ISSN:0975 -3583,0976-2833 VOL 15, ISSUE 12, 2024

Original research article

Silent Stroke: Role of carotid Doppler in hypertensive patients as a screening tool for the detection of silent stroke with MRI correlation

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Abstract

Background: In the overall senior population, silent stroke range in prevalence from 8% to 28%. The public and medical community have become more aware of the silent stroke as a result of the growing senior population and advancements in imaging technology. Cognitive impairment and a higher risk of later stroke are linked to silent stroke. In this study, hypertension individuals' Carotid Doppler ultrasound is used as a screening tool for silent cerebral stroke using MRI imaging.

Methods: This prospective study was conducted in the Department of radiology and imaging Sciences Meenakshi Hospital, Thanjavur. A total of 45 hypertensive patients fulfilling the inclusion and exclusion criteria were recruited for this study who were referred with symptoms of silent stroke. The patients were screened for signs using carotid doppler ultrasound and compared with MRI brain findings. Findings were recorded in Microsoft excel and analysed using SPSS Version 26 and Epi-info statistical package, version 7.

Results: Among the patients 32 were males and 13 were females. More than 60% (n=36) of the patients were above 60 years of age and giddiness was the most common symptom in 35% (n=16) of patients. There was a statistically significant association between PSV and MRI changes ($p<0.05$) but no significance between vessel wall thickness, presence of plaque or EDV. It had a moderate sensitivity (77%) and specificity (80%), good PPV (90%) and poor NPV (59%) with a moderate diagnostic accuracy (79%).

Conclusions: Carotid Doppler ultrasound can be an effective tool in screening the risk for stroke in patients with symptoms of silent stroke. It can visualise the vascular wall changes in the cerebral vessels to assess the risk for stroke. Yet it cannot be as effective as MRI or MRA. Hence it can be used as a screening tool and not as a diagnostic tool.

Keywords: Silent stroke, carotid Doppler ultrasound, MRI in hypertension

Introduction

A silent stroke is characterised by unusual symptoms such as speech slurring, dizziness, balance issues, numbness or slight weakness, or dizziness [1]. Recently, subgroups of patients with a history of atrial fibrillation, transient ischemic attacks (TIA), carotid disease, or symptomatic stroke have been discovered to have silent cerebral infarction. It is an indicator of a higher chance of experiencing a symptomatic stroke [2].

On the other hand, a stroke is characterised as an abrupt neurologic injury brought on by many pathologic processes. It typically manifests as infarction symptoms such as arm or limb paralysis, difficulty speaking or understanding, and visual impairments [3]. Strokes can range in severity from silent strokes that are only discovered through neuroradiologic examination to TIAs, which are defined as any sudden focal neurologic deficit that goes away in less than a day, to catastrophic events that cause severe incapacitation or even unexpected death [4].

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Materials and Methods

Study Area

This prospective study was conducted in the Department of radiology and imaging Sciences, Meenakshi Hospital, Thanjavur.

Study Population

Hypertensive Patients who are referred with symptoms such as numbness or mild weakness, slurring of speech, dizziness or problems with balance to the department of radiology, Meenakshi Hospital, Thanjavur for MRI brain from other disciplines and out patients departments.

Duration of Study

This study was conducted for 6 months during the period of June 2023 to December 2023. Data was collected for 6months.

Selection of Patients

1. **Inclusion Criteria**

1. Either sex.
2. Age of the participants between 50-70 years.
3. Hypertensive patients on medications for the same.

2. **Exclusion Criteria**

1. Patients below 50 years or above 70 years.
2. Patients with cerebral haemorrhage.
3. Patient with history of old stroke.
4. Comatose patients.
5. Patients presenting with other concomitant neurological or psychiatric disease.
6. Patients not consenting for the study.

This prospective study was conducted in the tertiary care hospital after getting clearance from the ethical committee for the duration of 6 months.

This study will include hypertensive individuals with the systolic BP >130 mm hg and diastolic BP >80 mm hg with symptoms such as numbness or mild weakness, slurring of speech, dizziness or problems with balance, who are referred to radiology department for MRI brain under the age group of 50-70 years. DWI and FLAIR sequences are taken with 1.5T PHILIPS MRI machine (FIgure 1 to 4). All the above patients are subjected to carotid Doppler using VOLUSON E8-high frequency linear transducer of frequency range 7.5-12 MHZ to assess carotid disease status (Figure 5 to 11). MRI findings like lacunar infarct, microbleeds and chronic ischemic changes are subjected to carotid artery doppler. Atherosclerotic changes like intima medial thickness, characterisation of plaque and percentage of area stenosis are calculated using SRU consensus.

1) Intima media thickness more 1mm is considered abnormal.
2) On grayscale, characterization of plaques can be performed:

- **Type I:** Predominantly hypoechoic with thin echogenic rim.
- **Type II:** Echogenic plaque with >50% hypoechoic areas.
- **Type III:** Echogenic plaque with <50% hypoechoic areas.
- **Type IV:** Uniformly echogenic plaque.

3) The degree of stenosis determined at grayscale and Doppler US should be stratified into the categories of normal (no stenosis), <50% stenosis, 50%-69% stenosis, ≥70% stenosis to near occlusion, near and total occlusion. ICA peak systolic velocity (PSV) and presence of plaque on gray-scale and/or color Doppler images are primarily used in diagnosis and grading of ICA stenosis.

