CARDIAC COMPLICATIONS IN ACUTE ISCHAEMIC STROKE WITH SPECIAL REFERENCE TO VENTRICULAR DYSFUNCTION

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ABSTRACT

To characterize cardiac complications occurring in acute ischaemic/thrombotic stroke patients admitted to the medical emergency at a tertiary care hospital in Chennai, South India. Many extensive studies have been made in the past regarding cardiac complications in haemorrhagic stroke, especially SAH.

METHODS

Observational study (Prospective cum retrospective) done in acute ischaemic stroke patients admitted within 24 hours of symptom onset. Patients with haemorrhage in CT, age less than 18 and TIA were excluded. Electrocardiogram and Echocardiography were performed at admission and at the end of 48 hours.

RESULTS

Hundred patients were included in this study. In our study group 39 patients had an ejection fraction less than 50%, 20 patients had ischaemic changes in the ECG, 11 patients presented with atrial fibrillation and one developed a ventricular tachycardia. Subgroup analysis revealed a higher NIHSS score among those with systolic dysfunction with ejection fraction less than 40% (10% versus 2%; $p<0.001$), atrial fibrillation on ECG (9% versus 3%; $p<0.05$), ischaemic changes on ECG (17% versus 3%; $p<0.05$) compared with those without these changes.

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.

KEYWORDS

Acute Ischaemic Stroke, Cardiac Complications, Systolic Dysfunction, Atrial Fibrillation, Electrocardiogram.

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INTRODUCTION

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.[1-3] Increase in the levels of serum catecholamines following a stroke.[4-6] has been thought to play a role, but the intricate mechanisms involved is still an enigma. The intrinsic autoregulation of blood flow is impaired in the ischaemic penumbra, making the cerebral perfusion mainly dependent on cardiac function.[7-9] Hence, cardiac dysfunction can lead to detrimental effects in acute stroke patients. This phenomenon has been well studied in SAH by earlier investigators. But whether similar effects cause significant damage in ischaemic/thrombotic stroke has been less studied.[10] Inpatients of acute ischaemic stroke undergo echocardiograms to look for a cardioembolic source, but there are no recommendations pertaining to management of systolic or diastolic dysfunction in acute ischaemic/thrombotic stroke. Early identification of these cardiac manifestations might prove valuable in defining a role in the management strategies like cardiac augmentation in acute ischaemic/thrombotic stroke patients. It had been always the heart to which attention was paid in cases of stroke, either as a source of embolism or cause of hypoperfusion. Recent turn is in the emphasis towards the mechanisms involving the brain injury as a cause for cardiac dysfunctions.[11] The insular cortex had been studied and found to cause cardiac sympathetic neural upregulation and ECG abnormalities.[12-15] Thus cardiac mortality is increased and can become a major cause of death in acute strokes. This study is designed to augment the existing studies on cardiac dysfunction in acute ischaemic/thrombotic stroke. It has been hypothesized that a considerable proportion of moderate-to-severe acute ischaemic/thrombotic stroke have systolic and diastolic ventricular dysfunction. Alternative manifestations like.[13] arrhythmias and ST-T changes were also included. This study intends to correlate ventricular dysfunction (Systolic and diastolic) with severity of acute ischaemic/thrombotic stroke based on NIHSS.

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AIMS AND OBJECTIVES

1. To assess the prevalence of cardiac complications in acute ischaemic/thrombotic stroke patients with special reference to ventricular dysfunction.

2. To correlate the cardiac manifestations with the severity of stroke based on NIH stroke scale.

MATERIALS AND METHODS

After getting the approval from the Ethical Committee, study was conducted on 100 consecutive patients admitted with acute ischaemic/thrombotic stroke over 6 months' duration.

Inclusion Criteria

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

Exclusion Criteria

1. Age younger than 18 years.
2. Evidence of cerebral haemorrhage on initial head CT.
3. Resolution of neurologic symptoms within 24 hours.
4. Presence of documented chest pain.
5. It was an observational study.

