LEFT VENTRICULAR HYPERTROPHY IN ISCHEMIC STROKE PATIENTS SINGLE CENTER EXPERIENCE

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ABSTRACT

OBJECTIVES

To ascertain the prevalence of left ventricular hypertrophy in individuals diagnosed with ischemic stroke.

METHODOLOGY

A cross-sectional study was undertaken in the Department of Medicine, Hayatabad Medical Complex, Peshawar, from October 1, 2020, to April 30, 2021, after obtaining ethical approval. The study included 143 individuals who had evidence of ischemic stroke on a CT scan. These patients were assessed for the presence or absence of left ventricular hypertrophy using transthoracic echocardiography.

RESULTS

The average age of the participants in the study was 57 years, with a standard deviation of 7.9 years. The sample consisted of 67.8% males and 32.2% females. The average BMI was calculated to be 25.3 + 3.3kg/m2, and 39.9% of the patients were from urban areas. Left ventricular hypertrophy was observed in 16.8% of patients who had experienced an ischemic stroke, with the greatest occurrence rate of 39.9% found in the age range of 50-60 years. There was no significant correlation observed between hypertension, diabetes, location of residence, age, and smoking.

CONCLUSION

Early and precise identification of the modifiable risk factors during the illness, followed by proper intervention, has the potential to decrease the incidence of debilitating stroke greatly. It is necessary to conduct large-scale population screening studies inside our local community. TTE should be carried out in all acute ischemic stroke patients to optimize the management of these patients.

KEYWORDS: *Ischemic Strokes, left ventricular hypertrophy, Transthoracic Echocardiography*

INTRODUCTION

Stroke continues to be a significant contributor to illness and death worldwide. In 2013, there were 25.7 million individuals who had survived a stroke, 6.5 million deaths caused by stroke, 113 million disability-adjusted life-years (DALYs) lost, and 10.3 million new instances of strokes.[1] Emerging countries bore the majority of the stroke burden, including 75.2% of all stroke-related deaths and 81.0% of the lost DALYs. With the exception of Japan, the population of Asia continues to have greater mortality rates associated with new strokes compared to the rest of the world.[2] Left ventricular hypertrophy (LVH), characterized by an enlargement of the left ventricle of the heart, is a well-established and autonomous risk factor for elevating the likelihood of stroke.[3] Di Tullio and colleagues conducted a case-control study involving 394 patients who were admitted with their first episode of ischemic stroke, as well as 413 controls who were matched in terms of race, age, and sex. The researchers observed that an increase in left ventricular (LV) relative wall thickness was independently associated with stroke, even after accounting for LV mass (odds ratio [OR], 1.6; 95% confidence interval [CI], 1.1 to 2.3).[4] Castilla-Guerra et al. found that 42.3% of patients with acute ischemic stroke exhibited signs of left ventricular hypertrophy (LVH) on transthoracic echocardiography (TTE).[5] According to Rodrigo C et al., 76.3% of patients with ischemic stroke showed signs of left ventricular hypertrophy (LVH).[6] In a study conducted by Sasikumar P et al., it was shown that 23.9% of the patients who had a stroke showed signs of left ventricular hypertrophy (LVH).[7] The renowned Israeli Heart Diseases project (IIHD), led by Tanne D et al., monitored a cohort of 10,000 individuals for over 20 years. The study conclusively showed that patients who experienced an ischemic stroke had a significantly higher risk of left ventricular hypertrophy (LVH) with a hazard ratio of 2.15.[8] Most people who show signs of left ventricular hypertrophy (LVH) on transthoracic echocardiography (TTE) are likely to continue without

symptoms unless a significant adverse event occurs.[9] This underscores the potential necessity for community screening on a broader scale. Eccentric hypertrophy, which increases the demand for oxygen in the heart muscle, can cause symptoms such as angina or ischemia. Furthermore, the presence of left ventricular hypertrophy (LVH) increases the likelihood of developing arrhythmias due to the interference of the enlarged heart muscle with the regular conduction of electrical signals. This increases the likelihood of developing atrial fibrillation, which can result in an ischemic stroke.[10] After examining the local literature, it is evident that there is a lack of information on the potential link between an elevated stroke risk and the presence of left ventricular hypertrophy (LVH). To go deeper into the concept, we initiated a pilot experiment focusing on admitted patients in our hospital who had confirmed evidence of stroke on CT scans and displayed signs of left ventricular hypertrophy (LVH).