Sample Size

The sample size formula used is as follows: **N = deff x Npqd21.962N – 1 + pq;** Where, **n**= Sample Size; ***deff*** = design effect; **p** = the estimated proportion; **q = 1-p; d** = desired absolute precision or absolute level of precision. Results from OpenEpi, Version 3 **- Sample size = 45 with 95% confidence limit.**

Statistical Analysis

Statistical Analysis was done by using SPSS Version 26 and Epi-info statistical package, version 7. All values were expressed as mean (SD) for continuous variables and number (percentages) for discrete variables. Chi-square test and fisher's exact test was used to find out association between the categorical variables. Independent sample 't'-test was used to find the significant difference of continuous variables between groups. P value <0.05 will be considered as statistically significant.

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Results

The study included 45 patients who were referred in from other departments and out-patients department in the Meenakshi Hospital, Thanjavur for Radiological investigations and opinion including both males and females. Majority of the study participants, 71% (n=32) were male. The sex distribution of the patients is shown in Figure 11. The mean age of the patients was 63.6 ± 5.46 years.The symptoms of the patients are shown in Figure 12. The most common symptom among the patients was giddiness 37.8% (n=17) and the least common symptom was imbalance 6.7% (n=3). The prevalence of cerebral changes on MRI is shown in Table 1 and 2 with most common finding being lacunar infarct 31.1% (n=14). Among the study participants 11.1% (n=5) has normal findings on MRI. The type of plaque and its prevalence observed on Carotid Doppler ultrasonography is shown in the Table 3. The most common plaque observed was Type II (26.7%) followed by Type I (22.2%) and Type IV (22.2%) plaque. The proportion of patients with plaque in presence of thickened intima media of blood vessels is summarised in table 4. The proportion of normal PSV findings among the study participants are summarised (Figure 13). The mean PSV findings of the participants was 93.39 ± 45.56 cms/s with a range of 236 cms/s. PSV findings more than 125cms/s was considered normal for the study. The mean EDV of the ICA was 14.69 ± 2.63 cms/s. The cut-off for classification as normal and high was fixed as <40 cms/s. There is no change in EDV findings with increasing age. Association between MRI changes on brain and different types of plaques are summarised (Table 6). An association between MRI findings of changes in the brain and PSV in ICA by Carotid Doppler ultrasonography was evaluated. The association is statistically significant with a p value <0.05 and increase in PSV leading to MRI changes in brain (Table 7). The diagnostic accuracy of Carotid Doppler ultrasound in assessing the risk of stroke in hypertension patients comparing the MRI findings of brain. It has lesser sensitivity (77.7%) and better specificity (80.0%). It also has a good positive predictive value while the negative predictive value is low. The diagnostic accuracy is also on a lesser level (Table 8).

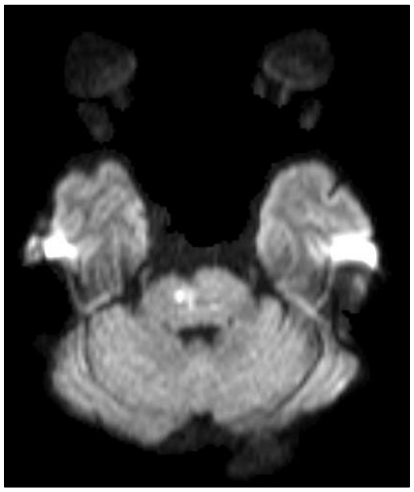

Fig. 1

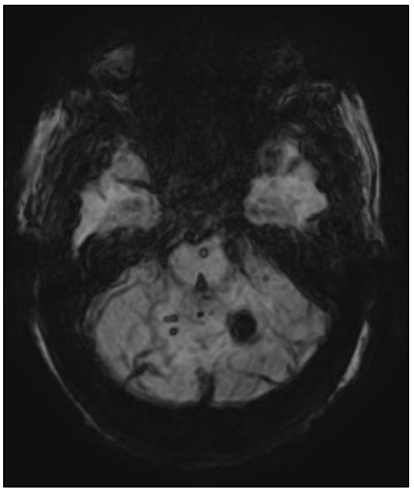

Fig. 2

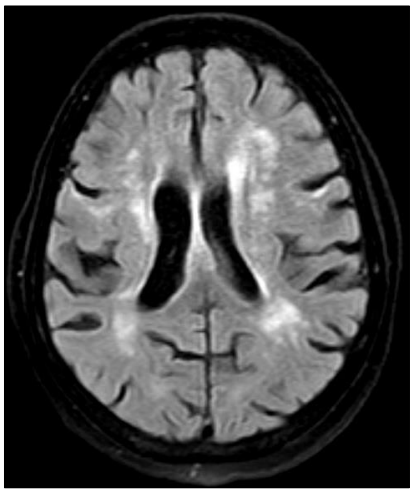

Fig. 3

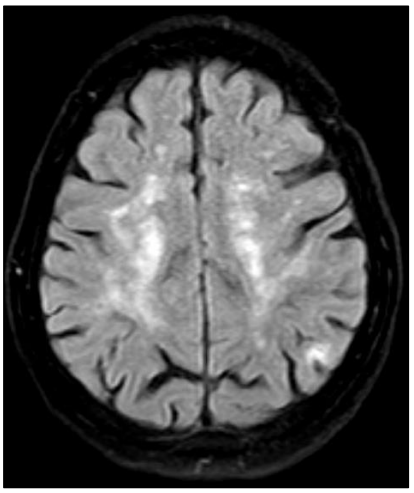

Fig. 4

Fig 1-4: DWI, FLAIR and SWI sequences showing acute infarcts in right hemipons and chronic ischemic changes as bilateral periventricular FLAIR hyperintensities. Evidence of blooming seen in SWI sequence which suggest microbleeds

