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Original research article

Is hypertension a risk factor of hemifacial spasm?

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ABSTRACT

Objectives: The published data on the relation between arterial hypertension (AH) and hemifacial spasm (HFS) are controversial. The aim of the study was to determine the prevalence of AH in HFS patients and the relation of AH and compression of the brainstem at the region of vasomotor center.

Materials and methods: The study included 60 of primary HFS patients and 60 healthy controls matched by age. AH was defined according to WHO criteria. The vessel compression of the brainstem was measure on MRI scans in selected region of vasomotor center located in the ventro-lateral medulla (VLM), between the pontomedullary junction, retro-olivary sulcus and the root entry zone (REZ) of the IX and X nerves. Modeling and compression severity of the VLM was graded in the 0–3 scale.

Results: The prevalence of AH in HFS patients did not differ significantly from the control group (61.6% vs 45.0%, p = ns). VML compression by vessel was frequently found in HFS patients with AH than without AH (97.2% vs 60.9%, $\chi^2 = 11.0$, $p = 0.0009$). A similar relation was also found in the control group. The higher rate of VML vascular compression was related to the presence of AH in both, HFS patients and control group.

Conclusion: The prevalence of AH in HFS patients does not differ from controls. The VLM compression in HFS patients and controls is related to AH diagnosis. The association between AH and VLM compression is stronger in patients with higher degree of VLM compression.

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1. Introduction

Hemifacial spasm (HFS) is a rare multi-etiological movement disorder characterized by involuntary, unilateral, clonic or/and tonic muscle contraction in the region innervated by the facial nerve. The primary HFS, occurring in a vast majority of cases, is caused by the conflict between artery and facial nerve at the root entry zone (REZ) of the brainstem. The secondary HFS can be caused by many different compressive (e.g.: tumor, arteriovenous malformation, Paget disease) or non-compressive (e.g.: trauma, Bell's palsy, stroke, multiple sclerosis) lesions. Small volume of the posterior cranial fossa and arterial hypertension (AH) were suggested risk factors of HFS. The relation between AH and HFS was studied, but the results are not equivocal. The prevalence of AH in HFS patients was reported in the range from 39% to 67% [1–5]. The higher incidence of AH in HFS patients than in controls and in patients with blepharospasm, the other facial movement disorder, was also published [1,2,6]. However, Tan et al. [3] on Asian population and Colosimo et al. [4] on Italian population did not found statistically higher prevalence of AH in HFS patients than in the control group.

The aim of the study was to determine AH as a risk factor for HFS in relation to the compression of the brainstem at the region of vasomotor center.

2. Material and methods

All patients diagnosed as HFS registered in the Movement Disorders Out-patient Clinic, University Hospital, Krakow, Poland, in 2004–2010 years, were identified. Patients with secondary HFS caused by compressive or non-compressive lesions and patients with diseases causing secondary hypertension (renal disorders, thyroid diseases, pheochromocytoma, Conn's syndrome, Cushing's syndrome, drug-induced, etc.), as well as significant with other neurological diseases, cancer and other current serious or unstable clinically important diseases, were excluded. All others, assessed as the primary HFS, were invited to the study.

Control group was recruited from patients complained of hypoacusis and/or tinnitus without pathological findings in laryngological and neurological examination, diagnosed in the Outpatient Clinic of the Otolaryngology Department, University Hospital in Krakow.

At the screening visit, after informed consent, patients were interviewed and examined by neurologist, and the diagnosis of primary HFS was confirmed.

The severity of HFS was assessed based on the Tan scale (graded 0–5) and Clinical Global Impression (CGI) scale. AH was defined according to the WHO/International Society of Hypertension Writing Group [7] criteria in patients with earlier AH diagnosis, who were receiving antihypertensive medication and in patients with the systolic blood pressure ≥140 mmHg and the diastolic blood pressure ≥90 mmHg on 3 separate measures over a month with a pressure gauge in both upper limbs after 20-minute rest. Each measurement was performed twice.

The standard MRI examination in all involved patients and control group subjects (GE Signa HDxt 1.5 T) was followed by 3D FIESTA (TE/TR//FA/FOV/slice thk/NEX: 2.4/5/55/22/1 mm/2) and 3D T1 SPRG (TE/TR//FA/FOV/slice thk/NEX: 2.6/23/20/22/1 mm/2). All MRI scans were reviewed by one radiologist (MH) unaware of the subjects' medical history.