METHODOLOGY

The Department of Internal Medicine at Hayatabad Medical Complex, Peshawar, conducted this cross-sectional study between October 1, 2020, and April 30, 2021, using sequential sampling approaches, a non-probability method. The design was chosen to see the prevalence of LVH in acute ischemic stroke patients so that further advanced studies can be carried out later. The WHO employed a sample size calculation formula with a 95% confidence level and a 7% margin of error. Maintaining an incidence of LVH in ischemic stroke at 23.9% resulted in a minimum required sample size of 143.[7] The hospital's Ethics Committee granted formal ethical approval following a comprehensive presentation of the study methods, objectives, and anticipated outcomes. All persons included in the study or their responsible carers provided fully informed, structured, and written consent. All adult patients regardless of gender, aged between 18 and 75 years, who had a confirmed diagnosis of an ischemic stroke on a non-contrast CT scan of the brain and were brought to the hospital were included in the study. Patients could have been admitted through either the casualty or outpatient departments. The study excluded patients who had previously experienced multiple cerebrovascular accidents, hypoglycemic or metabolic brain injuries, or had a history of meningitis or encephalitis within the past six months. In addition, the study excluded patients who had evidence of a space-occupying lesion on their CT brain scan. We obtained a comprehensive and organized record of all the established parameters from the patient or their family member. A two-dimensional M-mode transthoracic echocardiography (TTE) was done on each patient to find left ventricular hypertrophy (LVH) by figuring out the left ventricle's real mass. A measurement exceeding 8mm in women and 9mm in men was used as the diagnostic criteria. The operator remained an individual to minimize any observational bias. The data was analyzed using SPSS version 23. We computed the average and variability for quantitative data such as age, height, weight, and BMI. We analyzed categorical factors, including gender, HTN, DM, smoking status, domicile, and LVH, to determine their frequency and percentages. We assessed the impact of these factors by analyzing LVH with age, gender, BMI, DM, residency, HTN, and smoking status. We examined the effect changes using a chi-square test, with a significance level of $p < 0.05$. We displayed all results in tabular format.

RESULTS

The mean age of the 143 patients in the study was 57 + 7.9 years, with 67.8% males and 32.2% females having a mean BMI of 25.3 $\pm$ 3.3kg/m^2. The age distribution of the sample is shown in table 1.

Table 1: The age distribution of the sample n=143

Age	Frequency (%)
41-50 years	39 (27.3)
>50-60 years	57 (39.9)
>60-70 years	47 (32.9)
Total	143 (100.0)

The frequency of Diabetes and LVH is shown in Tables 2 and 3.

Table 2: Frequency of Diabetes (n=143)

Diabetes	Frequency	%age
Yes	51	35.7
No	92	64.3

Table 3: Frequency of LVH (n=143)

LVH	Frequency	%age
Yes	24	16.8
No	119	83.2

The risk stratification of the patients according to the age of the patients, gender, BMI, residence, presence or absence of HTN, DM, and Smoking are shown in Table 4.

Table 4: Risk Stratification concerning age, gender, BMI, residence, HTN, Smoking, and DM (n=143)

Variable	Age range	Presence of LVH		P-Value
		Yes (%)	No (%)	
Age of participants	41-50 years	(10) 25.6	(29) 74.4	0.082
	50-60 years	(05) 8.8	(52) 91.2	
	60-70 years	(09) 19.1	(38) 80.9	
	Total	(24) 16.8	(119) 83.2	
Gender	Male	20 (20.6)	77 (79.4)	0.075
	Female	04 (8.7)	42 (91.3)	
	Total	24 (16.8)	119 (82.2)	
BMI	20-24.9	10 (17.5)	47 (82.5)	0.265
	25-29.9	09 (13)	60 (87)	
	30-33	05 (29.4)	12 (70.6)	
	Total	24 (16.8)	119(83.2)	
Residence	Rural	09 (15.8)	48 (84.2)	0.796
	Urban	15 (17.4)	71 (82.6)	
	Total	24 (16.8)	119 (83.2)	
Hypertension	Yes	07 (14.3)	42 (85.7)	0.564
	No	17 (18.1)	77 (81.9)	
	Total	24 (16.8)	119 (83.2)	
Smoking	Yes	09 (15)	51 (85)	0.626
	No	15 (18.1)	68 (81.9)	
	Total	24 (16.8)	119 (83.2)	
Diabetes	Yes	08 (15.7)	43 (84.3)	0.794
	No	16 (17.4)	76 (82.2)	
	Total	24 (16.8)	119 (83.2)	