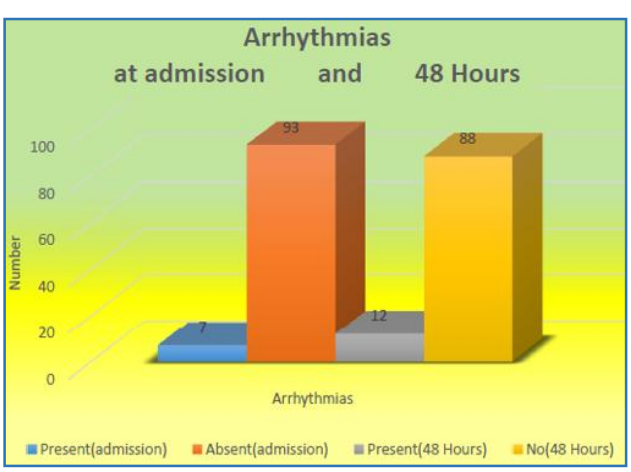
Data Collection

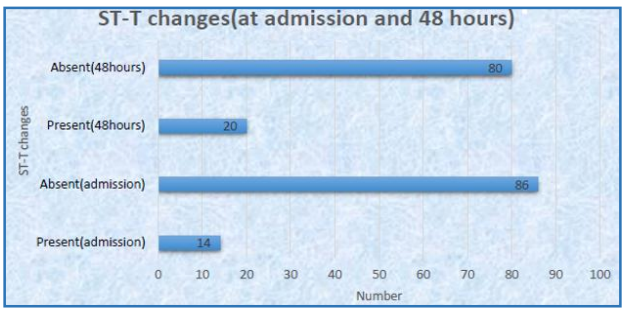
After obtaining informed consent, age, height, weight, risk factors and vital parameters were recorded in all patients. Complete physical examination followed by laboratory investigations were done. Investigations included haematocrit, haemoglobin, total and differential WBC count, platelet count, blood 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.

OBSERVATION AND RESULTS

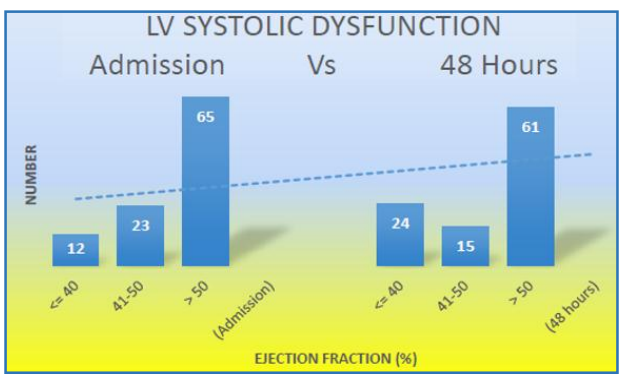
Majority of the population in the study fall in 51-70 years' age group. There was no significant difference in the stroke severity based on gender. In our study group hypertension was seen in 57%, diabetes mellitus in 31%, hyperlipidaemia in 20% and alcohol consumption in 13% of the patients. Arrhythmias were observed in 7% of the patients at admission. The number increased to 12% at 48 hours, although not statistically significant.

Among the 12 patients who had rhythm disturbances, 11 (92%) had atrial fibrillation and 1 (8%) developed ventricular tachycardia; 14% had ischaemic ST-T changes at admission which increased to 20% at 48 hours.

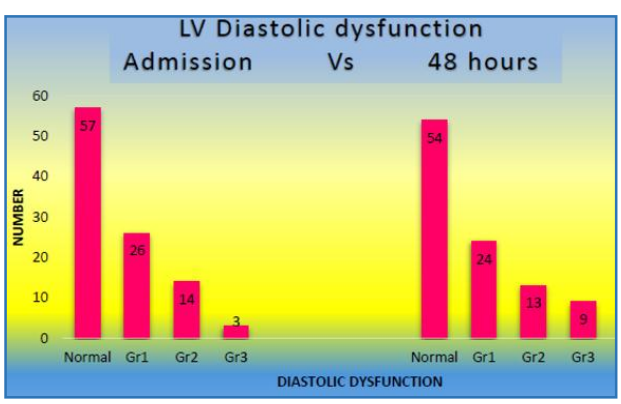




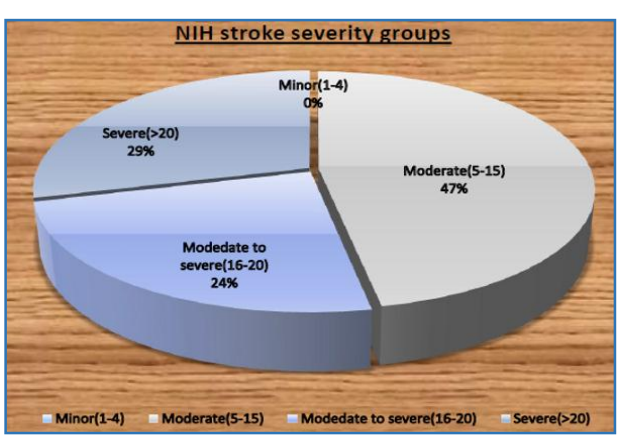
In our study, 12% had EF less than 40% at admission and this number doubled to 24% at 48 hours ECHO. EF 41-50% was observed in 23% at admission, but only 15% was observed at 48 hours. More than 60% showed normal LV systolic function based on EF at admission and at 48 hours.



