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ORIGINAL RESEARCH ARTICLE

To Study the Incidence of Lacunar Infarcts in Patients with Acute Ischemic Stroke and its Correlation with Carotid Artery Stenosis

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ABSTRACT

Introduction: Stroke remains the second leading cause of death worldwide, after ischemic heart disease. Lacunar infarcts are small deep infarcts ranging from 2 to 20 mm in size resulting from occlusion of a penetrating artery which accounts for approximately 25% of all ischemic strokes. The present study was undertaken to study the incidence of lacunar infarcts in patients with acute ischemic stroke and its correlation with carotid stenosis.

Materials and methods: This study was performed at the Department of Medicine at a tertiary-care hospital in Amritsar, Punjab, in 50 patients presenting with acute ischemic stroke with or without lacunar syndrome. All patients were diagnosed using diffusion-weighted imaging (DWI) on magnetic resonance imaging (MRI) of the brain. Carotid artery stenosis was measured with duplex ultrasound.

Results: Patients with acute ischemic stroke had a mean age of 61.36 ± 11.36 years. About 40% of the patients had lacunar infarcts with a higher frequency in male patients (64% vs 36%). There was no significant difference found in the incidence of hypertension (85% vs 70%), diabetes (20% vs 13.3%, *p* value = 0.529), alcohol drinking (20% vs 16.67.0%, *p* value = 0.764), smoking (23% vs 10% *p* value = 0.318), and carotid artery stenosis (35% vs 43.3%, *p* value = 0.828) between lacunar and non-lacunar infarct patients.

Conclusion: The difference in the incidence of potential risk factors such as hypertension, diabetes mellitus, alcoholism, smoking, and dyslipidemia was found to be statistically insignificant between lacunar and non-lacunar infarct groups. Carotid stenosis did not show any significant difference in the lacunar and non-lacunar infarct groups. The findings in our study are consistent with the hypothesis that severe carotid artery stenosis in lacunar infarction is an incidental finding.

Keywords: Carotid artery stenosis, Ischemic stroke, Lacunar infarct.

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INTRODUCTION

Stroke remains the second leading cause of death worldwide,[1] after ischemic heart disease. Early diagnosis and treatment is necessary to prevent mortality and morbidity.[2] Stroke or cerebrovascular accident is a clinical syndrome, and has been defined by the World Health Organization (WHO) as "rapidly developing clinical signs of focal (at times global) disturbance of cerebral function, lasting more than 24 hours or leading to death with no apparent cause other than that of vascular origin." Lacunar infarcts are small deep infarcts ranging from 2 to 20 mm in size resulting from occlusion of a penetrating artery which accounts for approximately 25% of all ischemic strokes. Each of the five classical lacunar syndromes as described by Fisher has a relatively distinct symptom complex.[3] Symptoms may occur suddenly, progressively, or in a fluctuating (e.g., the capsular warning syndrome) manner. Occasionally, cortical infarcts and intracranial hemorrhages can mimic lacunar infarcts, but true cortical infarct signs (such as aphasia, neglect, and visual field defects) are always absent in lacunar stroke.

The five classic lacunar syndromes are as follows:

1. Pure motor stroke/hemiparesis
2. Ataxic hemiparesis
3. Dysarthria/clumsy hand
4. Pure sensory stroke
5. Mixed sensorimotor stroke[4,5]

The frequency of carotid artery disease in lacunar strokes has been found to be from 3 to 39% in different studies and the presence of carotid stenosis in such lesions is of clinical interest and offers management by surgery. So the present study was carried out to find out the incidence of lacunar infarction in the cases of ischemic stroke and their correlation with carotid artery stenosis.

MATERIALS AND METHODS

The present study was conducted at the Department of Medicine at a tertiary care teaching hospital in Amritsar, Punjab. Research work was started after getting approval from the Research and Ethics Committee of the Institute. The study included 50 patients presenting with acute ischemic stroke with or without acute lacunar syndrome who were admitted to the ICU, emergency, and IPD. After applying exclusion and inclusion criteria, the subjects were included in the study. Written informed consent was obtained from every enrolled patient. Diagnosis was confirmed on the basis of magnetic resonance imaging (MRI) scan of the brain and duplex scanning of the carotid arteries. Patients underwent MRI with diffusion-weighted images (DWI) at the time of admission to hospital. The common carotid arteries were evaluated with high-resolution ultrasound equipped with a linear transducer with 7.5 MHz in the B mode.

Inclusion Criteria

Patients suffering from acute ischemic stroke admitted to the hospital.

Exclusion Criteria

History of Previous Stroke

Patients who did not give informed consent to participate in the study.

