Section: Medicine

Research

A PROSPECTIVE STUDY OF CARDIAC COMPLICATIONS IN ACUTE ISCHAEMIC STROKE WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION

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Abstract

Background: Acute central nervous system injury has long been associated with myocardial injury and dysfunction. Some have implicated serum catecholamine elevations in particular with strokes involving the insular cortex, but the mechanisms underlying this relation are poorly understood in acute ischemic stroke (AIS). **Materials and Methods:** After obtaining informed consent, age, height, weight, risk factors and vital parameters were recorded in all patients. Complete physical examination followed by laboratory investigations were done. Investigations included haematocrit, haemoglobin, total and differential WBC count, platelet count, blood sugar, serum urea, serum creatinine, serum electrolytes, lipid profile, liver function tests, X-ray chest and CT-brain (Plain). ECG and Echocardiogram done at admission and at 48 hours. ECG was noted for ischaemic changes and arrhythmias, and ECHO for EF and LVDD. Using NIH stroke scale, severity of the stroke assessment done. Data was analysed using SPSS package and Chi-square test. **Result:** Majority of the population in the study fall in 51-70 years' age group. There was no significant difference in the stroke severity based on gender. In our study group hypertension was seen in 57%, diabetes mellitus in 31%, hyperlipidaemia in 20% and alcohol consumption in 13% of the patients. Arrhythmias were observed in 7% of the patients at admission. The number increased to 12% at 48 hours, although not statistically significant. **Conclusion:** A subset of acute ischaemic stroke patients may have cardiac complications. Systolic dysfunction, atrial fibrillation and ischaemic changes on ECG may be associated with higher in-hospital mortality rate as indirectly evidenced by the significant correlation of cardiac complications with severity of stroke. These findings support the importance of the adjunctive role of cardiac monitoring strategies in acute ischaemic stroke.

INTRODUCTION

Acute central nervous system injury has long been associated with myocardial injury and dysfunction. Some have implicated serum catecholamine elevations in particular with strokes involving the insular cortex, but the mechanisms underlying this relation are poorly understood in acute ischemic stroke (AIS).[1] Cardiac dysfunction in stroke patients may be particularly damaging. Within the territory of tissue affected by an AIS (ie, the ischemic penumbra) intrinsic autoregulation of the vasculature is lost, rendering cerebral blood flow directly dependent on cardiac function.[2]

Any acute insult to the central nervous system has been known to cause a wide array of manifestations in the cardiovascular system. This can include asymptomatic ST-T changes, fatal or non-fatal arrhythmias, ventricular dysfunction, or cardiac dysautonomias.[3] Increase in the levels of serum catecholamines following a stroke, has been thought to play a role but the intricate mechanisms involved is still an enigma. The intrinsic auto regulation of blood flow is impaired in the ischemic penumbra, making the cerebral perfusion mainly dependent on cardiac function. Hence, cardiac dysfunction can lead to detrimental effects in acute stroke patients.[4] This study is designed to augment the existing studies on cardiac dysfunction in acute

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ischemic/thrombotic stroke. It has been hypothesized that a considerable proportion of moderate to severe acute ischemic/thrombotic stroke have systolic and diastolic ventricular dysfunction. Alternative manifestations like arrhythmias and ST-T changes were also included. This study intends to correlate ventricular dysfunction (systolic and diastolic) with severity of acute ischemic/thrombotic stroke based on NIHSS.

MATERIALS AND METHODS

Study Design: A prospective study.
Study Location: Malabar Medical college, Modakalur, Kozhikode.
Study Duration: May 2021 to April 2022
Sample Size: 200 patients

Inclusion Criteria
Patients presenting within 24 hours of symptom onset and diagnosed with acute ischaemic/thrombotic stroke.

Exclusion Criteria
Age younger than 18 years. Evidence of cerebral haemorrhage on initial head CT. Resolution of neurologic symptoms within 24 hours. Presence of documented chest pain. It was an observational study.
Study was conducted on 200 consecutive patients admitted with acute ischaemic/thrombotic stroke over 1 year duration.