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Right Side

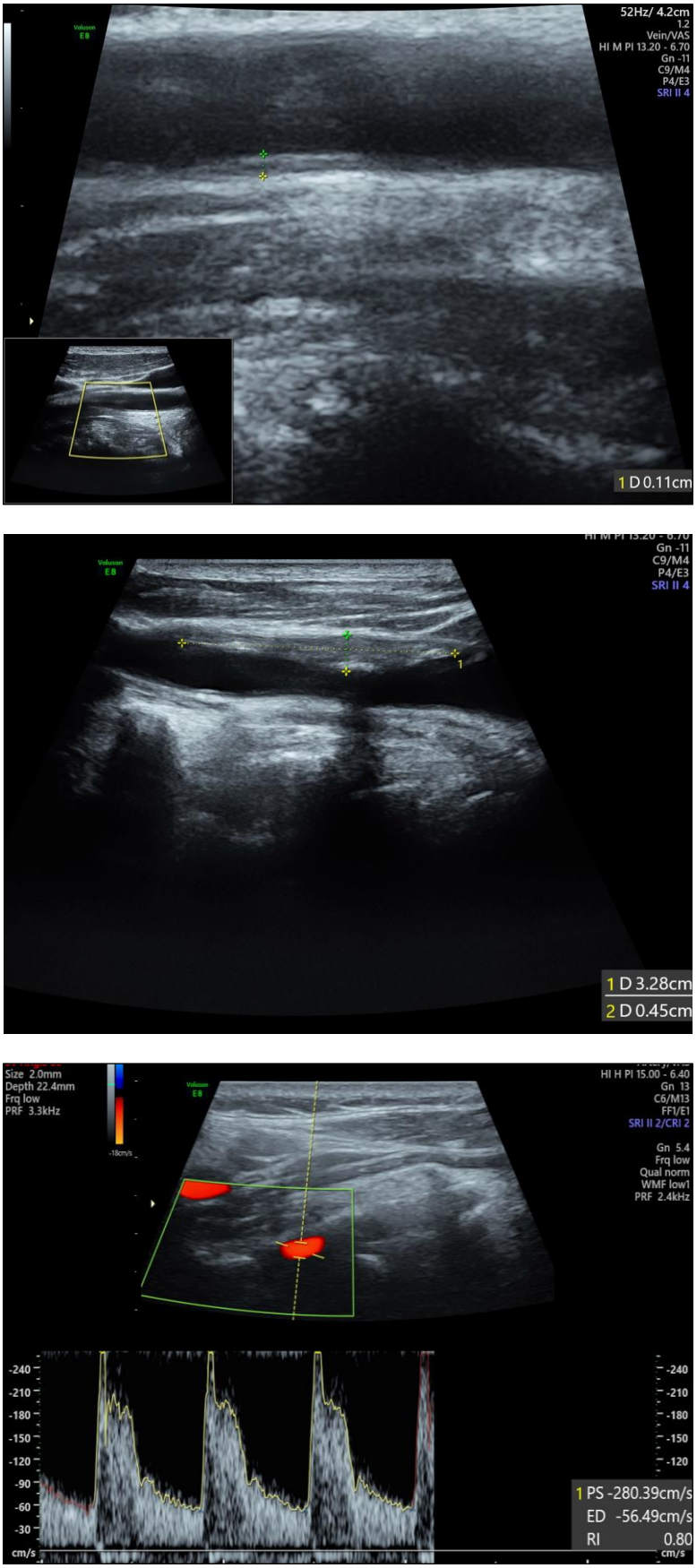

Fig 5-10: High frequency USG with a linear transducer of frequency range 7.5-12 MHZ to assess carotid artery in the form of intima media, plaque characterisation and doppler assessment of PSV

