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RESEARCH ARTICLE

Electrocardiographic changes in migraine

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ABSTRACT

Background: Migraine is the second most common cause for primary headache. Migraine often results in considerable disability and decrease in the patients' quality of life. Its prevalence is highest in the age group of 25-55 years, which are the peak years of economic productivity. In literature, autonomic nervous system impairment in migraine is well documented, i.e., migraine is of neural origin. Furthermore, autonomic dysfunction in migraine may also affect autonomic innervation of the heart resulting in electrocardiographic (ECG) changes. **Aims and Objectives:** To study ECG changes in patients of migraine. **Materials and Methods:** Thirty migraine female patients aged 25-55 were included. 30 age and gender-matched controls were chosen for comparison. In all the study individuals and controls, resting ECG was recorded. Statistical analysis was done by unpaired Student *t*-test. **Results:** The resting heart rate was found to be significantly higher in migraineurs compared to controls ($P < 0.05$). QTc interval although was within the normal range, it was significantly more ($P < 0.01$) in migraineurs when compared to controls. **Conclusion:** ECG changes were found in migraineurs which indirectly suggest disturbed autonomic innervation to heart.

KEY WORDS: P wave Dispersion; Electrocardiographic Changes; Migraine

INTRODUCTION

It has been proposed that both sympathetic and parasympathetic dysfunctions have played important roles in the pathophysiology of migraine.[1] Disturbance of autonomic nervous system (ANS) may affect atrial and ventricular repolarization of heart. Thus, disturbance in the autonomic innervation of heart in patients with migraine may also result in possible electrocardiographic (ECG) manifestations.[2] The objective of this study was just to study the electrical changes in heart which occur as part of the dysfunction of ANS in migraine patients. Furthermore, this study aims to give some information about the cardiovascular status and also, the ECG changes seen in patients of migraine, the knowledge of which may be applied by a physician while prescribing cardiac drugs to migraine patients.

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MATERIALS AND METHODS

In this study, data were collected from 30 migraine patients diagnosed according to Headache Classification Committee of the International Headache Society 2004,[3] in the age group of 25-55 years, attending neurology outpatient department. 30 age and gender-matched controls were selected. The study was performed during the morning session of 9 am to 12 noon in the Department of Physiology of Karnataka Institute of Medical Sciences, Hubli, Karnataka. Tests were conducted in the migraine-free period in between the attacks. Patients were not taking any drugs at the time of tests of this study. Although they were prescribed drugs by the physician, still they were taking drugs only on migraine attack, and since

this study tests were conducted in migraine-free period, the patients were not on any drugs.

The following inclusion and exclusion criteria were applied for selection process:

Inclusion Criteria

Individuals aged between 25 and 55 years, who report of headache lasting 4-72 h with normal physical examination and are diagnosed as having migraine by a neurophysician along with at least two of the following features:

1. Unilateral pain
2. Throbbing pain
3. Aggravated by movement
4. Moderate or severe intensity.

Plus at least one of the following features:

1. Nausea/vomiting
2. Photophobia and phonophobia.

(Adapted from the Headache Classification Committee of International Headache Society, 2004).[3]

Exclusion Criteria

1. Upper respiratory tract infections
2. Meningitis
3. Intracranial hemorrhage
4. Intracranial tumors
5. Temporal arteritis
6. Glaucoma
7. Any condition associated with ANS dysfunction such as diabetic neuropathy, familial dysautonomia, drugs affecting ANS (beta-blockers, etc.), and tobacco abuse either smoking or chewing.

ECG Recording in Migraineurs

ECG was recorded in supine position after a rest period of 10 min, AUTO mode in the instrument was selected, and the following parameters were noted in migraineurs and controls: Resting heart rate (beats/min), PR interval (ms), P wave (ms), QRS (ms), T wave (ms), QT interval (ms), QTc interval (ms), P axis (degrees), QRS axis (degrees), T axis (degrees), and P wave dispersion (in ms). P wave dispersion was calculated by noting the P wave duration in all the 12 leads. P wave duration was measured by scanning the ECG paper at 600 dpi resolution, and accurate measurement of P wave duration in all leads was noted down. The difference between the maximum and minimum duration was taken. Mean and standard deviation were calculated for all parameters. The statistical analysis was done using Student *t*-test. $P < 0.05$ was considered as significant (S), $P < 0.01$ was considered as highly significant (HS), and $P < 0.001$ was considered as very HS (VHS). Graph pad cloud software was used for analysis.

