Study of Incidence and Pattern of Electrocardiographic Changes in Cerebrovascular Diseases

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Abstract: The pathways involved in brain-heart interaction have been explicated in both animal and human studies. Neurogenic mechanism suggested that, sympathetic nervous system stimulation can produce the arrhythmia by its activation. In addition both anatomical and physiological evidence implicated the hypothalamus in cardiac control. Several experimental studies reported that electrical stimulation of posteriorly located area of cardiovascular sympathetic control and anterior parasympathetic control region. Cardiac dysfunction after cerebrovascular accidents is manifested by ECG changes, cardiac arrhythmias, elevated cardiac biomarkers and hemodynamic alteration. Present study was taken to know the incidence and pattern of ECG changes in patient with cerebrovascular disease. Total 56 cases of cerebrovascular disease patients were studied to know the ECG changes. They were categorized into 3 different cerebrovascular diseases based on the CT scan findings as cerebral infarction, cerebral hemorrhage and subarachnoid hemorrhage. Out of 56 patients, 69.6% of all cerebrovascular disease patients had some form of ECG abnormalities. Higher incidences of abnormal ECG found in cerebral hemorrhage patients 72.7%. Sinus bradycardia was seen in 18.1% of cerebral hemorrhage patients, none of the patients had sinus bradycardia in cerebral infarction and subarachnoid hemorrhage. The study was concluded that ECG changes in cerebrovascular disease patients were not associated with any particular site of cerebral lesion but rhythm changes in pattern of ECG were specific as categorized by CT scan findings.

Keywords: ECG, Hemorrhage, CT scan, Cerebrovascular disease.

INTRODUCTION
Cardiac abnormalities were described with various CNS diseases including seizures, trauma, ischemic stroke, ICH. Lower incidences of cardiac abnormalities may be seen in tumours, electroconvulsive therapy and meningitis [1]. The anatomy and physiology of pathways involved in brain-heart interaction have been observed in both animal and human studies. Functional areas of cerebral cortex with connection to autonomic nervous system may also elicit cardiac response. The autonomic emotional interaction with cardiovascular function has been linked to central nucleus of amygdale [2].

ECG abnormalities described in neurological disease are among the most striking deviation from the normal. Activation of the sympathetic nervous system may reproduce the arrhythmia. The changes in Electrocardiograph (ECG) was first accounted in upright T waves prolonged QTc in patients with subarachnoid haemorrhage was published in 1947 [3,4].

The widespread connection of insular cortex with other areas of brain which are involved in autonomic control explains the effects of cardiac changes during neurological injury. Several studies suggest that the insular cortex has a pivotal role in integrating autonomic response. Cerebrovascular stroke can alter cardiovascular tone by directly damaging the insular cortex or other interrelated areas shifting the balance towards a predominance of sympathetic activation [5]. Elevated baseline heart rate and blood pressure were noted after right-sided injury and significantly increased baroreflex sensitivity was obtained after left sided injury.

The insular cortex in rats as a site from which lethal cardiac arrhythmias and myocardial damage could be produced, resembling changes seen in patients after stroke and sudden death in patients with epilepsy [6, 7]. These micro stimulation experiments in the rat posterior insular cortex produced stereotyped ECG changes from progressive atrio-ventricular block leading to complete heart block, interventricular block, QT-interval...
prolongation’s, ST-segment depression and finally death in systole [8].

ECG abnormalities were frequently found in patients with subarachnoid hemorrhage and other cerebral vascular injury. ECG changes after stroke consisting of large inverted T waves, prolonged QT intervals and large septal U waves that has become distinctive of cerebral vascular injury. Further evidence of a neurogenic mechanism of cardiac injury comes from studies of cardiac function after SAH, which typically affects younger patients without a history of coexistent cardiac disease [9]. Global or regional left ventricular systolic dysfunction on echocardiogram has been described after SAH with an approximate incidence of 10 to 28 percent. Brain injury and subarachnoid haemorrhage have been reported to cause ‘J’ waves due to the prominent action potential notch in epicardium [10]. The current study was undertaken to present the incidence and pattern of ECG changes in patients with cerebrovascular diseases.

MATERIALS & METHODS

The present observational study was conducted in patients attended at tertiary care hospital, Bangalore. Total 56 cases of cerebrovascular disease patients were studied. The study was reviewed and approved by Institutional ethical committee. Patients with acute cerebrovascular diseases are included in the study. Cerebrovascular patients with past history of ECG abnormalities, underlying heart disease, hepatic diseases and renal diseases are excluded from the study.

The study subjects were categorized into 3 different cerebrovascular diseases based on the CT findings as cerebral infarction, cerebral hemorrhage and subarachnoid hemorrhage. CT scan brain was taken within 24-48 hrs, 12 lead ECG was taken and monitored on the day of admission. The ECG was recorded in all patients and interpreted by specialist consultant with rate, rhythm, ST segments, QRS complex, QT interval, T wave amplitude and morphology. Bizet’s formula was taken consideration for QTc interval calculation.

RESULTS

A detailed analysis of the ECG of all the patients was done. Out of 56 cerebrovascular disease patients, 69.6% of all stroke patients had some form of ECG abnormalities. Abnormal ECG was found in higher cases of cerebral hemorrhage patients (72.7%) than subarachnoid hemorrhage (50%), cerebral infarction (66.6%) cases (Table 1).

Depression of ST segment (32.1%) was observed in higher percentage in cerebral infarction cases. Prolonged QTc interval (26%) was second most common change found in ECG of cerebral infarction patients. ST elevation was most commonly noted in cerebral hemorrhage patients. Prolonged QTc interval was found 37.4% in subarachnoid hemorrhage cases (Table 2).