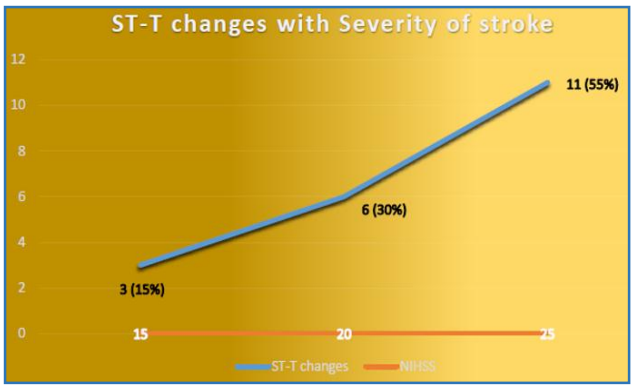
Grade 3 LV diastolic dysfunction was observed in 3% at admission, and this number tripled to 9% at 48 hours. Grade 1 LV diastolic dysfunction was noted in 25% of the patients; 50% had no LV diastolic dysfunction.



In our study 47% patients had a moderate stroke, 24% had a moderate-to-severe stroke and 29% had a severe stroke.



In our study, 20% had ST-T changes at the end of 48 hours and among them more than half (55%) belonged to the severe stroke group. Six patients (30%) belonged to the moderate-to-severe stroke group.

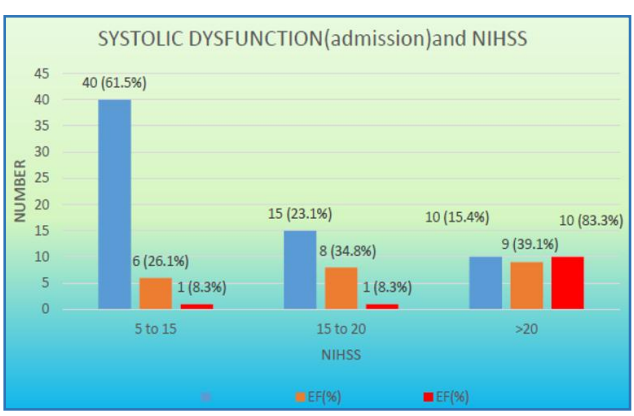


Severity of LV Systolic Dysfunction (At Admission) among Stroke Severity Groups

		NIHSS				P Value
Ejection Fraction (%)		5-15	16-20	>20	Total	
	>50	40	15	10	65	
	41-50	6	8	9	23	
	<40	1	1	10	12	<0.001**
					100	

**- highly significant at 1 level.

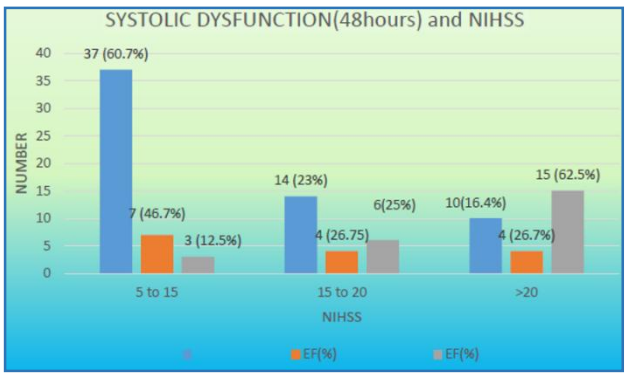
In the systolic dysfunction group, twelve patients had an EF of less than 40% and ten (83.3%) amongst them belonged to the severe stroke group which is statistically highly significant. P value obtained was <0.001. Twenty three patients had mild LV systolic dysfunction and the proportion of them in severe stroke group was more though not statistically significant. Sixty patients had normal LV systolic function and forty (61.5%) among them belonged to the moderate stroke group and this difference among severity groups is statistically highly significant. In our study, a total of thirty five patients had some degree of systolic LV dysfunction during admission.