The existence of conflict between artery and facial nerve at the REZ of the brainstem (neurovascular conflict – NVC) was checked bilaterally. Compression of facial nerve at REZ, caused by NVC was graded on an 4 – point scale: 0 – absence of compression, 1 – mild compression, 2 – moderate compression, 3 – severe compression.

Compression of the brainstem at the region of vasomotor center was assessed by modeling the ventro-lateral medulla (VLM), between the pontomedullary junction, retroolivary sulcus and the REZ of the IX and X nerves. Compression severity of the VLM was graded according to the 0–3 scale, which was used previously by Watters et al. [8] and Chan et al. [9]: 0 – lack of the vascular contact with the medulla oblongata; grade 1 – contact only; grade 2 – vascular contact with medulla and its mild depression; grade 3 – increased medulla depression with displacement, deformity or rotation.

2.1. *Statistical analysis*

Numerical variables are expressed as mean ± SD. Qualitative variables are described as the absolute value of cases in the distinctive group. The variables' distribution were checked by the Shapiro–Wilk test. Statistically significance were assessed by χ^2 test between the quantitative variables, with the Yates's or Fisher's correction, if necessary. Student's t test was performed to evaluate data, follow a normal distribution, for other variables the Mann–Whitney test was used. To determine the correlation between numerical variables the correlation analysis was performed. Pearson correlation coefficient was calculated for normally distributed variables and the Spearman's rank correlation coefficient for a non-parametric measure. For the risk factors' assessment the logistic regression models were applied. Wald test was used to estimate the relationship between independent variables. p-Values <0.05 were considered statistically significant. Statistical analysis was performed using commercially available software (STATISTICA v. 6.0, StatSoft Inc. version 9.2 Poland) licensed for the Krakow University.

3. Results

115 of 129 registered HFS patients was assessed as a primary HFS and invited to the study. The 60 subjects followed the inclusion and exclusion criteria and agreed to be involved to the study. There were 42 (70.0%) women; the mean age was: 58.3 ± 9.1 years, and the mean duration of HFS symptoms was: 9.2 ± 6.9 years. The control group consisted of 60 subjects: 62% women, the mean age: 60.3 ± 10.9 years. There was any statistically difference in age and sex between patients and control group.

AH was diagnosed in 37 (61.6%) of HFS patients compared to 27 (45.8%) of the controls (the difference is statistically not significant) and similar to the prevalence of AH in the all 129 registered HFS patients (59.0%). The mean duration of AH in the HFS patients was slightly, but insignificantly, longer than

Table 1 – The correlation of the VLM compression and AH in the HFS patients and in the control group. The values in the columns 1–6 represent the average number and percentage of VLM compression for right and left side.

HFS patients			Control group			Statistical significance				
All patients $n = 60$	Patients with AH $n = 37$ (61.6%)	Patients without AH $n = 23$ (38.3%)	All patients $n = 60$	Patients with AH $n = 27$ (45.0%)	Patients without AH $n = 33$ (55.0%)	2 vs 3	5 vs 6	1 vs 4	2 vs 5	3 vs 6
1	2	3	4	5	6	7	8	9	10	11
39 (65.0%)	27.5 (74.3%)	11 (50.0%)	29 (48.3%)	18 (68.5%)	10.5 (31.8%)	$\chi^2 = 3.86$ $p = 0.049$	$\chi^2 = 7.89$ $p = 0.005$	Ns	Ns	Ns

Table 2 – Severity of the VLM compression by cerebral artery in HFS patients and control cases. The values represent the average number and percentage of VLM compression for right and left side.

Compression grade	HFS patients			Control group		
	All $n = 60$ (%)	With AH $n = 37$ (%)	Without AH $n = 23$ (%)	All $n = 60$ (%)	With AH $n = 27$ (%)	Without AH $n = 33$ (%)
0	21	9	12	31	8.5	22.5
	35.0%	24.3%	52.2%	51.1%	31.5%	68.2%
1	24	14.5	9.5	18	9.5	8.5
	40.0%	39.2%	41.3%	30.0%	35.2%	25.7%
2	13	11.5	1.5	10	8	2
	21.7%	31.1%	6.5%	16.7%	29.6%	6.1%
3	2	2	0	1	1	0
	3.3%	5.4%	0%	1.6%	3.7%	0%

in controls (11.3 ± 8.4 vs 8.1 ± 7.7 year, $p = 0.13$). In 25 (67.6%) patients the AH was diagnosed before HFS onset, in 4 (10.8%) patients AH was diagnosed at the same time as HFS and in 8 (21.6%) patients the diagnosis of AH was later then the first symptoms of HFS.