RESULTS

The study included 50 patients admitted in ICU, emergency, and IPD with acute ischemic stroke having lacunar or non-lacunar infarcts, after applying inclusion and exclusion criteria.

Table 1: MRI findings with lacunar infarcts

MRI brain	*No.*	*% age*
Lacunar infarct	20	40.0
Non-lacunar infarct	30	60.0
Total	50	100.0

Table 1 shows that out of 50 patients included in the study, MRI of the brain of 20 (40%) patients showed lacunar infarcts while 30 (60%) patients had non-lacunar infarcts.

Table 2 shows that there was no statistically significant *p* value for risk factors such as alcohol, smoking, hypertension, diabetes mellitus and dyslipidemia in both lacunar and non-lacunar infarct groups.

Out of the 20 patients with lacunar infarcts, 7 (35%) cases showed carotid artery stenosis in duplex scan while 13 (65%) patients had no stenosis, as given in Table 3.

Carotid artery stenosis was classified per the North American Symptomatic Carotid Endarterectomy Trial (NASCET). Out of 20 patients who had lacunar infarct in the MRI, 3 (15%) patients had less than 40% of carotid artery stenosis, and 3 (15%) cases had 41 to 70% carotid artery stenosis, while only 1 (5%) patient had more than 70% of carotid artery stenosis. Thirteen (65%) patients had no carotid artery stenosis.

In 30 patients of non-lacunar infarct, 6 (20%) cases had less than 40% carotid artery stenosis, 4 (13.3%) patients had 40 to 70% carotid artery stenosis and 3 (10%) patients had carotid artery stenosis more than 70%. No carotid artery stenosis was found in 17 (56.66%) patients. This observation was found to be statistically insignificant (p value > 0.05) as shown in Table 4.

According to the NASCET classification, more than 70% of the carotid artery stenosis is considered significant. Out of 20 patients with lacunar infarct, only 1 (5%) had significant carotid artery stenosis (>70%). In 30 patients with non-lacunar infarcts, 3 (10%) had significant carotid artery stenosis. It was seen that significant carotid artery stenosis was more common in non-lacunar infarcts as compared with that of patients with lacunar infarct, although there was no statistically significant difference between two groups (p value > 0.05), as given in Table 5.

DISCUSSION

Lacunar infarcts, as popularized in the modern era by Fisher,[6] are small, deep infarcts in the vascular territory of small penetrating branches of the large cerebral arteries.

Table 2: Incidence of risk factors in lacunar and non-lacunar infarct patients

Risk factors	*Lacunar*	*Non-lacunar*	*p value*
Alcohol	4	5	0.523
Smoking	4	3	0.217
Systolic blood pressure (mm Hg)	172.30 ± 27.88	174.93 ± 29.89	0.755
Diastolic blood pressure (mm Hg)	100.20 ± 14.17	103.93 ± 13.92	0.361
Diabetes mellitus	4	4	0.401
LDL (mg/dL)	116.20 ± 31.41	116.32 ± 26.73	0.984
HDL (mg/dL)	42.45 ± 8.09	44.90 ± 9.75	0.357
Cholesterol (mg/dL)	193.00 ± 64.52	186.47 ± 65.82	0.730
Triglyceride (mg/dL)	118.05 ± 35.37	113.43 ± 24.15	0.588

$p < 0.05$ significant

Table 3: Carotid artery stenosis in patients with lacunar infarcts

Duplex scanning of carotid artery in lacunar infarct patients	*No.*	*% age*
Stenosis absent	13	65.0
Stenosis present	7	35.0
Total	20	100.0

Table 4: Percentage of carotid artery stenosis in patients of lacunar and non-lacunar infarcts

Percentage of carotid artery stenosis	*Lacunar (n = 20)*	*Non-lacunar (n = 30)*
1–40	3 (15%)	6 (20%)
41–70	3 (15%)	4 (13.3%)
>70	1 (5%)	3 (10%)
No stenosis	13 (65%)	17 (56.66%)

$p = 0.828$ (insignificant)
$p < 0.05$ significant

Table 5: Significant carotid artery stenosis in patients of lacunar and non-lacunar infarcts

Significant carotid artery stenosis (>70%)	*Lacunar (n = 20)*	*Non-lacunar (n = 30)*
Present	1 (5%)	3 (10%)
Absent	19 (95%)	27 (90%)

$p = 0.523$

Approximately, 20% of all ischemic strokes are considered to be lacunar, and they tend to reoccur. Based on pathological assessment of early cases, such infarcts were believed to reflect primary arterial changes in these small vessels, but recent reports of patients suggest additional mechanisms. The original descriptions of lacunar stroke have been expanded to include a number of additional clinical syndromes. With the advent of CT and without extensive pathological confirmation, the label of lacunar infarct is now widely applied to any small deep infarct. A diagnosis of lacunar infarct frequently implies a specific pathophysiological mechanism, precluding further investigation for other possible cerebrovascular causes.