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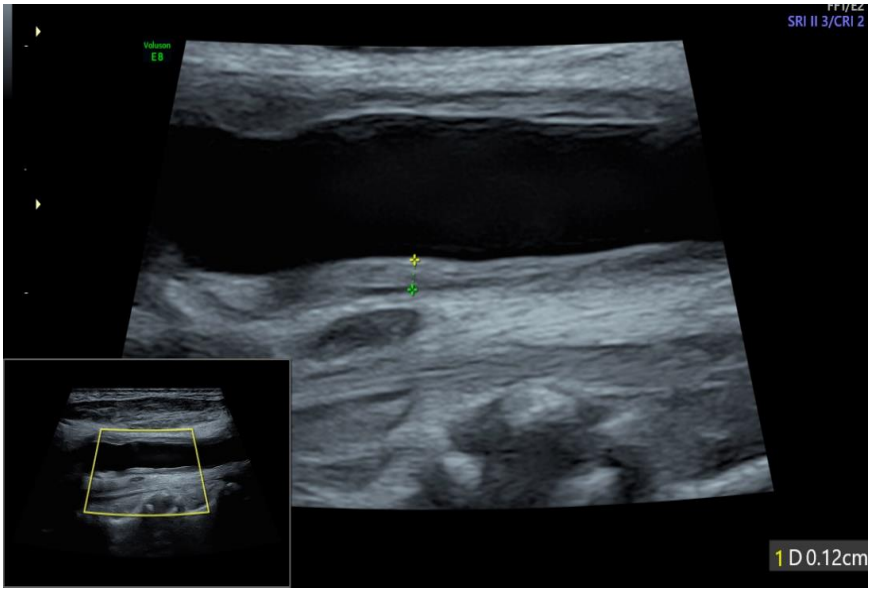
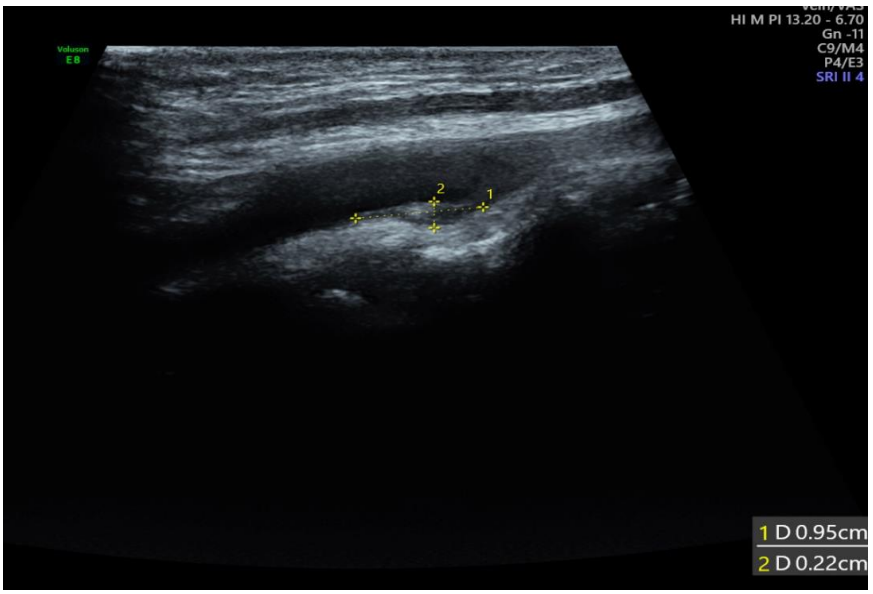
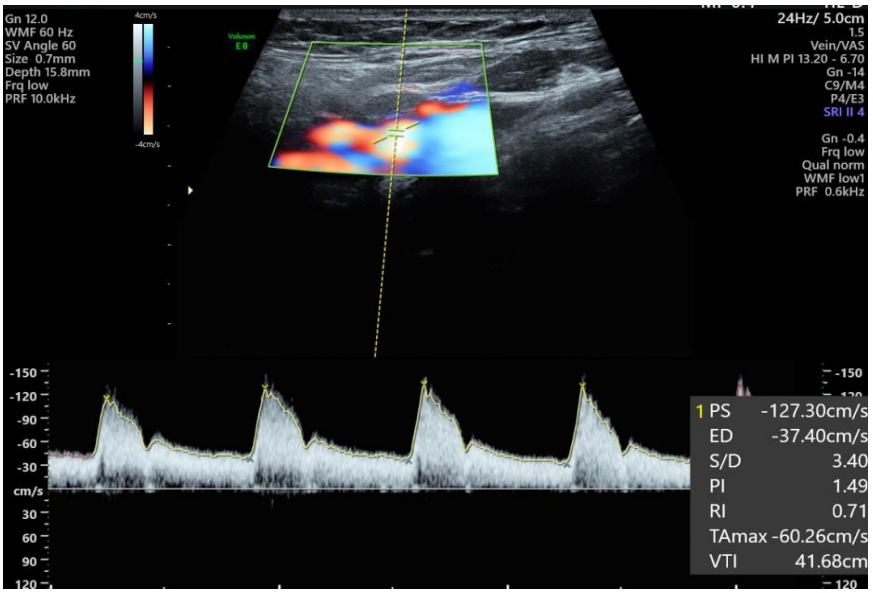
Left Side







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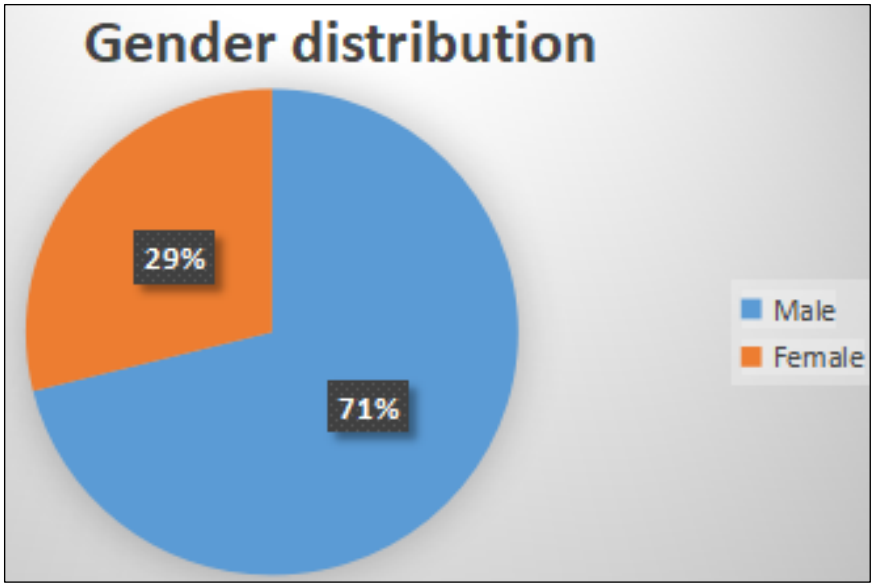

Fig 11: Sex distribution of the patients (n=45)

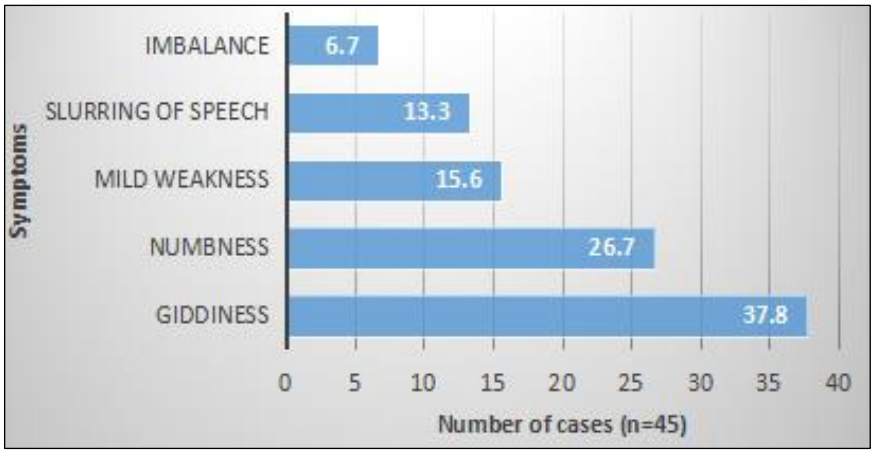

Fig 12: Presenting symptoms of the patients (n=45)