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

		NIHSS				P Value
Ejection Fraction (%)		5-15	16-20	>20	Total	
	>50	37	14	10	61	
	41-50	7	4	4	15	<0.001**
	<40	3	6	15	24	
					100	

**- highly significant at 1 level.

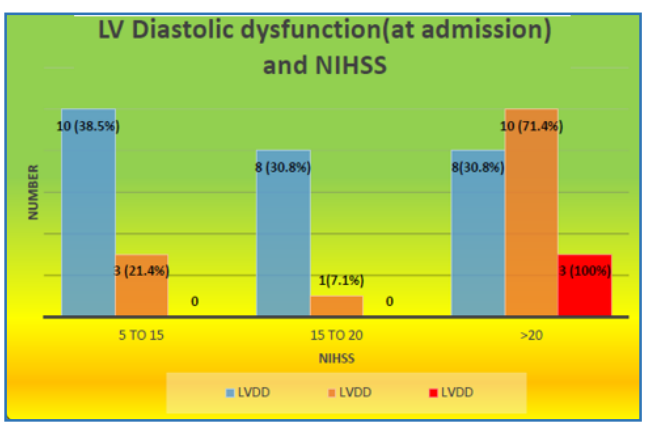


In our study, a total of thirty nine patients had some degree of systolic LV dysfunction at 48 hours. In the systolic dysfunction group at 48 hours, 24 patients had an EF of less than 40% and fifteen (62.5%) amongst them belonged to the severe stroke group which is statistically highly significant. P value obtained was <0.001. Sixty one patients had normal LV systolic function and thirty seven (60.7%) among them belonged to the moderate stroke group and this difference among stroke severity groups is statistically highly significant.

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

		NIHSS				P Value
LVDD Grade		5-15	16-20	>20	Total	
	Normal	34	15	8	57	
	1	10	8	8	26	
	2	3	1	10	14	<0.001**
	3	0	0	3	3	
					100	

**- highly significant at 1 level.



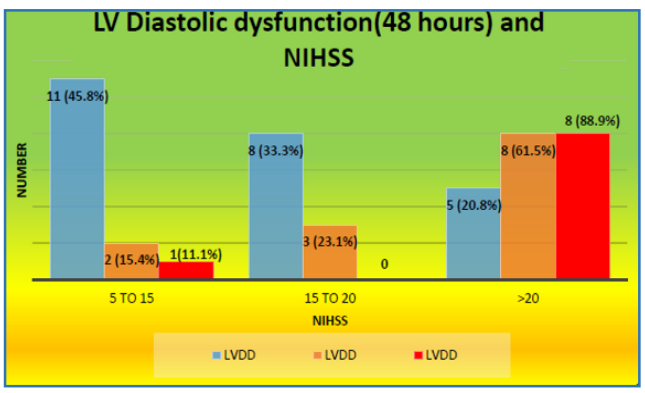
In our study, a total of forty three patients had some degree of LV diastolic dysfunction during admission. In the LVDD group at admission, three patients had Grade 3 LVDD and all 3 (100%) of them belonged to the severe stroke group which is statistically highly significant. P value obtained was <0.001. Fourteen patients had Grade 2 LVDD and 10 of them (71.4%) belonged to severe stroke group, which is statistically highly significant (p<0.001).

Fifty seven patients had normal LV diastolic function and 34 (59.6%) of them belonged to the moderate stroke group and this difference among severity groups is statistically highly significant.

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

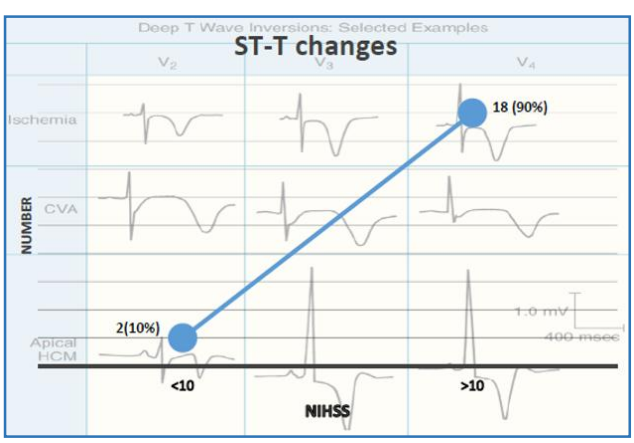
LVDD Grade		NIHSS				P Value
		5-15	16-20	>20	**Total**	
	Normal	33	13	8	**54**	<0.001**
	1	11	8	5	**24**	
	2	2	3	8	**13**	
	3	1	0	8	**9**	
					100	

