Original article :

A Study of Electrocardiographic Changes in Acute Cerebrovascular Accidents: A Prospective Institutional Based Study

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Abstract
Background: Experimental and clinical studies have addressed a neurogenic catecholamine-mediated mechanism of injury or "catecholamine hypothesis" of cardiac dysfunction. The aim of this study to assess the relation of ECG changes in acute cerebrovascular accident to the location of cerebral lesion.
Material & Methods: The observational study was conducted at Department of General Medicine, Santosh Medical College and Hospital, Ghaziabad, Uttar Pradesh (India) on all patients admitted to medical ward with acute cerebrovascular accidents. Study population consisted of 50 patients. ECG was then interpreted with rate, rhythm, ST segment, QRS complex, T wave amplitude and morphology and QT interval was calculated. QTC interval was calculated based on Bazetts formulae.
Results: Present study showed that ST segment changes were most commonly noted after cerebral hemorrhage. 33% of patients with infarction had ST depression. ST elevation was found in 50% of patients with ICH.
Conclusion: It can be concluded that cardiac disturbance were diverse and frequent in the setting of acute neurological injury.

Keywords: ECG, ST Segment, QRS Complex, T Wave, ICH, ST Segment.

INTRODUCTION

The anatomy and physiology of pathways involved in brain-heart interaction have been elucidated in both animal and human studies. The ability to reproduce the arrhythmia by activation of the sympathetic nervous system suggested a neurogenic mechanism.[1,2] The medulla has been described as the principal site of vagal parasympathetic and sympathetic areas involved in cardiac control.[3,4]

In addition both anatomical and physiological evidences implicate the hypothalamus in cardiac control.[5] Electrical stimulation experiments suggests a posteriorly located area of cardiovascular sympathetic control and anterior parasympathetic control region.[6]

Beattie an colleagues first described cardiac arrhythmias after hypothalamic stimulation.[7] Arrhythmias from hypothalamic stimulation were subsequently confirmed in other animal models.[8,9] Areas of cerebral cortex with connection to autonomic nervous can also elicit cardiac response. The autonomic – emotional interaction with cardiovascular function have been linked to central nucleus of amygdale.[10,11]

There is a well-demonstrated, unique, apical-sparing pattern of regional wall motion abnormality that differentiates SAH patients from those with the typical patterns seen in CAD. The most frequently affected segments are the basal and mid-ventricular portions of the anteroseptal and anterior walls and the mid-ventricular portions of

the inferoseptal and anterolateral walls.[12,13] A retrospective study of SAH patients demonstrated reversibility and both global and regional left ventricular dysfunction, most commonly affecting the anterior and anteroseptal walls that do not involve the apex.[14] Younger age and anterior aneurysm position were independent predictors of this pattern.[15] This apical-sparing pattern of left ventricular dysfunction argues against an obstruction or vasospasm of coronary arteries against an obstruction or vasospasm of coronary arteries and provides indirect evidence of neurally mediated mechanism of injury.

Experimental and clinical studies have addressed a neurogenic catecholamine-mediated mechanism of injury or "catecholamine hypothesis" of cardiac dysfunction.[16] The aim of this study to assess the relation of ECG changes in acute cerebrovascular accident to the location of cerebral lesion.

MATERIALS & METHODS

The observational study was conducted at Department of General Medicine, Santosh Medical College and Hospital, Ghaziabad, UP (India) on all patients admitted to medical ward with acute cerebrovascular accidents. Study population consisted of 50 patients.

Inclusion Criteria

- All patients with acute cerebrovascular accidents.

Exclusion Criteria

- Patients with underlying heart diseases.
- Patients on drugs.
- Previously diagnosed patients with electrolyte abnormalities.
- Patient with hepatic or renal diseases.

Methods

All patients with acute cerebrovascular accidents were studied. They were assessed with serum electrolytes, X ray and blood urea and sugar 12 lead ECG was taken and monitored on the day of admission. CT scan was taken within 24-48 hrs.

Screening ECHO was done on all patients with ECG changes. Patients showing cardiomegaly on X ray were excluded from the study.

Patients were categorized based on the CT finding as cerebral infarction, cerebral hemorrhage and sub- arachnoid hemorrhage. ECG was then interpreted with rate, rhythm, ST segment, QRS complex, T wave amplitude and morphology and QT interval was calculated. QTC interval was calculated based on Bazetts formulae.