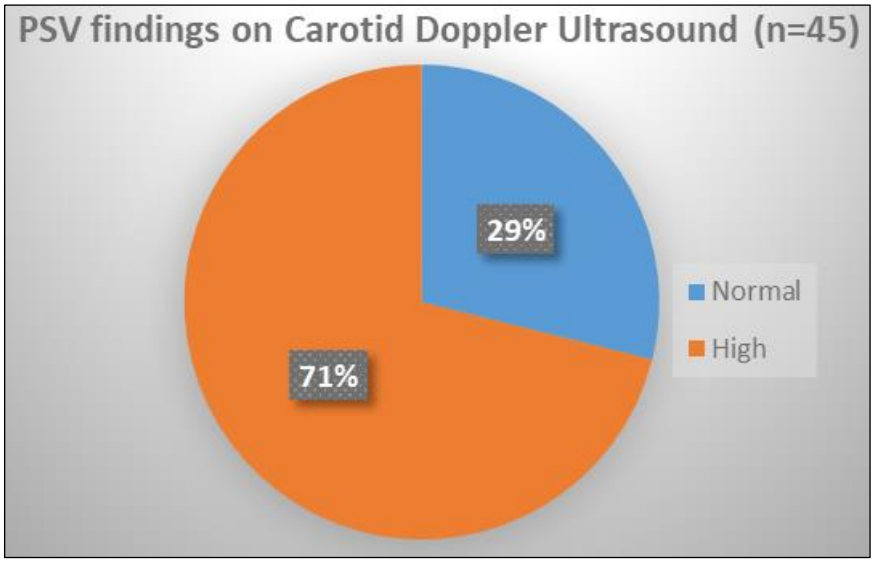

Fig 13: Pie chart representing the proportion or normal and high flow in Peak Systolic Volume among the patients (n=45)

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Table 1: Cerebral changes on MRI (n=45)

Cerebral Changes	Frequency (n=45)	Percentage (%)
Lacunar Infarct	14	31.1
Micro Bleed	4	8.9
Chronic Ischemic Changes	40	88.9

Table 2: Brain changes on MRI

MRI Changes	Frequency (n=45)	Percentage (%)
Changes Present	40	88.9
Changes Absent	5	11.1

Table 3: Type of plaque in Carotid Doppler Ultrasonography. (n=45)

Type of Plaque on Carotid Doppler Ultrasonography	Frequency (n=45)	Percentage (%)
Type I Plaque	10	22.2
Type II Plaque	12	26.7
Type III Plaque	5	11.1
Type IV Plaque	10	22.2

Table 4: Distribution of Intima Media Thickness with Plaque

IMT Thickness	Presence of Plaque	
	Present	Normal
Thickened	32 (100)	0 (0)
Absent	0 (0)	13 (100)

Table 5: Category of stenosis with age classification (n=45)

Level of Occlusion	Frequency (n=45)	Percentage (%)
No Stenosis	21	46.7
ICA Stenosis <50%	26	57.8
ICA Stenosis 50-69%	5	11.1
ICA Stenosis >70%	4	8.9
No Near Occlusion	45	100
No Total Occlusion	45	100

Table 6: Association between MRI changes on brain and different types of plaques. (n=45)

Type of Plaque	MRI Findings		Odds Ratio	$\chi 2$	p value
	Change	Normal			
Type 1	9 (22.5%)	1 (20%)	1.161	0.016	0.899
Type 2	12 (30%)	0 (0%)	n/a	2.045	0.153
Type 3	5 (12.5%)	0 (0%)	n/a	0.703	0.933
Type 4	9 (22.5%)	1 (20%)	1.161	0.016	0.899

Table 7: Association between MRI changes on brain and PSV on Carotid Doppler Ultrasound. (n=45)

PSV	MRI Findings		Odds ratio	$\chi 2$	p value
	Change	Normal			
High	31 (77.5%)	1 (20%)	13.777	7.153	0.031*
Normal	9 (22.5%)	4 (80%)			

Table 8: Assessing the Diagnostic accuracy of Carotid Doppler Ultrasound with MRI brain

Factors	Percentage (%)	95% CI (%)
Sensitivity	77.7	65.3-89.7
Specificity	80.0	68.3-91.7
PPV	90.5	81.9-99.0
NPV	59.2	44.9-73.6
Diagnostic Accuracy	77.9	65.6-89.9

Table 9: Risk factors and causes of stroke

Risk factors for stroke [7, 29]	Causes of Stroke [7, 30, 31]
Fixed risk factors	
a. Age	1. Atherosclerotic thrombosis
b. Gender (male >female except at extremes of age)	2. Embolism
c. Race (African>Asian >European)	3. Transient Ischaemic Attack