**- highly significant at 1 level.

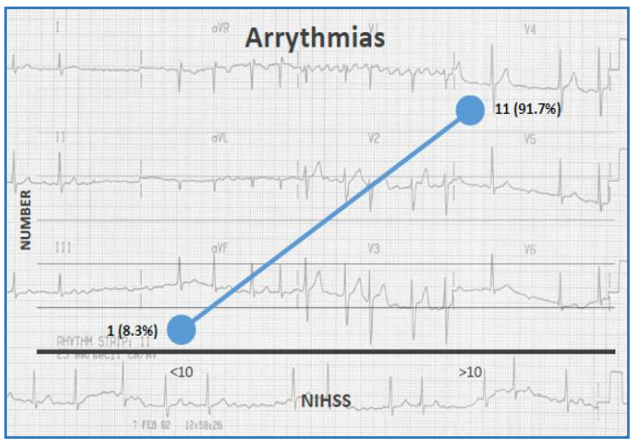


In our study, a total of forty four patients had some degree of LV diastolic dysfunction at 48 hours. In the LVDD group at 48 hours, nine patients had Grade 3 LVDD and 8 (88.9%) of them belonged to the severe stroke group which is statistically highly significant. P value obtained was <0.001. Thirteen patients had Grade 2 LVDD and 10 of them (61.5%) belonged to severe stroke group, which is statistically highly significant (p<0.001). Fifty four patients had normal LV diastolic function and 33 (61.1%) of them belonged to the moderate stroke group and this difference among severity groups is statistically highly significant.

In our study, twenty patients had ST-T changes in their ECG at the end of 48 hours and among them 18 (90%) patients belonged to the NIHSS >10 group.

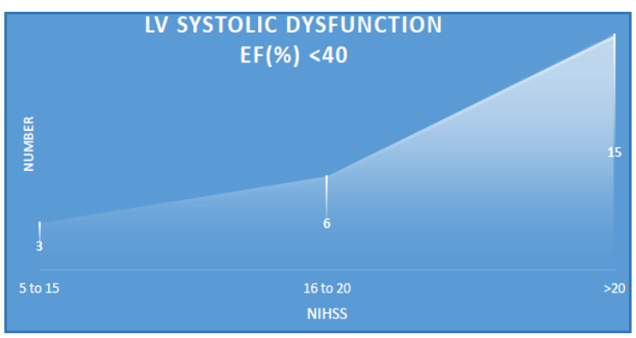


In our study, twelve patients had ST-T changes in their ECG at the end of 48 hours and among them 11 (91.7%) patients belonged to the NIHSS >10 group.

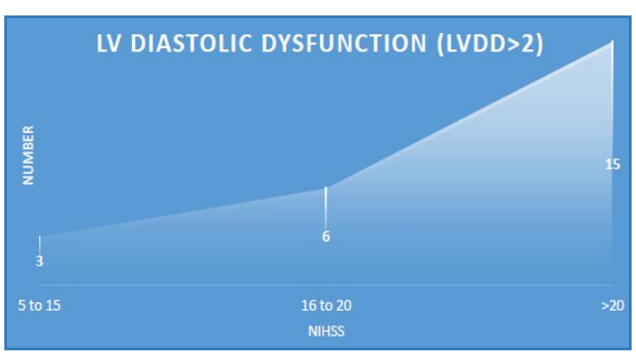


In our study, 39 patients had some degree of LVSD in the echo at the end of 48 hours and among them 34 (87.2%) patients belonged to the NIHSS >10 group.

