Scholars Journal of Applied Medical Sciences (SJAMS)	ISSN 2320-6691 (Online)
Abbreviated Key Title: Sch. J. App. Med. Sci.	ISSN 2347-954X (Print)
©Scholars Academic and Scientific Publisher	
A Unit of Scholars Academic and Scientific Society, India www.saspublishers.com	Physiology

Study of Incidence and Pattern of Electrocardiographic Changes in Cerebrovascular Diseases

Dr. Naveen P^{*}, Dr. Benjamin Lalrinpuia, Dr. Lalhruaia Ralte

Department of Physiology, Mizoram Institute of Medical Education & Research, Falkawn - 796005, Aizawl, Mizoram, India



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

Cardiac abnormalities were described with various CNS diseases including seizures, trauma, ischemic stroke, ICH. Lower incidences of cardiac abnormalities mav 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].

INTRODUCTION

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 after 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. Incluences of abilitimal ECO 3 in cerebiovascular diseases				
Cerebrovascular diseases	No. of Cases	Abnormal ECG	Percentage (%)	
Cerebral infarction	30	20	66.6	
Cerebral Hemorrhage	22	16	72.7	
Subarachnoid Hemorrhage	04	02	50	
Total	56	39	69.6	

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

Table-2. Tattern of ECG changes in cerebiovascular disease patients					
ECG changes	Cerebral infarction	Cerebral Hemorrhage	Subarachnoid		
	(n=30)	(n=22)	Hemorrhage (n=04)		
Elevated ST	4%	40%	32.3%		
Depression of ST	32.1%	11%	1%		
Tall 'T' wave	9%	24%	29.3%		
'T' wave inversion	21.2%	16%	0		
Prolonged 'QTc"	26%	9%	37.4%		
Interval					
Q Wave	8%	0	0		
U wave	0	0	0		

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

Table-3: Showing rhythm disturbances in ECG in cerebrovascular disease patients.					
	Cerebrovascular diseases	Sinus Tachycardia	Sinus Bradycardia		
	Cerebral infarction				
	(n=30)	6(20%)	0		
	Cerebral Hemorrhage				
	(n=22)	5(22.7%)	4(18.1%)		
	Subarachnoid Hemorrhage				
	(n=04)	2(50%)	0		

Naveen P et al., Sch. J. App. Med. Sci., Jun 2018; 6(6): 2432-2435

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 Lacerda [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

- 1. Donaldson JW, Pritz MB. Myocardial stunning secondary to aneurysmal subarachnoid hemorrhage. Surg Neurol. 2001; 12(3):55-61.
- Kreus KE, Kemilä SJ, Takala JK. Electrocardiographic changes in cerebrovascular accidents. Journal of Internal Medicine. 1969 Jan 12;185(1-6):327-34.
- Ako J, Sudhir K, Farougue HM. Transient left ventricular dysfunction under severe stress: brainheart relationship revisted. Am J Med. 2006; 6(2):119-10.
- 4. Bybee KA, Kara T, Prasad A, Lerman A, Barsness GW, Wright RS, Rihal CS. Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. Annals of internal medicine. 2004 Dec 7;141(11):858-65.
- 5. Levy A. The exciting causes of ventricular fibrillation in animals under chloroform anaesthesia. American Heart Journal, 1980: 64(2); 393-98.
- Wittstein IS, Thiemann DR, Lima JA. Neurohumoral features of myocardial stunning due to sudden emotional stress. N Engl J Med. 2005; 35(2):539.
- Lane RD, Wallace JD, Petrosky PP. Supraventricular tachycardia in patients with right hemisphere strokes, Journal of Medicine.1992; 2(3):362.
- 8. Algra A, Gates PC, Fox AJ. Side of brain

Naveen P et al., Sch. J. App. Med. Sci., Jun 2018; 6(6): 2432-2435

infarction and long-term risk of sudden death in patients with symptomatic carotid disease. Clin Auton Res. 2003; 13(2):2871-79.

- 9. Oppenheimer S. Cerebrogenic cardiac arrhythmias:: Cortical lateralization and clinical significance. Clinical Autonomic Research. 2006 Feb;16(1):6.
- 10. Kono T, Morita H, Kuroiwa T, Onaka H, Takatsuka H, Fujiwara A. Left ventricular wall motion abnormalities in patients with subarachnoid hemorrhage: neurogenic stunned myocardium. Journal of the American College of Cardiology. 1994 Sep 1;24(3):636-40.
- 11. Fentz V, Gormsen J. Electrocardiographic pattern in patients with cerebrovascular accidents. Circulation.1962; 25:21.
- Lindgren A, Wohlfart B, Pahlm O, Johansson BB. A Study of ECG changes in stroke patients without primary heart disease. Department of Neurology, University Hospital, Lund, Sweden.1994;12(8):32-41.
- 13. Arrudo and Lacerda. ECG findings in acute cerebrovascular hemorrhage-Arq neuropsychiatry.1992;50(3):269-74.
- 14. Keller and Williams. Cardiac dysarrhythmias associated with CNS dysfunction; Journal of Neuroscience.1993; 25(2):349-55.
- 15. Cruickshnok, Neil Dcoyer and J. Price. ECG changes and their prognostic significance in CVA –Journal of Neurology. 1974; 3(7);735-759.
- Myers MG, Norris JW, Hachinski VC, Weingert ME, Sole MJ. Cardiac sequelae of acute stroke. Stroke. 1982 Nov 1;13(6):838-42.
- 17. A. Andreoli, G di Pasquale, G Pinelli, P Grazi, F Tognetti and C Testa. The study of frequency and severity of cardiac arrhythmias in 70 cases with Subarachnoid haemorrhage in the acute phase. An Article published in journal of American Heart Association.1987;94(11):574-584.
- Kuroiwa T, Morita H, Tanabe H, Ohta T. Significance of ST segment elevation in electrocardiograms in patients with ruptured cerebral aneurysms. Acta neurochirurgica. 1995 Sep 1;133(3-4):141-6.
- Yamour BJ, Sridharan MR, Rice JF, Flowers NC. Electrocardiographic changes in cerebrovascular hemorrhage. American heart journal. 1980 Mar 1;99(3):294-300.
- 20. Kono T, Morita H, Kuroiwa T, Onaka H, Takatsuka H, Fujiwara A. Left ventricular wall motion abnormalities in patients with subarachnoid hemorrhage: neurogenic stunned myocardium. Journal of the American College of Cardiology. 1994 Sep 1;24(3):636-40.
- 21. Cropp GJ, Manning GW. Electrocardiographic changes simulating myocardial ischemia and infarction associated with spontaneous intracranial hemorrhage. Circulation. 1960 Jul 1;22(1):25-38.

Corresponding Author:

Dr. Naveen. P,

Associate Professor,

Department of Physiology,

Mizoram Institute of Medical Education & Research,

Falkawn - 796005,

Aizawl, Mizoram, India.

Mobile: 9620090631,

Email:naveenphysiol@gmail.com