacy.

Some case-control studies found a significantly higher prevalence of hypertension among patients with primary HFS than among patients with other neurological diseases or healthy controls (21,24), other series failed to find a significant difference in the prevalence of arterial hypertension in patients with primary HFS (24-26). In our study 58.2% of the patients (n=32) had at least one RF and 30.9% had hypertension (n=17).

Symptoms of HFSs can be relieved by medication, injections, or surgery. Plenty of rest and stress reduction are also recommended. But there is no cure for HFS. Anticonvulsants, baclofen, anticholinergics, and clonazepam have been used in treatment for many years. However, it is a critical development that patients with botulinum toxin treatment show almost complete recovery, albeit temporary (2).

Partial empty sella may be an incidental radiological finding in an asymptomatic patient with preserved pituitary function, or it may be a sign of IIH with increased CSF pressure (27). In our study, an empty sella image was detected in 5 cases. Three of these patients had high opening pressure at the lumbar puncture, while other CSF findings were completely normal, and these three patients were also diagnosed with IIH. One recovered with acetazolamide treatment alone, while the other two required botulinum toxin injections. We can interpret the empty sella as an incidental finding in this series because empty sella is a very common anatomical variation. But since empty sella may be a sign of IIH, we can also interpret that one patient in our series had HFS due to elevation of intracranial pressure and improved well with acetazolamide treatment.

MRI features of IIH in patients with unilateral facial spasm association have already been described in the literature and recently called "IIH-spasm syndrome" (28-30).

The etiology of IIH is unknown. It can cause headaches, pulsatile tinnitus, double vision, papilledema, and sixth cranial nerve palsy. In addition to these typical findings, atypical presentations, including HFS, have been reported. Chen et al. (31) reported a 43-year-old female patient who presented with a 2-year history of left-sided HFS. MRI demonstrated bilateral anterior inferior cerebellar artery vascular loops involving the internal auditory canals as well as IIH-associated findings. After the lumbar puncture, which revealed an elevated CSF opening pressure, the patient was put on acetazolamide treatment, resulting in complete resolution of the HFS (31). In our study, we had four patients with a history of chronic headaches accompanied by partial empty sella. Three of them had elevated CSF opening pressure. One of these patients improved after 5 days with only acetazolamide treatment. An empty sella sign is usually seen in middle-aged, obese women with hypertension. Additionally, these patients may also have headaches, endocrine disorders, and visual disturbances if the sella is enlarged (32). If HFS is accompanied by headache and an empty sella finding, IIH should be suspected. Another case that should be considered was HFS with pulmonary involvement after a SARS-CoV-2 infection two weeks ago. There are several neurological symptoms after the global outbreak of Coronavirus disease-2019 (COVID). The central nervous system, meninges, cranial nerve, spinal cord, and peripheral nerve involvement have been reported. HFS cases that developed after the COVID pandemic began to be published as case reports (33,34). Where these patients will evolve in the long-term follow-up is one of the medical concerns, and in this respect, it would be appropriate to deepen the anamnesis in newly admitted HFS cases.

Conclusion

Botulinum toxin injection is the most common symptomatic treatment of HFS in the clinical practice of neurology. Vascular compression is frequently noted in the imaging of patients with primary HFS, however, as in our case series, partial empty sella finding, which may be a sign of IIH, should not be overlooked.

Although rare, IIH may occur with HFS findings. Here, HFS symptoms may also improve when IIH is treated.

Ethics Committee Approval: According to a protocol approved by the Local Ethics Committee of University of Health Sciences Turkey, Istanbul Training and Research Hospital (approval number: 199, date: 17.06.2022), all participants gave written informed consent according to the Declaration of Helsinki.

Informed Consent: Informed consent was obtained.

Peer-review: Externally peer-reviewed.

Authorship Contributions: Concept - T.E., Z.Y., M.T.A., N.K.; Design - T.E., Z.Y., M.T.A., N.K.; Data Collection or Processing - T.E., Z.Y., E.K., E.A.A., M.T.A., N.K.; Analysis or Interpretation - T.E., Z.Y., E.K., E.A.A.; Literature Search - T.E., Z.Y., N.K.; Writing - T.E., Z.Y., M.T.A., N.K.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