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d. Previous vascular event:	4. Intra Cranial Haemorrhage
• Myocardial infarction	5. Sub Arachnoid Haemorrhage
• Stroke	6. Hypertensive haemorrhage
• Peripheral vascular disease	7. Head trauma
e. Heredity	8. Metastatic brain tumour
f. Sickle cell disease	9. Amyloidangiopathy
g. High fibrinogen	10. Trauma and dissection of carotid and basilar arteries
ii. Modifiable risk factors	11. Dissecting aortic aneurysm
a. Hypertension	
b. Cigarette smoking	
c. Hyperlipidaemia	
d. Diabetes mellitus	
e. Oestrogen-containing drug	
f. Polycythaemia	
g. Heart disease:	
h. Excessive alcohol intake	

Discussion

Stroke lowers quality of life and is one of the main causes of death, illness, and disability [5]. Ischemic infarcts account for 80-85% of strokes among the causes. The patient, family, and carers are all greatly impacted by neurological disabilities associated to stroke [6]. Almost 17% of people will stay in institutions and 25-30% will require significant help with daily tasks. One of the most concerning aspects of modern health care is the financial strain that comes with treating stroke sufferers after they are admitted to the hospital [5, 7].

Generally, clots that obstruct brain blood vessels account for 80% of ischemic strokes. Ischemia results from the blockage preventing oxygen and blood from getting to that location [8]. The risk of dementia and eventual stroke is doubled in the presence of silent infarcts [9]. Since the greatest risk factor for stroke is hypertension, the introduction of antihypertensive medication has been primarily responsible for the sharp decrease in stroke-related mortality that has occurred since 1975 [10].

One can categorise the aetiology of ischemic cerebrovascular illness into two groups: non-cardiac and cardiac. Atherosclerosis, arteritis, hematologic diseases, cerebral vasospasm, and reversible cerebral vasoconstriction are some of the significant non-cardiac causes [11]. The primary cause of heart attacks is atherosclerotic emboli, which come from Coronary artery disease (CAD). The left atrial thrombus is the primary cause of systemic emboli. Strokes can also be less frequently caused by atrial septal aneurysms and tumors (myxomas) [12, 13].

A silent stroke, also known as an asymptomatic cerebral infarction, is one in which the patient is usually unaware that they have experienced a stroke and does not exhibit any obvious symptoms. A silent stroke nevertheless damages the brain and increases the patient's risk of future massive stroke and transient ischemic attack, even in the absence of noticeable symptoms. Most silent strokes result in lesions that can be found using neuroimaging, such MRI. Silent stroke is more common in older persons, but it can also strike younger adults. Women seem to be more susceptible to silent stroke; predisposing variables include hypertension and current cigarette smoking [16, 43].

These include small haemorrhages and mild ischemic strokes, such as lacunar strokes. Leukoaraiosis is another possibility: because the white matter has fewer blood arteries than the cerebral cortex, it is more vulnerable to vascular blockage. The reason these strokes are called "silent" is that they usually impact "silent" parts of the brain that do not significantly impair a victim's motor abilities, such as contralateral paralysis, slurred speech, pain, or altered touch perception. A silent stroke is a major contributor to vascular cognitive impairment, can result in a loss of control over one's bladder, and usually affects brain regions linked to mood regulation, mental processes, and cognitive functions. A transient ischemic attack (TIA) is not the same as a silent stroke. Stroke symptoms can appear in TIA and can persist for several minutes to a whole day before they go away. A transient ischemic attack (TIA) increases the risk of a catastrophic stroke and subsequent silent strokes [14, 16]. The various risk factors and causes are summarised (Table 9).

Eighty-five percent of stroke cases are caused by ischemic stroke. It could be caused by athero thrombosis of the cerebral vessels (53%) which can be further divided into embolic (32%), small vessel thrombosis (Lacunar strokes: 20%), and big vessel thrombosis (33%). Atherosclerosis and thrombosis of intracranial cerebral blood vessels and extracranial carotid arteries occur similarly to that of coronary arteries. Plaque development and carotid stenosis are caused by atherosclerosis of the carotid arteries [14].

Numerous researchers have looked into the connection between silent infarction and carotid disease [14]. The researchers came to the conclusion that there is a high probability that the processes of infarction and carotid disease are similar and share risk factors, including hypertension [15, 16]. When a silent stroke is discovered, it is easier to begin antihypertensive and anticoagulant medication as soon as feasible [17].

For the diagnosis of acute stroke, magnetic resonance imaging (MRI) is usually considered to be superior to computed tomography (CT). When evaluating individuals who have suffered an acute stroke, the

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optimal imaging modality should be able to distinguish between intracranial haemorrhage and cerebral ischaemia, as well as distinguish cerebrovascular causes from other possible causes. MRI has benefits when evaluating acute stroke patients [18, 19].

Acute cerebral ischemia triggers a series of biological reactions, such as the disruption of electrolyte transport across the cellular membrane and the consequent intracellular accumulation of water (cytotoxic oedema) [8]. Water molecules' random translational motion decreases when extracellular water content decreases. By detecting the random motion of water molecules using the impact of magnetic gradients on protons, diffusion weighted pictures are produced. In comparison to the surrounding normal brain tissue, areas with decreased water diffusion are displayed as being hyperintense [20, 21]. More than 90% of patients with a final clinical diagnosis of acute ischemic stroke have brain ischaemia detected by DWI, which has higher interobserver reliability and sensitivity than computed tomography [22].

Almost all brain imaging protocols include the FLAIR sequence, which is especially helpful in identifying minute alterations in the CSF and in the periventricular area surrounding the hemispheres that might be signs of chronic ischemia. Within six hours after the start of symptoms, SWI enables the early diagnosis of acute haemorrhage. Additionally, SWI can detect prior microbleeds in cases of acute ischemia [15].