Table 1: The Incidence of ST Segment Changes in the Study Group

Study group	Total No Cases	ST Segment Elevation	ST Depression Segment	Percentage With ST Segment Changes
Cerebral Infarction	27	1(3%)	9(33. 3%)	10(37%)
Cerebral Hemorrhage	20	10(50%)	2(10%)	12(60%)
Subarachnoid Hemorrhage	3	1(33. 3%)	0	1(33. 3%)

Table 2: Location and Type of Cerebro Vascular Lesion

Cerebral Lesion	Total No of Patients	No of Patients with Hemorrhage	No on Patients with Infarct
Basal Ganglia	1	NIL	1
Thalamus	5	2	3
Capsuloganglion	31	13	18
Frontal	3	1	2
Temporo-Parietal	7	3	4
Parietal	2	2	0
Occipital	1	1	0

Table 3: Percent and No of Patients with Specific ECG Changes

Cerebral Lesion	Rhythm Disturbance	ST Segment Changes	QTC Prolongation	T Wave Changes
Basal Ganglia	1(100)	1(100)	1(100)	NIL
Thalamus	2(40)	1(20)	2(40)	2(40)
Capsuloganglion	6(31)	11(36)	6(31)	11(36)
Frontal	2(66)	1(33)	2(66)	2(66)
Parietal	2(100)	1(50)	NIL	NIL
Temporo-Parietal	2(28)	2(28)	2(28)	1(14)
Occipital	1(100)	NIL	NIL	NIL

RESULTS

Present study showed that ST segment changes were most commonly noted after cerebral hemorrhage. 33% of patients with infarction had ST depression. ST elevation was found in 50% of patients with ICH (table 1).

DISCUSSION

The most common abnormality noted was ST segment changes in patient with cerebral hemorrhage. 60 percent of patients had the above changes. Of which 50% had ST segment elevation and 10% had ST segment depression. This findings consistent with study of Frentz and Gorsmen[17] who reported an incidence of 71 % with ICH and 15% with infarction and also study of Lindgren et al who showed ST segment depression in lateral leads.

QT_C prolongation was the next common abnormality noted in our study. 50 percent of patients with intracerebral hemorrhage had QTc prolongation.

This is consistent with study of Arruda and Lacerda[18] which showed 67% of patients with ICH and also study of Keller and Williams19 in patients with stroke.

Regarding the relationship between the locations of CVA lesions and ECG abnormalities, Frentz and Gormsen, and Kreus et al.[17] briefly noted that ECG changes appeared to bear no relationship to arteriographic findings.

Recently, however Yamour Et Al.[20], using the computerized tomographic (CT) scan, suggested that frontal lobe hemorrhages were associated especially with the ECG abnormalities of corrected QT interval (QTC) prolongation and neurogenic T

waves. However in our study there no specific correlation of ECG changes with site of cerebral lesion.

All these patients with ECG abnormalities, a screening echo were performed to rule out cardiac abnormalities associated. Out of 27 patients with cerebral infarction only 3 had regional wall motion abnormalities. 5 patients out of 20 patients with ICH had regional wall motion abnormalities.

These were predominantly involving the basal and midventricular portion of anteroseptal and anterior walls and ventricular portion of inferoseptal and antero lateral walls differentiating from the CAD which involved apical portion.

In present study; author also tried to follow up these patients to see the reversal of the ECG changes. Author could only follow 10 patients, whose ECG changes were completely reverted. Other 40 patient didn't turn up for follow up study.

All the patients (100%) with SAH had ECG abnormalities. Among the 3 patients noted in present study 1 had berry aneurysm for which clipping was done. Rest of the 2 patients died during hospital stay.

Sites which contribute to regulation of the cardiovascular function are known to be the anterior half of the cerebral cortex which includes the top of the frontal lobe, the motor and premotor cortex, and anterior part of the temporal lobe, hypothalamus, the limbic system, and the cerebellar hemisphere. The intimate functional connections between the hypothalamus, and posterior orbital and anterior insula and those between the hypothalamus and peripheral sympathetic nerves have also been demonstrated.

These findings suggest that the structures related to cardiovascular function are widely distributed within the central nervous system. Therefore, it is likely that CVA lesions not only in the frontal lobe, but also in the temporo-parietal lobe and basal ganglia can destroy or irritate such widely spread neurons or pathways regulating the cardiovascular system, resulting in ECG changes.

CONCLUSION

It can be concluded that cardiac disturbance were diverse and frequent in the setting of acute neurological injury. More importantly the presence of cardiac abnormalities has significant impact on clinical management and affects cardiac and neurological outcome.

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