RESULT

The following ECG parameters: QTc interval and QRS axis showed significant difference between controls and migraine individuals. QTc interval, although within the normal range, was longer significantly in migraine individuals as compared to the controls, and QRS axis (in degrees) was significantly less in migraine individuals as compared to the controls. The resting heart rate in migraineurs was significantly higher than in controls. This may indicate a slight parasympathetic hypoactivity or sympathetic hyperactivity (Tables 1-3).

DISCUSSION

ECG Changes in Migraineurs

The following ECG parameters were noted in migraineurs and controls:

Resting heart rate (beats/min), PR interval (ms), P wave (ms), QRS complex (ms), T wave (ms), QT interval (ms), QTc interval (ms), P axis (degrees), QRS axis (degrees),T axis (degrees), and P wave dispersion (in ms).

There was significant difference in QTc interval and QRS axis between migraineurs and controls. The other remaining

Table 1: Anthropometric data

Parameters	Mean±SD		*t*	*P*
	Controls	Individuals		
Age (years)	26.83±4.04	28.57±5.44	1.467	0.14 (NS)
Height (cm)	153.1±7.09	153.8±5.38	0.429	0.668 (NS)
Weight (kg)	48.53±6.84	51.23±7.76	1.45	0.152 (NS)
BMI (kg/m^2)	20.74±2.98	21.52±3.21	1.01	0.314 (NS)

SD: Standard deviation, NS: Not significant

Table 2: PR interval (ms), P wave (ms), QRS complex (ms), and T wave (ms)

Parameters	Mean±SD		*t*	*P*
	Controls	Individuals		
QT interval (ms)	361.47±22.06	360.57±22.81	0.145	0.885 (NS)
QTc interval (ms)	402.13±21.03	416.33±16.44	2.91	0.005 (HS)
P wave dispersion (ms)	45.33±13.19	47.33±16.49	0.519	0.605 (NS)

SD: Standard deviation, NS: Not significant, HS: Highly significant

Table 3: QT interval (ms), QTc interval (ms), and P wave dispersion (ms)

Parameters	Mean±SD		*t*	*P*
	Controls	Individuals		
PR interval (ms)	139.97±14.79	144.27±22.02	0.887	0.378 (NS)
P wave (ms)	106.4±9.15	107.83±8.85	0.616	0.539 (NS)
QRS complex (ms)	76.57±6.22	79.57±6.40	1.84	0.07 (NS)
T wave (ms)	161.23±15.37	163.47±15.62	0.558	0.578 (NS)

SD: Standard deviation, NS: Not significant, HS: Highly significant

parameters did not show any statistically significant difference between migraineurs and controls:

The QTc interval of migraineurs was 416.33 ± 16.44 ms and that of controls was 402.13 ± 21.03 ms. Thus, although the QTc interval was within the normal range in migraineurs, it was significantly more in migraineurs when compared to the controls.

Duru et al., in their study, they have discovered significantly increased QTc interval in migraineurs along with increased QTc dispersion and increased P wave dispersion during migraine attacks.[4]

Aygun et al. have also reported increased QTc and PR intervals during migraine attacks compared with pain-free periods.[5] It has been reported that dysfunction of the ANS influences ventricular repolarization, and therefore, increases the QT interval on the ECG.[6]

The QT interval reflects depolarization and repolarization of myocardial cells. Factors that augment depolarization or delay repolarization of myocardial cells can increase QT interval length. Genetic factors and nongenetic factors such as electrolyte disturbances and drugs influence the QTc.

There is indirect evidence that the ANS activity also influences QTc.

QTc is prolonged in patients with diabetic autonomic neuropathy[7] and familial dysautonomia patients.[8]

Similarly, QTc is increased in patients with primary autonomic failure due to pure autonomic failure or multiple system atrophy.[9]

It has been reported that prolonged QT intervals appear to correlate with the severity of autonomic dysfunction in various diseases such as diabetes mellitus.[7]

Furthermore, manipulation of the ANS in experiments demonstrated the influence of imbalanced sympathetic activity on the heterogeneity of repolarization in the myocardium, resulting in QT interval prolongation.[10,11]

Further studies have shown that a prolonged QT interval and P wave dispersion in the pain-free period can be predictors of atrial and ventricular arrhythmias.[4,6]

These findings may reflect atrial and ventricular repolarization abnormalities that are affected by a disturbed ANS.

CONCLUSION

QTc interval is found to be significantly more in migraineurs when compared to the controls suggesting disturbed autonomic innervation to the heart. Our study has focused on duration of various intervals and axes of various waves, and further analysis of ECG could not be done to study the heart rate variability in migraine patients as our instrument was not equipped for it.

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