References

- Chaudhry N, Srivastava A, Joshi L. Hemifacial spasm: The past, present and future. *J Neurol Sci* 2015; 356: 27-31.
- Jankovic J. *Parkinson Disease and Other Movement Disorders in Bradley and Daroff's Neurology in Clinical Practice*. 7th ed. London: Elsevier; 2016.
- Abbruzzese G, Berardelli A, Defazio G. Hemifacial spasm. *Hand Clin Neurol* 2011; 100: 675-80.
- Lee JA, Kim KH, Park K. Natural History of Untreated Hemifacial Spasm: A Study of 104 Consecutive Patients over 5 Years. *Stereotact Funct Neurosurg* 2017; 95: 21-5.
- Jäger L, Reiser M. CT and MR imaging of the normal and pathologic conditions of the facial nerve. *Eur J Radiol* 2001; 40: 133-46.
- Auger RG. Hemifacial spasm: clinical and electrophysiologic observations. *Neurology* 1979; 29: 1261-72.
- Auger RG, Piepgras DG. Hemifacial spasm associated with epidermoid tumors of the cerebellopontine angle. *Neurology* 1989; 39: 577-80.
- Nielsen VK. Pathophysiology of hemifacial spasm: I. Ephaptic transmission and ectopic excitation. *Neurology* 1984; 34: 418-26.
- Uzun N, Kızıltan ME, Savrun FK. Hemifacial spazmda elektrofizyolojik ve radyolojik bulgular-2: Spazm aktivitesi. *Parkinson Hastalığı ve Hareket Bozuklukları Dergisi* 2001; 4: 36-41.
- Jannetta PJ, Abbasy M, Maroon JC, Ramos FM, Albin MS. Etiology and definitive microsurgical treatment of hemifacial spasm. Operative techniques and results in 47 patients. *J Neurosurg* 1977; 47: 321-8.
- Nishi T, Matsukado Y, Nagahiro S, Fukushima M, Koga K. Hemifacial spasm due to contralateral acoustic neuroma: case report. *Neurology* 1987; 37: 339-42.
- Meinke U, Ferbert A. Blink reflex in patients with an ischaemic lesion of the brain-stem verified by MRI. *J Neurol* 1993; 241: 37-44.
- Levin JM, Lee JE. Hemifacial spasm due to cerebellopontine angle lipoma: case report. *Neurology* 1987; 37: 337-9.
- Savrun FK, Kızıltan ME, Uzun N. Hemifacial spazmda kraniyal görüntüleme ve elektrofizyolojik bulgular-1: Beyin sapı refleksi eksitabilitesi. *Parkinson Hastalığı ve Hareket Bozuklukları Dergisi* 2001; 4: 30-5.
- Zappia JJ, Wiet RJ, Chouhan A, Zhao JC. Pitfalls in the diagnosis of hemifacial spasm. *Laryngoscope* 1997; 107: 461-5.

16. Hermier M. Imaging of hemifacial spasm. *Neurochirurgie* 2018; 64: 117-23.
17. Yaltho TC, Jankovic J. The many faces of hemifacial spasm: differential diagnosis of unilateral facial spasms. *Mov Disord* 2011; 26: 1582-92.
18. Kızıltan ME, Kızıltan G. Hemifacial spasm ve beyin sapı refleksleri. *Parkinson Hastalığı ve Hareket Bozuklukları Dergisi* 2000; 3: 24-31.
19. Kimura J. Blink reflex in facial dyskinesia. *Adv Neurol* 1988; 49: 39-63.
20. Jannetta PJ, McLaughlin MR, Baker FG, Kalia KK. Microvascular decompression. May M, editör. *The Facial Nerve*. May's second ed. New York, Stuttgart: Thieme; 2000. p. 483-9.
21. Defazio G, Berardelli A, Abbruzzese G, Coviello V, De Salvia R, Federico F, et al. Primary hemifacial spasm and arterial hypertension: a multicenter case-control study. *Neurology* 2000; 54: 1198-200.
22. Digre K, Corbett JJ. Hemifacial spasm: Differential diagnosis, mechanism, and treatment. Jankovic J, Tolosa E, editors. *Advances in neurology*, vol 49: Facial dyskinesias. New York: Raven Press; 1988. p. 51-176.
23. Barker FG, Jannetta PJ, Bissonette DJ, Shields PT, Larkins MV, Jho HD. Microvascular decompression for hemifacial spasm. *J Neurosurg* 1995; 82: 201-10.
24. Defazio G, Martino D, Aniello MS, Masi G, Logroscino G, Manobianca G, et al. Influence of age on the association between primary hemifacial spasm and arterial hypertension. *J Neurol Neurosurg Psychiatry* 2003; 74: 979-81.
25. Colosimo C, Chianese M, Romano S, Vanacore N. Is hypertension associated with hemifacial spasm? *Neurology* 2003; 61: 587.
26. Tan EK, Chan LL, Lum SY, Koh P, Han SY, Fook-Chong SM, et al. Is hypertension associated with hemifacial spasm? *Neurology* 2003; 60: 343-4.
27. Sharma S, Hashmi MF, Kumar A. Intracranial Hypertension. 2022 Feb 16. In: *StatPearls* [Internet]. Treasure Island (FL): StatPearls Publishing; 2022.
28. Muzerengi S, Moor C, Davies M. Facial spasm and headaches: should we call it "IIH-Spasm syndrome? *J Headache Pain* 2013; 14(Suppl 1): 225.
29. Selky AK, Purvin VA. Hemifacial spasm. An unusual manifestation of idiopathic intracranial hypertension. *J Neuroophthalmol* 1994; 14: 196-8.
30. Haouimi A. Idiopathic intracranial hypertension (IIH) - spasm syndrome. Case study. *Radiopaedia.org*. (accessed on 07 March 2022)
31. Chen BS, Newman NJ, Biousse V. Atypical presentations of idiopathic intracranial hypertension. *Taiwan J Ophthalmol* 2020; 11: 25-38.
32. Chan KM, Cammody RF. The sella and juxtaseilar region. Zimmerman RA, Gibby WA, Carmody RF, editors. *Neuroimaging, Clinical and Physical Principles*. Springer-Verlag 2000. pp. 1077-107.
33. Wang L, Shen Y, Li M, Chuang H, Ye Y, Zhao H, et al. Clinical manifestations and evidence of neurological involvement in 2019 novel coronavirus SARS-CoV-2: a systematic review and meta-analysis. *J Neurol* 2020; 267: 2777-89.
34. Vanaparthy R, Malayala SV, Balla M. COVID-19-Induced Vestibular Neuritis, Hemi-Facial Spasms and Raynaud's Phenomenon: A Case Report. *Cureus* 2020; 12:e11752.