HFS patients with AH did not differ from other HFS patients in relation to sex (women: 62.1% vs 60.8%), the mean HFS duration (9.6 ± 16.6 vs 5.4 ± 2.9 year, $p = 0.24$), the side of the HFS (right side: 35.1% vs 43.4%; $p = 0.52$), HFS severity according to the CGI scale (5.1 ± 1.4 vs 5.1 ± 1.4, $p = 0.88$.), HFS severity according the Tan scale (3.2 ± 1.1 vs 3.0 ± 1.3, $p = 0.59$) or to the NVC on symptomatic side (97.2% vs 91.3%; $p = 0.29$). Patients with the HFS and AH were older than patients without AH (61.9 ± 10.3 vs 54.2 ± 11.1 years; $t = 2.7$, $p = 0.008$) and had later onset of disease (55.1 ± 10.9 vs 48.6 ± 11.0 year; $t = 2.2$, $p = 0.03$).

The VML compression was frequently found in patients with diagnosis of AH compared to the patients without AH diagnosis in the HFS group (97.2% vs 60.9% $\chi^2 = 11.0$, $p = 0.0009$), as well as in the control group (100% vs 57.6% $\chi^2 = 12.6$ $p = 0.004$). The VLM in patients with HFS and in control group was correlated with the prevalence of AH (Table 1). The higher rate of VML compression (2 or 3 degree) was frequently found in HFS patients with AH then in HFS patients without AH. The lower rate of VML compression (0 and 1 degree) was frequently found in patients without AH (Table 2). The correlation between rate of VML compression and AH was statistically significant on the right side ($\chi^2 = 5.89$, $p = 0.01$), and the left side ($\chi^2 = 5.11$, $p = 0.02$) of compression. Similar correlation was observed in the control group (right side: $\chi^2 = 10.46$, $p = 0.001$, left side: $\chi^2 = 3.87$, $p = 0.049$). There was no correlation between the NVC compression severity on nerve VII of the symptomatic and asymptomatic side and the AH occurrence ($\chi^2 = 0.20$, $p = 0.65$). Posterior inferior cerebellar artery (PICA) and vertebral artery (VA) were responsible for the most cases of VLM compression in both, HFS patients and control cases (Table 3).

41 (68.3%) patients with HFS revealed compression of VLM and NVC with VII nerve in the same side. There were no statistically significant differences of occurrence AH in group with the compression on VLM and NVC on the same side 28 (75.7%) and group with compression VML and NVC on opposite side 13 (56.5%).

Table 3 – Frequency of the VLM compression caused by cerebral artery among HFS patients and control cases.

Artery compressing on the VLM	HFS patients – n (%)		Control group – n (%)	
	Right sided compression $n = 38$	Left sided compression $n = 41$	Right sided compression $n = 25$	Left sided compression $n = 33$
Posterior inferior cerebellar artery (PICA)	20 (52.6%)	22 (53.6%)	14 (56%)	16 (48.5%)
Vertebral artery (VA)	13 (34.2%)	16 (39.0%)	10 (40%)	10 (33.3%)
VA and PICA	4 (10.5%)	3 (7.3%)	1 (4.0%)	2 (6.1%)
Nonspecific aberrant or ectatic artery	1 (2.6%)	0 (0%)	0 (0%)	5 (15.1%)

4. Discussion

The study showed that AH was only slightly and not significantly frequent in HFS patients than in controls, and do not support the view that AH is a risk factor of HFS. It is consistent with the last two publications [3,4], and different from previous studies on this topic. The reason of this difference could be due to selection of the control group. The control group in our study and in both last studies was selected from healthy subjects, but in many earlier studies controls were recruited from persons with other neurological diseases. The prevalence of AH in the Polish population was studied several times, and the results were in the wide range from 29 to 76% [10–12], but clearly related to the age. In the study of Zdrojewski et al. [10] the prevalence of AH for people aged 40–59 years was 34%, and for people aged over 59 years was 57%, similarly to the results in our control group (45%) aged 50–70 years. The significance of the slight difference between AH prevalence in HFS and the control group in our study is also diminish by the fact that in 22% of HFS patients AH was diagnosed later than HFS.

