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Cardiovascular responses to tilting in female migraine patients with impaired autonomic reaction

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Abstract

Background: Orthostatic hypotension (OH) is the second most common cause of syncope, prevalent in migraine patients. **Objective:** To assess the cardiovascular responses to tilt test in female migraine patients. **Methods:** This experimental study was conducted on 70 newly diagnosed female migraine patients (age 15–30 years & BMI 18.2-26.59kg/m²). Among them, 20 migraine patients were with normal (group MN) and 20 patients had impaired autonomic reaction (group MI). Twenty (20) apparently healthy subjects with similar age and BMI were also enrolled as control. Autonomic reactivity of all participants was assessed by conventional autonomic function test (CAFT). Head Up Tilt Test (HUTT) of all subjects was done by tilting 60^0 for 10 min using a motorized tilt table. Cardiovascular responses to tilting were observed by change in Heart rate (Acceleration index and Brake index); change in systolic blood pressure (SBP_{30s-0} and SBP_{1min-0}); change in diastolic blood pressure (DBP_{30s-0} and DBP_{1min-0}), after the tilting from supine position. Data were analyzed using one-way ANOVA followed by Bonferroni's post hoc test. **Results:** In this study, the Acceleration index was significantly lower in Group MI compared to Group MN ($p \leq 0.001$) and control ($p \leq 0.01$). But the Brake index was significantly lower ($p \leq 0.05$-, $p \leq 0.01$) in both group of patients than control. Again no significant difference found in Brake index between Group MN and Group MI. $SBP_{30sec-0}$ was significantly higher in both of the patient group ($p \le 0.01$, $p \le 0.001$) than those of control. In addition $SBP_{30sec\ -0}$ was also significantly higher ($p \le 0.001$) in Group MI compared to Group MN. Again, SBP_{1min-0} was significantly higher($p \le 0.001$) in MI compared to MN and control. DBP30sec-0 and DBP1min-0 were significantly lower ($p<0.001$, $p<0.05$, $p<0.001$) in Group MI compared to Group MN and control. **Conclusion:** It can be concluded that cardiovascular responses to tilting was diminished in migraine patients, which was more pronounced in patients with impaired autonomic reaction.

Keywords: Migraine, Syncope, Orthostatic hypotension, Tilt test.

Introduction

Migraine is a disabling primary headache disorder that is linked with alterations in neurological, gastrointestinal and autonomic functions. It is characterized by repeated episodes of intense headache, commonly associated with symptoms such as nausea, photophobia, phonophobia and occasionally preceded by visual or sensory auras.[1] The global prevalence of migraine estimated to be 1.1 billion. Over the past three decades, there has been a significant increase in the occurrence of migraine worldwide.[2-3] According to the World Health Organization (WHO), migraine is one of the top twenty most disabling illnesses in the world. Approximately 12% of the global population is affected by migraine.[4] Both men and women suffer from migraines, but women are disproportionately affected, at a ratio of 3:2.[5] Hormonal influences, particularly during different phases of the menstrual cycle, may impact autonomic nervous system (ANS) activity, though their effect is minimal during the early follicular phase.[6-7] The occurrence of migraine reaches its peak between the ages of 15 and 24.[8]

In individuals with migraine, the presence of autonomic nervous system (ANS) dysfunction is well recognized and is thought to contribute to symptoms like nausea, vomiting, diarrhea, pallor, flushing, piloerection and sweating.[9] The pathophysiology of migraine plays key role for development of both sympathetic and parasympathetic nervous system disturbances.[10]

Ewing and Clarke's five-cardiovascular reflex test protocol remains a clinical standard for bedside autonomic testing. By cardiovascular reflex test, decreased parasympathetic function in migraine patients compared to controls has been reported.[10-12]By these conventional autonomic function tests patients with and without autonomic impairment can be classified[13-14]

A sudden drop in cerebral perfusion often leads to syncope, which is a frequent symptom with multiple etiologies, migraine being one of them.[15] Forty-six percent of individuals with migraine have experienced syncope at least once and are more prone to recurrent syncopal episodes compared to those without migraine. [16-17]

OH is a common manifestation of autonomic dysfunction and second most common cause of syncope.[18] Migraine has been found associated with OH and also occasionally with syncope.[19]

Head-up tilt table testing (HUTT) is a highly effective orthostatic tolerance test for evaluating episodes of syncope caused by autonomic dysfunction. The tilt table test was first introduced in 1986 as a method to diagnose vasovagal syncope.[20] It has been used for over half a century to assess physiological responses and is now regarded as the “gold standard” for syncope evaluation.[21] It is widely used to assess orthostatic intolerance conditions such as orthostatic hypotension, neurally mediated syncope, postural tachycardia.[22]

Migraine patients experience recurrent syncopal episodes due to OH which is related to impaired autonomic reaction. Conflicting reports of higher rates of OH in migraine patients compared to controls published. Moreover, very limited data exist regarding cardiovascular responses to tilting in female migraine patients with impaired autonomic reaction.