DISCUSSION

Stroke continues to be the leading cause of admission in neurology and acute medicine units worldwide.[11] WHO defines stroke as the sudden, rapid onset of a new neurological deficit that is secondary to obstruction or rupture of the cerebral vascular system lasting more than 24 hours, confirmed or refuted by a CT scan, along with the exclusion of non-vascular causes.[12] The mechanisms defining the mechanisms of LVH increasing the risk of ischemic strokes remain under discussion. Some researchers propose that LVH increases the workload in the ventricular system and wall tensions, ultimately resulting in varied ventricular geometries. The resultant diastolic dysfunction and changes in pressure and wall movements lead to a sequel in the form of an increased risk of ischemic strokes.[13] Advancing age, male gender, and black race are non-modifiable risk factors that increase the occurrence of ischemic stroke. However, diabetes, hypertension, smoking, central obesity, and dyslipidemia also pose a considerable threat. Fortunately, there is an opportunity to reduce the risk if these factors are appropriately addressed significantly.[14,15] The Dutch TIA trial study group has also stressed the TTE evidence of LVH as an independent risk factor for stroke and TIAs.[16] We observed that 16.8% of our patients had evidence of LVH, which is lower than reported by Amin et al., where 25% of the 120 patients with ischemic stroke had confirmation of LVH. The prevalence of LVH in ischemic stroke in our regional data is 3.6% based on a small series of 55 patients with acute ischemic stroke reported by Khan NI et al., which is much lower than in our enrolled population. The possible explanation for the increase in LVH in our study may be a difference in the study population and the upsurge of DM and HTN over the past decade.[17] We found that 20.6% of men and 8.7% of women had signs of LVH. This differs from the study by Hayward AC et al., who found that women were more likely to have LVH because their end-systolic volume was higher and their LV cavity was lower. The variance in the study population and the contribution of modifiable and non-modifiable risk factors can explain the difference.[18] Our results showed the fortified occurrence of LVH in patients with a high BMI, from 17.5% in the 20-24.9 group to 29.4% in the 30-33 group. The results appear to agree with Kwon HS et al.'s study, which reported a similar positive correlation between increasing BMI and higher Framingham Risk Scores.[19] This calls for intervention in this modifiable risk factor with a resultant decrease in the occurrence of ischemic strokes. We didn't witness any statistical differences in the frequency of LVH in patients with ischemic stroke about the area of living, with 15.8% in rural areas and 17.4% in urban areas, which is in total alignment with the results of a huge cohort of 06 million people in primary care by Kapral MK, who reported more prevalence of the risk factors in those from urban settings without prior stroke but similar occurrences like our study population when they had a stroke.[20] We observed that, while doing the risk stratification, the patients with evidence of hypertension (14.3%) and diabetes (15%) were not more likely to have evidence of LVH than those with none of these additional illnesses. These results are surprising because a linear positive correlation remains between LVH and increased CV morbidity and mortality.[21] The reason could be due to a small sample size and the poor representation of all segments of patients. Lanni and Colle When you have high blood pressure (HTN) and a certain variation in the platelet glycoprotein IIIa gene (GPIIIa PlA2), Lanni and his colleagues found that you are three times more likely to have an ischemic stroke. This seems to be at odds with our findings, which showed that patients with HTN did not have a higher risk of LVH compared to patients who did not have hypertension. Mass population studies are necessary to ensure that all population segments are included.[22]

LIMITATIONS

This study has some limitations, like we study only the prevalence of LVH in ischemic stroke patients. We did not correlate the degree of LVH with stroke or LVH

with the severity of ischemic stroke. These can be the recommendation for further studies to be conducted on the severity of acute ischemic stroke with LVH. Despite some limitations, this study shows the importance of LVH in acute ischemic stroke, which has clinical implications, such as preventing the progression of LVH, which can have favorable outcomes on the prevalence of acute ischemic stroke.

CONCLUSIONS

Stroke remains a major danger for the susceptible population, and timely recognition of the changeable risk factors at the early stages of the illness and suitable intervention should effectively decrease the incidence of this incapacitating condition. Large-scale population-based research is necessary to define the risks and factors contributing to stroke precisely.