When carotid plaques result in cerebral ischaemia symptoms, they are categorised as symptomatic. Plaque rupture and the ensuing thrombus development are to blame for this [7, 23]. Continuous wave Doppler was used in the early stages of carotid ultrasonography procedures. The next significant development was Duplex Ultrasonography, an elegant way to combine Doppler information with real-time images. The most recent advancement in Doppler Ultrasonography is colour flow, which grades stenosis and photographs the plaque by superimposing color-coded flow information on grayscale images [24].

The initiation of understanding of the blood vessels began with the emergence of the blood vessel imaging which began in 1929. It was when Forssmann injected himself with contrast medium through a large bore catheter to understand it. Despite being known to be a risky treatment, angiography's diagnostic potential was soon recognised, and a whole new area of neurosurgery swiftly developed. In Edinburg, Dott treated a brain aneurysm in 1932. In 1954, the first Carotid endarterectomy was carried out at Eastcott in London. The understanding of dangers has advanced along with angiographic technology. The danger of angiography may exceed the benefits of treatment in cases where the latter are negligible, such as in individuals with low grade Carotid stenosis. As a result, less intrusive methods of imaging blood vessels have been developed, such as magnetic resonance angiography, CT angiography, and Carotid Doppler Ultrasonography [28].

Continuous wave Doppler was used in the early stages of carotid ultrasonography procedures. The next significant development was Duplex Ultrasonography, an elegant way to combine Doppler information with real-time images. Colour flow Doppler ultrasonography, which superimposes color-coded flow information on grayscale pictures, is the most recent advancement.

Atherosclerotic changes like intima medial thickness, characterisation of plaque and percentage of area stenosis are calculated using SRU consensus.

1. Intima medial thickness more 1mm is considered abnormal.
2. On grayscale, characterization of plaques can be performed:

- **Type I:** Predominantly hypoechoic with thin echogenic rim.
- **Type II:** Echogenic plaque with >50% hypoechoic areas.
- **Type III:** Echogenic plaque with <50% hypoechoic areas.
- **Type IV:** Uniformly echogenic plaque.

3. The degree of stenosis determined at grayscale and Doppler US should be stratified into the categories of normal (no stenosis), <50% stenosis, 50%-69% stenosis, ≥70% stenosis to near occlusion, near and total occlusion [25, 26].

Research has shown that echo lucency is associated with an increased risk of stroke. By characterizing the plaque, we can decide the prompt treatment like anti platelet or carotid endarterctomy [27]. The present study aims to use carotid Doppler as a screening tool for silent cerebral stroke with MRI imaging in hypertensive patients.

This study assessed the changes in the Carotid arteries in hypertensive patients by means of Carotid Doppler ultrasound who were presented with symptoms of silent stroke. It was planned to assess the changes in brain detected using the MRI brain in hypertension patients with symptoms in comparison with Carotid Doppler of ICA in the patients [55, 56]. In this study we included 45 hypertensive patients who had symptoms of silent stroke. Among the study participants 32 were males and 13 were females. The patients were selected between the ages 50 years to 70 years of age. Majority of the study participants were between the age group of 60 to 70 years of age and least were between the age group of 50 to 60 years.

The mean age of the study participants was 63 ± 4.46 years years with a range of 20 years. Similar to our studies, in several studies conducted in Asia and Europe, the most common age group presenting with

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symptoms were above 60 years and males were predominantly involved with symptoms of silent stroke [5, 7, 29, 31]. This observation could probably be due to the increased high risk factors among males leading increased symptoms.

The most common presenting symptom was giddiness followed by numbness of the limbs. The symptoms increased in prevalence in patients more than 60 years. Similar findings were observed in other studies with increased symptoms in people more than 60 years and the MRI changes were prominent. It's apparent from the findings that the symptoms increase with increasing age (57-59). These findings are possibly due to vessel wall changes with increasing ages and with high blood pressure and increased possibility of brain parenchyma changes on MRI. As the study involved only hypertensive patients, all patients had blood pressure ≥130/85 mmHg.

Cerebral changes were present in majority of the hypertensive patients. The most common cerebral change among hypertensive patients was chronic ischemic changes followed by lacunar infarction and micro bleed on MRI brain. The overall prevalence of normal brain findings was very less. This finding were evident and there was presence of Carotid Doppler ultrasound abnormality in the vessel wall thickness and the patency. The findings w identical with the findings from other studies in different populations in different centers with increased chronic ischemic changes with hypertensive patients [8, 10, 17, 24, 60]. This may be due to the increased IMT, occlusion and stenosis of ICA in hypertensive patients with increased age which is evident as MRI changes.

In our study, Intima media thickness was increased in more than 60% of the hypertensive patients with symptoms of silent stroke on both sides and the findings were more prominent in patients more than 60 years. Similar findings were found in studies conducted in different geographical locations [25, 40, 61]. This finding could be due to the vascular changes with increasing age and with hypertension. Hence analysis was done using intima media thickness as a constant factor to find the association of other factors for risk of stroke.

The prevalence of type of plaque increased with increasing age in hypertensive patients in our study findings. This increase in the vascular plaque was significantly associated with increased IMT in risk for stroke in the patients. Similarly the changes in brain on MRI increased with the presence of the plaque. Plaque type was considered as a high risk for stroke and Type II and Type III plaque was considered vulnerable. For analysis any level of plaque was considered as presence of plaque [35, 53]. The increase in the prevalence of plaque increased with increasing age of the patient and with the presence of MRI changes. But this association was not statistically significant. This finding was not identical with other studies and the risk of stroke increased with increasing prevalence of plaque and brain changes in other studies was not evident in our studies. This could be due to genetic predisposition in Indian population and in particular Tamil Nadu [24, 37, 55, 61].