Therefore, this study aimed to investigate cardiovascular responses to tilting in female migraine patients with impaired autonomic reaction. The outcome of this study will provide the clinicians guideline to develop a preventive strategy to reduce the risk of syncope in migraine patients.

Methods

Study Design and setting

This Experimental study was conducted in the Department of Physiology, Bangladesh Medical University (BMU), Shahbag, Dhaka from March 2023 to February 2024.

Study participants and sampling

A total of 70 female migraine patients attending the Outpatient Department of Neurology, BMU, were enrolled in this study. All patients were diagnosed by a neurologist according to the International Headache Society (IHS) diagnostic criteria for migraine.[1] Twenty apparently healthy female subjects were enrolled by purposive sampling. Migraine patients were further divided into 2 subgroups according to the outcome of CAFT. Twenty (20) patients with normal autonomic function were designated as MN and 20 patients with impaired autonomic function were designated as MI. For comparison, age and BMI matched 20 apparently healthy females were enrolled as Control.

Exclusion criteria

Participants were excluded if they had a history of fainting or unconsciousness, diagnosed orthostatic intolerance, pregnancy, lactation, menstruation, hormonal contraceptive use or used drugs affecting ANS function. Patients were free from any systemic illnesses such as cardiovascular (myocardial infarction, hypertension, arrhythmia, heart failure), neurological or psychiatric disorders, diabetes mellitus, respiratory (asthma, COPD), renal, thyroid, liver diseases or rheumatoid arthritis.

Data collection procedure

The aim and procedure of this study was explained and informed written consent was taken from all seventy participants. Then a detailed personal, medical, family and dietary history, MRI reports was taken from them. A thorough physical examination of the subjects was done. Height and weight were taken and BMI was calculated. Then for biochemical tests, 4 ml of venous blood was collected under aseptic precautions from seventy patients as well as from healthy control for screening of random blood glucose, serum creatinine, TSH and ALT. In day 2, after getting biochemical test reports, selected participants were instructed to finish dinner by 9:00 pm, sleep well, avoid stress and sedatives, and take a light breakfast without tea or coffee before attending the Noorzahan Begum Neurophysiology Laboratory, Department of Physiology, BMU between 8-9 am for autonomic tests.

Conventional autonomic function tests (CAFT)

In previously described way these tests were performed.[23] The HR was measured directly and continuously by ECG (True Beat 200 ECG machine, Clarity Medical Pvt Ltd, India). Parasympathetic reactivity was assessed by observing heart rate variation during deep breathing, the immediate heart rate response to standing, and the heart rate response to the Valsalva maneuver. Sympathetic reactivity was assessed by measuring blood pressure changes in response to standing and during sustained isometric handgrip exercise. By these tests, patients with normal and impaired autonomic reaction were classified.[13-14] The presence of parasympathetic neuropathy was determined when two or more heart rate based tests were impaired, whereas sympathetic neuropathy was identified when one or more of the two blood pressure based tests showed impairment.[13]

Tilt test

Subjects were briefed on the procedure for full cooperation, then asked to wear a gown and to rest on the tilt table for 10 minutes. Three Velcro straps secured them to prevent detachment during 60° tilting. Supine HR, SBP and DBP were recorded in a quiet, temperature-controlled (25°C) lab.[24] Tilting was done at 60° for 10 minutes using a motorized tilt table (Hi-Lo Mode; 220V; Cat No: IEMR4093HL; International Electro Medical Co., India), data were collected at one-minute intervals for 10 minutes following tilting. BP was measured using an automated sphygmomanometer (HME-7120, Omron Health Care Co. Ltd, Vietnam). Subjects were instructed to avoid leg movement and report any discomfort (chest pain, dizziness, nausea, sweating) for immediate termination of the test. Cardiovascular responses were assessed by calculating HR (acceleration and brake index), SBP changes (SBP_{30s-0} and SBP_{1min-0}), and DBP changes (DBP_{30s-0} and DBP_{1min-0}).[25]