Present study shows maximum 6 cases of sinus tachycardia in cerebral infarction cases, 5 in cerebral hemorrhage cases and 2 in subarachnoid hemorrhage. Sinus bradycardia was seen in 4 cases of cerebral hemorrhage patients, none of the patients had sinus bradycardia in cerebral infarction and subarachnoid hemorrhage (Table 3.)

Table-1: Incidences of abnormal ECG’s in cerebrovascular diseases

<table>
<thead>
<tr>
<th>Cerebrovascular diseases</th>
<th>No. of Cases</th>
<th>Abnormal ECG</th>
<th>Percentage (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral infarction</td>
<td>30</td>
<td>20</td>
<td>66.6</td>
</tr>
<tr>
<td>Cerebral Hemorrhage</td>
<td>22</td>
<td>16</td>
<td>72.7</td>
</tr>
<tr>
<td>Subarachnoid Hemorrhage</td>
<td>04</td>
<td>02</td>
<td>50</td>
</tr>
<tr>
<td>Total</td>
<td>56</td>
<td>39</td>
<td>69.6</td>
</tr>
</tbody>
</table>

Table-2: Pattern of ECG changes in cerebrovascular disease patients

<table>
<thead>
<tr>
<th>ECG changes</th>
<th>Cerebral infarction (n=30)</th>
<th>Cerebral Hemorrhage (n=22)</th>
<th>Subarachnoid Hemorrhage (n=04)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated ST</td>
<td>4%</td>
<td>40%</td>
<td>32.3%</td>
</tr>
<tr>
<td>Depression of ST</td>
<td>32.1%</td>
<td>11%</td>
<td>1%</td>
</tr>
<tr>
<td>Tall ‘T’ wave</td>
<td>9%</td>
<td>24%</td>
<td>29.3%</td>
</tr>
<tr>
<td>‘T’ wave inversion</td>
<td>21.2%</td>
<td>16%</td>
<td>0</td>
</tr>
<tr>
<td>Prolonged ‘QTc’ Interval</td>
<td>26%</td>
<td>9%</td>
<td>37.4%</td>
</tr>
<tr>
<td>Q Wave</td>
<td>8%</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>U wave</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>
DISCUSSION

Study was conducted in tertiary care hospital, Bangalore to find out the ECG changes in cerebrovascular disease. The cerebrovascular disease was confirmed and classified based on the findings observed in CT scan brain, which was taken within 24-48hrs of admission.

On observation of ECG’s of cerebrovascular disease study subjects, it was found that ST segment (Elevation or Depression) changes were most commonly observed in all patients. Similar findings were also reported by Frentz and Gorsmen [11]. The incidence of ST segment was 36.1% in cerebral infarction patients, 51% in cerebral hemorrhage patients and 33.3% in subarachnoid hemorrhage patients. The study of Lindgren et al, [12] also showed ST segment depression in lateral leads.

Out of 56 patients, prolonged QTc interval was observed in 26%, 9% and 37.4% in cerebral infarction, cerebral hemorrhage and subarachnoid hemorrhage respectively. The findings of our study are in agreement with Arruda and Lacera [13], Keller and Williams [14]. Cruickshant et al. [15], reported tall T waves, short PR interval as common changes in cerebrovascular disease. T wave inversion was observed in 21.2% of patients with intracerebral haemorrhage, 16% patients with cerebral infarction.

The findings of rhythm changes in present study were in correlation with study of M G Myers et al. [16]. Rhythm disturbances in ECG of subarachnoid hemorrhage in the acute phase were in consistent with A. Andreoli and Collegues [17]. The study concluded by Kuroiwa T et al [18], also reported rhythm disturbances in patients with aneurysmal subarachnoid hemorrhage. Computerized tomographic (CT) scan was used to study the characteristics of cerebrovascular diseases. Frontal lobe hemorrhages were associated with ECG abnormalities of QT interval prolongation and abnormal T waves were reported by Yamour B J [19], by using the CT scan. Q wave changes were noted in 8% of patients with cerebral hemorrhage. It is in correlation with study of Kono T [20], and Crop GJ Manning et al. [21]. The findings of present study suggest that the structures related to cardiovascular function are widely distributed within the central nervous system. Therefore, it is learnt that cerebrovascular accident lesions of frontal lobe as well as temporo-parietal lobe and basal ganglia can destroy or irritate such widely spread neurons or pathways regulating the cardiovascular system, which may result in ECG changes.

CONCLUSION

The study summarizes that ECG changes in cerebrovascular disease patients were not associated with any particular site of cerebral lesion. Cerebrovascular disease has significant impact on clinical management, affects cardiac and neurological outcome. The wise knowledge of pattern of ECG changes which are occurring in patients with cerebrovascular accidents is important because, it may lead to erroneous judgment as cardiac dysfunction. It may be helpful for the better treatment and management of patient.

REFERENCES

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8. Algra A, Gates PC, Fox AJ. Side of brain

Table-3: Showing rhythm disturbances in ECG in cerebrovascular disease patients.

<table>
<thead>
<tr>
<th>Cerebrovascular diseases</th>
<th>Sinus Tachycardia</th>
<th>Sinus Bradycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cerebral infarction (n=30)</td>
<td>6(20%)</td>
<td>0</td>
</tr>
<tr>
<td>Cerebral Hemorrhage (n=22)</td>
<td>5(22.7%)</td>
<td>4(18.1%)</td>
</tr>
<tr>
<td>Subarachnoid Hemorrhage (n=04)</td>
<td>2(50%)</td>
<td>0</td>
</tr>
</tbody>
</table>