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RESULTS

Majority of the population in the study fall in 51-70 years' age group. There was no significant difference in the stroke severity based on gender. In our study group hypertension was seen in 57%, diabetes mellitus in 31%, hyperlipidaemia in 20% and alcohol consumption in 13% of the patients. Arrhythmias were observed in 7% of the patients at admission. The number increased to 12% at 48 hours, although not statistically significant.
Among the 24 patients who had rhythm disturbances, 22 (92%) had atrial fibrillation and 2 (8%) developed ventricular tachycardia; 14% had ischaemic ST-T changes at admission which increased to 20% at 48 hours.

Table 1: Severity of LV Systolic Dysfunction (At Admission) among Stroke Severity Groups

Ejection fraction (%)		NIHSS				P value
		5-15	16-20	>20	Total	0.001
	>50	80	30	20	130	
	41-50	12	16	18	46	
	<40	2	2	20	24	
					200	

Table 2: Severity of LV Diastolic Dysfunction (At Admission) among Stroke Severity Groups

Ejection fraction (%)		NIHSS				P value
		5-15	16-20	>20	Total	10.001
	>50	74	28	20	122	
	41-50	14	8	8	30	
	<40	6	12	30	48	
					200	

Table 3: Severity of LV Systolic Dysfunction (At 48 hours) among Stroke Severity Groups

LVDD Grade		NIHSS				P value
		5-15	16-20	>20	Total	0.001
	Normal	68	30	16	114	
	1	20	16	16	52	
	2	6	2	20	28	
	3	0	0	6	6	
					200	

Table 4: Severity of LV Systolic Dysfunction (At 48 hours) among Stroke Severity Groups

LVDD Grade		NIHSS				P value
		5-15	16-20	>20	Total	0.001
	Normal	66	26	16	108	
	1	22	16	10	48	
	2	4	6	16	26	
	3	2	0	16	18	
					200	

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DISCUSSION

Many studies had been made in this context. Yet, the studies varied in many aspects and laid importance on one aspect out of the many complex manifestations involved with cardiovascular and cerebrovascular systems.[5] A number of studies in the past have concentrated upon the varied ECG manifestations occurring in acute CVA including both ischaemic and haemorrhagic. But whether similar effects cause significant damage in ischaemic/thrombotic stroke has been less studied. The insular cortex had been studied and found to cause cardiac sympathetic neural upregulation and ECG abnormalities.[6] A number of neurosurgical studies have shown that ECG abnormalities and left ventricular dysfunction (Wall motion hypokinesias) can occur in haemorrhagic stroke, especially SAH. Also described are myocardial stunning and myocardial necrosis. Other findings were increased levels of natriuretic factors, catecholamines in the plasma. Myocardial perfusion too gets affected regionally. When an acute ischaemic/thrombotic stroke happens in any patient with underlying heart disease, the damage is severe. The autoregulation of blood flow is lost in the ischaemic penumbra as the main factor which determines it, 'the cardiac function' is under stake.[7]

In our study the results are similar to that available in the literature, which suggests that the incidence of systolic dysfunction in acute ischaemic/thrombotic stroke patients can range from 14% to 30%.

Some studies also throw light on the ischaemic changes in ECG which claims a range of 36% to 74% in haemorrhagic strokes. 'Oppenheimer et al' in their study observed an incidence of 15- 20% after ischaemic stroke. In our study, 40 patients had ischaemic changes in the ECG. The incidence goes in hand with the literature and also 26 out of 40 had a NIHSS >10. These changes occur due to a neural mechanism and not associated CAD.[8,9]

48 patients had an EF 20 and 21 (87.5%) of them had a NIHSS >15, which is highly statistically significant ($p<0.001$). The incidence of 24% found in our study is similar to that quoted in other studies in this domain. A similar study by 'Wira et al' observed a systolic dysfunction of 28.5%. In our study, the study design did not include a control group nor was echocardiography repeated in the follow-up period in all the patients.[10]

CONCLUSION

A subset of patients who suffer acute ischaemic/thrombotic stroke develop an array of cardiac complications. These cardiac manifestations are either caused directly by the neural effects or other reasons not within the scope of this study. Active atrial fibrillation, ischaemic changes in the ECG, left ventricular systolic and diastolic dysfunction are associated more with moderate-to-severe and severe stroke groups. Thus, this subset of patients may be associated with higher in hospital mortality rates and poor outcomes. This study emphasizes the role of cardiac monitoring in the acute stroke setting. Further studies can throw light on cardiac augmentation strategies, which could be adopted in the management protocol of acute ischaemic/thrombotic stroke patients.

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