Acceleration and brake indices were measured from ECG tracings according to previously described methods.[25] The acceleration index reflects the initial increase in heart rate immediately after tilting, expressed as the percentage rise from the pre-tilt heart rate, while the brake index represents the recovery of heart rate toward its pre-tilt value, expressed as the percentage change from the initial heart rate reached during tilting. [25-26] SBP change after tilting was assessed by calculating $SBP_{30sec-0}$ and SBP_{1min-0} previously.[27] $SBP_{30sec-0}$ is the difference between the fall at 30 seconds post-tilt and pre-tilt SBP; SBP_{1min-0} is the difference between the fall at 1 minute post-tilt and pre-tilt SBP. These two SBP change values reflect the degree of systolic blood pressure reduction at two distinct time points after tilting. An increase in DBP over time after tilting indicated the diastolic blood pressure response to passive orthostatic stress, which was noted in all groups. The changes in DBP over time after tilting represent the magnitude of diastolic blood pressure increase at two separate time points.[26]

Statistical analysis

Data were expressed as Mean ± SD. Statistical analysis was done using SPSS version 25. One-way ANOVA followed by Bonferroni's post hoc test was done. p value of <0.05 was considered as statistically significant.

Results

Age, BMI ,Resting pulse rate, SBP and DBP before tilting was also similar in all three groups (Table I)

Acceleration index and Brake index

After passive orthostatic stress as induced by tilting maneuver, the acceleration index was significantly lower in group MI than those of group MN ($p\leq0.001$) and Control ($p\leq0.01$). There was no significant difference between control and group MN.

The Brake index was significantly lower in MN ($p\leq0.001$) and MI ($p\leq0.01$) than those of Control. Furthermore, no significant difference was found in Brake index between group MN and MI (Table II)

Changes of SBP and DBP

In this study, SBP initially dropped to its lowest point within 30 seconds of tilting, then gradually increased but remained below pre-tilt levels at 1 minute. Control subjects showed a slight SBP decrease at both 30 seconds and 1 minute. Migraine patients with impaired CAFT had a significantly greater SBP fall at both time points, while those with normal test showed a significant fall only at 30 seconds. All groups exhibited a compensatory SBP rise by 1 minute, but this response was markedly diminished in the impaired test group. DBP, on the other hand, increased at both 30 seconds and 1 minute post-tilt. Though after that it tends to decrease with time, still it remained above the pre-tilt value at 1 minute after tilting. Patients with impaired test showed a reduced DBP rise at both time points compared to migraine with normal test and controls, who had similar DBP responses (Table II)

Table I : Characteristics of the three groups of subjects included in the study(N=60)

Groups	Group MN (n=20)	Group MI (n=20)	Control (n=20)
Age (Years)	23.80±4.61	25.85±4.15	26.40±4.36
BMI (Kg/m^2)	22.87±1.90	21.88±2.66	22.89±1.39
HR (beats/min)	82.80±9.89	79.75±10.44	78.30±7.24
SBP (mm Hg)	124.75± 12.51	127.20±10.83	124.30± 9.96
DBP (mm Hg)	80.45±5.73	76.60±7.45	78.90±8.05

Data were expressed as mean ± SD. Statistical analysis were done one way ANOVA followed by Bonferroni's post hoc test; Group MN- Migraine patients with normal CAFT, Group MI- Migraine patients with impaired CAFT, Control- apparently healthy control with normal CAFT. n- Number of the subjects in each group; N- total number of subjects. HR-heart rate, SBP-Systolic blood pressure, DBP- Diastolic blood pressure. CAFT- Conventional Autonomic Function Test.

Table II: Cardiovascular responses Indexes induced by tilting up (N=60)

Variables	Group MN (n=20)	Group MI (n=20)	Control (n=20)
Acceleration index (%)	14.49±4.04	09.30±2.43* ###	13.56±4.50
Brake index (%)	6.15±2.41$^{\$}$	5.67±1.62*	8.92±4.74
$SBP_{30sec-0}$(mm Hg)	-8.25±4.29$^{\$}$	-15.65±4.15***###	-4.10±2.07
SBP_{1min-0} (mm Hg)	-3.75±2.59	-12.50±4.89***###	-2.60±4.50
$DBP_{30sec-0}$(mm Hg)	4.65±1.81	2.00±1.58***#	6.65±2.71
DBP_{1min-0} (mm Hg)	5.55±1.87	2.80±2.06***###	7.00±1.97

Data were expressed as mean ± SD. Statistical analysis were done by one way ANOVA followed by Bonferroni's post hoc test; SBP_{30s-0} -Baseline and 30 seconds after tilt difference;
SBP_{1min-0} -Baseline and 1 minute after tilt difference; $DBP_{30sec-0}$-Baseline and 30 seconds after tilt difference; DBP_{1min-0} -Baseline and 1 minute after tilt difference; Group MN- Migraine patients with normal CAFT; Group MI- Migraine patients with impaired CAFT; Control- Apparently healthy subjects with normal CAFT; CAFT-Conventional Autonomic Function Test, N= total number of subjects. n- Number of the subjects in each group. *This depicts comparison of Control vs Group MI, *p<0.05, ***p<0.001. $^{\$}$This depicts comparison of Control vs Group MN, $^{\$}$p<0.05, $^{\$\$\$}$p<0.001, $^{\#}$This depicts comparison of Group MN vs Group MI, $^{\#}$p<0.05, $^{\#\#\#}$p<0.001.