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REFERENCES

1. Feigin VL, Krishnamurthi RV, Parmar P, Norrving B, Mensah GA, Bennett DA, et al. Update on the Global Burden of Ischemic and Hemorrhagic Stroke in 1990-2013: The GBD 2013 Study. Neuroepidemiol 2015; 45(3):161-76.
2. Rajsic S, Gothe H, Borba HH, Sroczynski G, Vujicic J, Toell T. Economic burden of stroke: a systematic review on post-stroke care. European J Health Econ. 2019; 20(1):107-34.
3. Wang H, Wang S, Yi X, Tao Y, Qian H, Jia P et al. Estimate of ischemic stroke prevalence according to a novel 4-tiered classification of left ventricular hypertrophy: insights from the general Chinese population. Ann Med. 2018; 50(6):519-28.
4. Di Tullio MR, Zwas DR, Sacco RL, Sciacca RR, Homma S. Left ventricular mass and geometry and the risk of ischemic stroke. Stroke. 2003;34:2380–2384.
5. Castilla-Guerra L, Fernandez-Moreno MD, Alvarez-Suero J, Gonzalez A. Echocardiographic assessment of hypertensive left ventricular hypertrophy in patients with acute ischemic stroke or transient ischemic attack. J Stroke Cerebrovasc Dis. 2011.
6. Rodrigo C, Weerasinghe S, Jeevagan V, Rajapakse S, Constantine G. Addressing the relationship between cardiac hypertrophy and ischemic stroke: an observational study. Int Arch Med. 2012; 5(1):32.
7. Sasikumar P, Rani PS. Assessment of left ventricular function in ischemic stroke in GDMCH Dharmapuri. Stroke 2017; 4(6):64-70.
8. Tanne D, Yaari S, Goldbourt U. Risk profile and prediction of long-term ischemic stroke mortality: a 21-year follow-up in the Israeli Ischemic Heart Disease (IIHD) Project. Circulation. 1998; 98:1365–1371.
9. Unverzagt FW, McClure LA, Wadley VG, Jenny NS, Go RC, Cushman M, Kissela BM, Kelley BJ, Kennedy R, Moy CS. et al. Vascular risk factors and cognitive impairment in a stroke-free cohort. Neurology. 2011; 77:1729–1736.
10. Bornstein AB, Rao SS, Marwaha K. Left Ventricular Hypertrophy. [Updated 2023 August 8]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024 Jan-.
11. The International Stroke Trial (IST): a randomised trial of aspirin, subcutaneous heparin, both, or neither among 19435 patients with acute ischemic stroke. International Stroke Trial Collaborative Group. Lancet. May 31 1997;349(9065):1569-81.
12. Shiber JR, Fontane E, Adewale A. Stroke registry: hemorrhagic vs. ischemic strokes. Am J Emerg Med. Mar 2010; 28(3):331-3.
13. Bauml MA, Underwood DA. Left ventricular hypertrophy: an overlooked cardiovascular risk factor. Cleve Clin J Med. 2010; 77:381–387.
14. Tissue plasminogen activator for acute ischemic stroke. The National Institute of Neurological Disorders and Stroke rt-PA Stroke Study Group. N Engl J Med. December 14 1995; 333(24):1581-7.
15. Hacke W, Kaste M, Bluhmki E, Brozman M, Dávalos A, Guidetti D, et al. Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. N Engl J Med. September 25 2008; 359(13):1317-29.
16. Predictors of major vascular events in patients with a transient ischemic attack or non-disabling stroke. The Dutch TIA Trial Study Group. Stroke. 1993; 24:527–531.
17. Khan NI, Naz L, Mushtaq S, Rukh L, Ali S, Hussain Z. Ischemic stroke: prevalence of modifiable risk factors in male and female patients in Pakistan. Pak J Pharm Sci. 2009; 22:62–67.
18. Hayward CS, Kalnins WV, Kelly RP. Gender-related differences in left ventricular chamber function. Cardiovascular research. 2001 Feb 1;49(2):340-50.
19. Kwon HS, Song KH, Yu JM, Kim DS, Shon HS, Ahn KJ, Choi SH, Ko SH, Kim W, Lee KH, Nam-Goong IS. Framingham risk score assessment in subjects with pre-diabetes and diabetes: A cross-sectional study in Korea. Journal of Obesity & Metabolic Syndrome. 2021 Sep 9; 30(3):261.
20. Kapral MK, Austin PC, Jeyakumar G, Hall R, Chu A, Khan AM, Jin AY, Martin C, Manuel D, Silver FL, Swartz RH, Tu JV. Rural-Urban Differences in Stroke Risk Factors, Incidence, and Mortality in People With and Without Prior Stroke. Circ Cardiovasc Qual Outcomes. 2019 Feb; 12(2):e004973.
21. Del Zoppo GJ, Saver JL, Jauch EC, Adams HP Jr. Expansion of the time window for treatment of acute ischemic stroke with intravenous tissue plasminogen activator: a science advisory from the American Heart Association/American Stroke Association. Stroke. Aug 2009; 40(8):2945-8.
22. Lanni F, Santulli G, Izzo R, Rubattu S, Zanda B, Volpe M, Iaccarino G, Trimarco B. The Pl (A1/A2) polymorphism of glycoprotein IIIa and cerebrovascular events in hypertension: increased risk of ischemic stroke in high-risk patients. J Hypertens. 2007; 25:551–556.

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