The relation of HFS and AH could be explained by the compression of the REZ of VII nerve and of the brain stem in VML area by the one of the cerebral artery. The compression of the REZ of VII nerve cause HFS and compression of the VML cause AH. The compression on VML was detected in MRI in almost all patients and controls with AH and in about 60% of patients and controls without AH. The previous studies in animal models or in human population, suggested the compression on VLM is a cause of AH [13–21]. The reason for that is the location of the neurons responsible for the blood pressure control in VLM area. Pulsating compression on VLM in rats increased activity of postsynaptic neurons of this region by glutamate receptors activation and resulted in AH [22]. In the study on patients with AH and compression on the VLM confirmed by MRI, there was significantly higher concentration of norepinephrine compared to both the controls without AH and the group of patients with AH but without conflict with VLM [23]. In study on young volunteers with compression on VML but without AH, Hohenbleicher et al. [24] found out the reduction of modulation of bar receptors reflex sensitivity under the influence of either physical activity or psychological stress.

The correlation between the compression of VLM and AH was confirmed in several studies by means of operational validation [14,25,26], MRI [17,18,27] or neuropathological verification [16]. There are also surgery publications from 80 and 90 decades of the XX century describing regression of AH after surgical decompression of VLM and concomitant compression syndrome of V, VII and IX nerves roots. This is important indirect prove that idiopathic AH is caused by the compression of VLM [13,14,26,28]. Total relief of AH was in 55–85% patients [14,28].

In several angiography or autopsy using studies left sited VLM compression was more frequently related to idiopathic AH than right sited VLM compression (75–83% vs 0–17%), significantly more often as compared to the control group (11–35%) [16,18,27]. The studies using MRI, however, do not showed the difference in left vs right side of VLM compression in AH patients [8,29–31] and founded much more VLM compression signs in control subjects, up to 55% [8]. It was explained by some methodological issues including lack of neuroimaging procedures standardization, thick (5 or 7 mm) MRI layers, incorrect selection of a control group or inappropriate assessment of the compression [21]. Our study, using standardized MRI methodology, similarly to the previous MRI studies, do not confirmed the view that the left site VLM compression is more frequently related to the AH diagnosis than the right site VLM compression.

The most important result of our study is the finding of the relation between AH and the VLM compression supporting by the stronger association between higher degree of compression and AH in HFS patients. The correlation between VLM compression by vessel and the presence of AH was analyzed in three previous studies in HFS patients. Nakamura et al. [5] comparing 82 HFS patients and 82 controls do not found any difference in AH prevalence, but similar to our study reported the correlation between AH and the VLM compression visualized in MRI. The compression on VLM was found in 86% of HFS patients with AH in comparison to 33% patients without AH and to 50% of the controls with AH and 15% of the controls without AH. The correlation between VLM compression an AH was also reported twice by the same authors from Singapore. In the first study the VLM compression was present in 77.5% of HFS patients with AH and in 53% without AH [9]. The next study documented significant more frequent presence of VLM compression in patients with both AH and HFS than in patients with AH only and in the controls (respectively 82.2% vs 72.4% vs 41.2%) [32]. The authors assessed also the effect of VLM compression rate in four point scale. The average of compression rate was significantly higher in HFS patients with AH than without [9]. The higher rate of compression was statistically more frequent in patients with HFS and AH than in HFS patients without AH.

The most common vessels responsible for the VLM compression in our study was PICA (53%) and VA (36%). This is consistent with many other studies, e.g. Kleineberg et al. [27] revealed that VLM compression is caused mainly by PICA (35%), VA (29.4%) and anterior inferior cerebellar artery (AICA) (19%).

The result of our study strongly support the view on the relation between VLM compression and AH in HFS patients, but some limitations of the study should be mentioned, especially in the concluding on the relation between AH and VLM compression in general population. At first, the assessment of the relation between VLM compression and AH was not the primary goal of this study. The correlation was shown on the occasion of the VII-th nerve compression assessment. At second, the number of patients was too small to assess the relation between AH and VLM in general population. HFS is a relatively rare disease and it is not possible to include hundreds of HFS patients to study relation of VLM compression and AH. It should be also noted that not all studies on general population support the view on the relation between VLM compression and AH [8,29–31]. At third, the presence of VLM compression in our and other studies was revealed in over 50% of patients without AH. It suggests that the relation between VLM compression and AH is not direct and even doubtful. Further studies on general population and HFS patients are necessary.

5. Conclusions

The study does not support the view that AH is a risk factor of HFS. VLM compression by vessel was frequently found in HFS patients and AH, and controls with AH. The higher rate of VLM compression also correlated with AH, both in HFS patients and controls.

Conflict of interest

None declared.

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None declared.

Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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