Discussion

This study assessed cardiovascular responses to orthostatic stress in migraine patients using a passive tilt table test, comparing responses between migraine with normal autonomic reaction and impaired autonomic reaction as well as with healthy controls.

This study observed a significantly reduced acceleration index in the MI group compared to the MN group, which was similar in diabetic patients with autonomic dysfunction..It was suggested that impaired autonomic activation is the underlying cause, and a similar mechanism may be responsible for the reduction observed in our study. [26-27]

The MN group exhibited an Acceleration index similar to the control group suggesting autonomic function in MN was similar to healthy subjects.. Previous studies involving other autonomic function tests suggested that in migraine patients, autonomic dysfunction predominantly involves parasympathetic control and baroreflex sensitivity. However, sympathetic responses are often preserved[28-29] This preserved sympathetic activity, together with intact vagal withdrawal at tilt onset, may explain the near-normal or slightly higher Acceleration index observed in the MN group.

In this study the lower Brake Index observed in both migraine patient groups compared to controls is consistent with diabetic patients[24] suggesting impaired parasympathetic reactivation and diminished sympathetic compensatory response. This observation is indicating poor parasympathetic reactivation is Migraine associated complication.

In this study resting BP status in migraine patients did not find baseline sympathetic dysfunction [28,30]

In the control subjects, the tilting effect showed only a slight fall in SBP at both time points whereas significantly greater fall in SBP was noted at both time points in MI patients, on the other hand the MN group showed a significant fall only at 30 seconds. These findings suggested cardiovascular sympathetic hypofunction in migraine[31].This fact emphasized the reduced sympathetic output causing insufficient vasoconstriction and weaker baroreflex-mediated compensation, resuling a greater drop in SBP[31].

In addition, a reduced rise of DBP at 30 seconds and 1 minute after tilting in the MI group contrast, the similar post-tilt rise of DBP in group MN and control. The maximal rise of DBP in control subjects at both 30 seconds and 1 minute after tilting agrees previous reports.[32] But the post tilt reduced rise of DBP in MI migraine patients might be attributed to sympathetic hypofunction, possibly combined with mild parasympathetic dysfunction and CNS-mediated autonomic dysregulation, resulting in insufficient peripheral vasoconstriction during postural stress.

In this study, group MN exhibiting a lower Brake index, a greater fall in SBP, a less rise in DBP immediately after tilt, as well as a lesser recovery of SBP and DBP over time demonstrated weakness in sympathetic activation power due to gravitational stress on circulation. Similar explanation might work against much lower Brake index, much greater drop of SBP and lesser rise of DBP MI group. Therefore migraine may have some contribution to this sympathetic attenuation despite their normal autonomic function test and greater degree of sympathetic weakness in patients with impaired autonomic activity.

In this study no migraine patient experienced syncope during tilt. It might be due to the fact that the autonomic dysregulation was not that severe in these patients to develope syncope.In the present study, six patients of MI group and one patient of MN group but none among control subjects showed evidence of orthostatic hypotension.. The increased occurrence of OH in migraine attributed to the greater sympathetic hypofunction.[33]

These observations suggest an enhancement of parasympathetic inhibition coupled with inadequate sympathetic compensatory activity which was more severe in migraine patients with autonomic dysfunction. These phenomena were linked to early-stage damage in the parasympathetic nervous system and later-stage damage in the sympathetic nervous system. Previous studies also showed similar tilt response in migraine patients but their autonomic function was not known.[32-33]

It is evident that the greater deviation of autonomic modulation than normal in order to compensate the gravitational stress induced challenge to the cardiovascular neural regulation was associated with autonomic neuropathy in migraine. As a result, migraine patients who already have developed autonomic neuropathy showed inadequate autonomic regulation and poor cardiovascular control over time.

So, early diagnosis of autonomic dysfunction in migraine is very important to reduce the rate of morbidity and mortality. HUTT is an excellent technique for the assessment of autonomically mediated syncope.

Conclusion

This study concluded that cardiovascular responses to tilting may be diminished in migraine patients, which could be more pronounced in patients with impaired autonomic reaction.

Ethical clearance

The ethical aspects of this study involving human subjects followed Helsinki (1964) ethical guideline and was first approved by departmental ethical and academic committee. It was then further reviewed and approved by Institutional Review Board (IRB) of BMU.

Conflict of interest Authors of this article declares no conflict of interest.

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