The PSV findings was classified as high and normal based on the carotid doppler ultrasound. It was compared with the MRI findings of the patients. The prevalence of high PSV was high among those who had changes on MRI brain when compared to those with normal PSV. This finding was statistically significant with with the MRI changes with those having high PSV 13 times higher odds of developing stroke identified by MRI and this association was statistically significant. Several studies on symptomatic hypertensive patients had similar changes on carotid doppler ultrasound which was comparable with the MRI changes in brain. Hence it is evident that with high PSV the MRI changes of silent brain is prominent [33, 46, 52, 61].

The EDV of all the participants on carotid doppler ultrasound with symptoms of stroke and MRI changes was high above the normal range. Among them majority of them had changes in MRI brain and approximately 10% has normal MRI findings. But statistical analysis was not able to be done as none of the participants had normal EDV. But when compared with other studies, there was statistically significant change in the EDV among those with brain changes on MRI. This change in other studies is contributed to the decreased blood flow and availability for tissues leading to the MRI changes [33, 51, 52]. More study samples are needed for further proof of findings to be compared with other studies.

The presence of stenosis was high among the study participants with hypertension and Cerebral changes on MRI. The most common form of stenosis was <50% stenosis of ICA. All the patients has a patent ICA with no total occlusion or near total occlusion. This high prevalence of stenosis of ICA ranging from <50% to >70% of stenosis was not statistically significantly associated with increased cerebral changes on MRI, leading to risk of stroke. But most studies had a varied finding that increased cerebral changes with increased level of stenosis. Such varied findings observed could possibly be due to the reduced blood flow and turbulence which could lead to thrombus formation or intracranial hemorrhage where there is a stenosis [6, 32, 49, 62].

When considering the level of occlusion, stenosis <50% if the ICA was more common than stenosis of 50-70% and >70%. When finding the association between the cerebral changed on MRI and the level of occlusion, all levels of stenosis were not strongly associated with bran changes on MRI for the risk of silent stroke.

In our study, none of the hypertensive patients had near occlusion or total occlusion of ICA. All the study participants had a patent ICA. But in other studies, with different study populations near occlusion was

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significantly associated with silent stroke and embolic stroke while total occlusion was significantly associated with either hemorrhagic stroke of embolic stroke. This difference in observation may be due to the lack of details regarding the duration since the onset of hypertension and the presentation of symptoms among the patients [63, 64, 65].

The IMT on Carotid doppler ultrasound of the study participants was high among those with cerebral changes on MRI. The prevalence of IMT with MRI finding were associated with the risk of stoke in the hypertensive patients. But this increased thickened IMT was not statistically significant with the risk of stoke among the patients with MRI changes. Several studies had similar association of thickened IMT with cerebral changes in patients with risk of stroke. Buth those findings were statistically significant with the MRI changes [33, 53, 61]. The varied findings could be due to confounders like the duration of hypertension and the associated risk factors among the patients.

On analysis of the sensitivity and specificity of carotid doppler ultrasound with the MRI brain of hypertension patients with symptoms the sensitivity and specificity was at moderate level above 75%. It had a good positive predictive value (PPV) and poor negative predictive value (NPV). For a diagnostic test to be reliable it should have good sensitivity specificity, PPV and NPV which should be ideally above 95%. It helps to identify those who are positive for the disease and exclude those who don't have the disease. The PPV helps to identify those who actually have disease among those with symptoms while NPV helps to rule out the individuals without disease from those who don't have symptoms. The diagnostic accuracy was also on moderate level from our findings.

Several studies have had different sensitivity, specifically, PPV and NPV. In a study the sensitivity, specificity PPV and NPV were high above 90% indicating carotid doppler ultrasound as a reliable diagnostic tool [66]. Whereas other studies presented with similar findings to our findings with moderate sensitivity, specificity, PPV and NPV [67, 68, 69]. From this we can conclude that Carotid doppler ultrasound can be used as a screening tool for the risk of stroke and its not superior to MRI brain or other diagnostic or screening in identifying the risk of stroke.

Hence from the above findings we can come to a suggestion that Carotid Doppler ultrasound can be used as a reliable source for screening the risk of stroke in hypertension patients by assessing the IMT, Type of plaque, presence of stenosis or occlusion, percentage of stenosis, PSV and EDV but not as a diagnostic tool.

Conclusion

Doppler Ultrasonography of Carotid arteries is a significant tool to identify the changes in Carotid arteries in Hypertensive patients who are prone for silent stroke. Vessel wall abnormalities are more evident by doppler ultrasonography. Changes in the vessel wall thickness on both sides were able to be compared to identify the possibility of damage due to stenosis or occlusion. The type of plaque due to atherosclerosis and the level of stenosis or occlusion was more prominent to assess the risk in the patients. The flow levels and abnormality were identified but the relation with the vessel wall thickness was not evident. In spite of the following findings, Carotid Doppler ultrasonography cannot be substituted for angiography as it has its own pros and conns. The sensitivity and specificity is moderate hence it cannot identify those with silent stoke among those with symptoms of stroke and cannot exclude all those without risk for stroke from those without symptoms. The PPV was higher indicating that it can truly identify the risk of stroke when screened positive. But low NPV indicated that it can't rule out those screened negative as those without risk of stroke. The diagnostic accuracy is also at a lower level indicating it as a poor diagnostic tool but can be recommended as a screening tool. MRI is the most reliable method for diagnosing the risk of silent stroke. More research with larger samples is required for further proof of literature to bring light on the effectiveness of Carotid Doppler ultrasonography. Through the findings of this study, it can be suggested that Carotid Doppler ultrasonography can be used as a screening tool for detecting asymptomatic Carotid disease in patients with the risk of silent stroke.

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