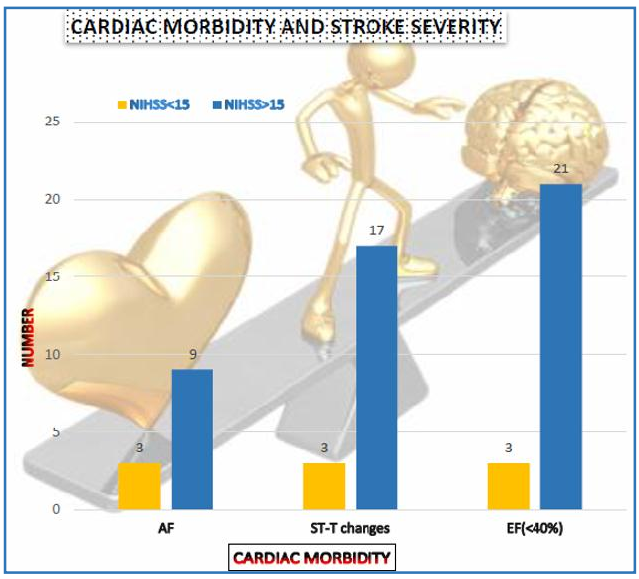
This graph shows the rising trend of the moderate-to-severe LVSD curve across the stroke severity group; 15 patients (62.5%) fall in the severe stroke group. The difference in distribution is statistically highly significant (p<0.001).



This graph shows the rising trend of the Grade 2-3 LVDD curve across the stroke severity group; 8 patients (88.9%) fall in the severe stroke group. The difference in distribution is statistically significant (p<0.050).



The graph below summarizes the incidence of cardiac complications in acute ischaemic stroke. The number of patients with AF, ST-T changes and moderate-to-severe LV dysfunction are divided with an arbitrary median of NHISS 15; 81.1% of AF, 85% of ST-T changes, 87.5% of significant LV systolic dysfunction were found in the 'moderate-to-severe' and 'severe' stroke groups (NIHSS>15).



DISCUSSION

There had been a constant emphasis on the complex relationship between cerebrovascular accidents and cardiovascular diseases. 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. 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. A number of neurosurgical studies have shown that ECG abnormalities and left ventricular dysfunction (Wall motion hypokinesias) can occur in haemorrhagic stroke, especially SAH.[14-17] 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.[16-17] When an acute ischaemic/thrombotic stroke happens in any patient with underlying heart disease, the damage is severe.[18-19]

The autoregulation of blood flow is lost in the ischaemic penumbra as the main factor which determines it, 'the cardiac function' is under stake.[20] 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%.

ST-T Changes

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, 20 patients had ischaemic changes in the ECG. The incidence goes in hand with the literature and also 18 out of 20 had a NIHSS >10. These changes occur due to a neural mechanism and not associated CAD.

Systolic Dysfunction

24 patients had an EF <40 at 48 hours and 15 (62.5%) of them had a NIHSS >20 and 21 (87.5%) of them had a NIHSS >15, which is highly statistically significant ($p<0.010$). 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. Hence, whether the systolic dysfunction is a transient one or a pre-existing one could not be ascertained. Yet from the demographics of our study, it is found that only 9 patients had pre-existing heart failure. So, the possibility of a temporal association of the systolic dysfunction and acute ischaemic stroke is definitely feasible.[21-23] Nevertheless, from our study we found that systolic dysfunction correlated well with the NIHSS severity as similar correlations with mortality were observed in other studies.[24]

Arrhythmias

Eleven patients in our study had atrial fibrillation. One patient had a VT. About atrial fibrillation, there is only one study.[25] which proposes AF as a predictor of mortality. In our study, 11 out of 12 patients had an NIHSS >10 which is an indirect predictor of mortality. The exact mechanisms of how mortality is increased is poorly understood. An NIHSS of 10 is chosen as a comparing destination point, because few studies in the literature have found that the excellent prognosis rate decreases significantly when the NIHSS is more than 10. All these changes are associated with increased mortality, which was reflected in our study by the severity of NIHSS. Death was not taken as an outcome in our study. A study of stroke patients had shown that left insular cortical lesions are more likely to produce ECG abnormalities and cardiac sympathetic neural upregulation. Such changes can lead to increased cardiac mortality, which can be a major cause of mortality in stroke.[26-28]

LIMITATIONS

Our study is limited by its sample size, which is small. The study design did not include a control group nor was echocardiography repeated in the follow-up period in all the patients. Hence, whether the systolic dysfunction is a transient one or a pre-existing one could not be ascertained. Higher rates of systolic dysfunction were not compared with age matched controls.

Yet from the demographics of our study, it is found that only 9 patients had pre-existing heart failure. So, the possibility of a temporal association of the systolic dysfunction and acute ischaemic stroke is definitely feasible. Nevertheless, from our study we found that systolic dysfunction correlated well with the NIHSS severity as similar correlations with mortality were observed in other studies. Yet, this is a considerable finding warranting further studies. Another limitation is that, in our study we did not control for other factors affecting LV function and any other previous medications which would affect the LV function during the time of performing the echocardiography.

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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