In our study, among the 50 patients included, 40% (n = 20) patients showed lacunar infarct, while 60% (n = 30) of the patients had non-lacunar infarct. Out of the 20 (40%) patients with lacunar infarct, 2 (10%) were less than 50 years, 14 (70%) between the age of 51 and 70 years and 4 (20%) with age greater than 70 years. This group included 9 (45%) female and 11 (55%) male patients. Similarly, in patients with non-lacunar infarct, 7 (23.33%) were with age less than 50, 19 (63.33%) between the age 51 and 70, and 4 (13.33%) greater than 70 years. In this group, there were 9 (30%) females and 21 (70%) males. In a study conducted by Intizari[7] on 1,158 patients with hemispheric stroke, 493 (42.5%) had features of lacunar stroke which is similar to our study. An analysis done on 893 patients of ischemic stroke in the stroke registry of Nizam's Institute of Medical Sciences, Hyderabad,[8] showed 16% of stroke patients with lacunar infarction. In contrast to our results, a study conducted on stroke in south Alabama,[9] lacunar infarct composed of 13% of stroke patients. The higher incidence of lacunar infarction in our study was probably due to a small number of patients in the study.

Nine (18%) patients were alcoholic out of the 50 patients and all of them were males, with 2 patients less than 50 years and 7 patients with an age group of 51 to 70 years. This result was not statistically significant (p value = 0.274). Out of these 9 patients, four (20%) had lacunar infarct and five (16.67%) had non-lacunar infarct (X^2: 0.990; p value = 0.764). In our study, 7 (14%) patients were smokers. Results showed that out of the seven smokers, four (23%) were in the lacunar stroke group and three (10%) in the non-lacunar stroke group. A higher percentage of smokers were found in patients with lacunar stroke, although the results were not statistically significant (p value = 0.318). In the present study, 16% (n = 8) patients were diabetic. All the 50 patients who presented to us with stroke had a history of hypertension and were on antihypertensives. Among the 20 patients with lacunar infarcts, 14 (70%) were hypertensive and, among 30 patients with non-lacunar infarcts, 21 (70%) patients were hypertensive at admission. So the same percentage of patients was found to be hypertensive in both groups. The mean value of systolic blood pressure in lacunar and non-lacunar infarcts in our study was 172.10 ± 28.33 mm Hg and 174.73 ± 30.26 mm Hg, respectively, with no statistically significant difference (p value = 0.759). The mean value of diastolic blood pressure in lacunar and non-lacunar infarcts in our study was 99.75 ± 14.89 mm Hg and 103.93 ± 13.92 mm Hg, respectively, with no statistically significant difference (p value = 0.316). In our study, 78% patients (n = 39) had hypercholesterolemia and triglyceride levels were high in 18% (n = 9) of the patients. On the one hand, out of 20 patients with lacunar infarct, 15 (75%) patients had high cholesterol level, 5 patients (25%) had high triglyceride level, 16 (80%) had low HDL levels, and 5 (25%) patients had high LDL levels. On the other hand, in 30 patients with non-lacunar infarcts, 24 (80%) had high cholesterol levels, 4 (13.33%) patients with high triglyceride levels, 22 (73.33%) with low HDL levels, and 5 (16.67%) patients with high LDL levels. The results were not statistically significant as p value was 0.730, 0.558, 0.357, and 0.984 for cholesterol, triglyceride HDL, and LDL levels, respectively, between the two groups.

In our study, 20 (40%) out of the 50 acute ischemic stroke patients had carotid artery stenosis. In a study conducted by Avishek et al.[10] showed 71% of prevalence of high carotid intima media thickness in patients of cerebral ischemic stroke. In another study conducted by Fernandes et al.[11] showed 72% of the patients with high

carotid intima media thickness (0.8 mm) in patients with acute ischemic stroke. In our study, 35% of the lacunar infarct patients had carotid stenosis. Out of 20 patients who had lacunar infarct in the MRI, 3 (15%) patients had less than 40% of carotid artery stenosis, 3 (15%) cases had 41 to 70% of carotid artery stenosis while only 1 (5%) patient had more than 70% carotid artery stenosis. 13 (65%) patients had no carotid artery stenosis. A similar study conducted by Inzitari[7] on patients with carotid artery stenosis presenting with lacunar stroke suggested that it was more commonly associated with milder (<50%) degrees of carotid artery stenosis. In a study conducted by Tegeler,[12] the prevalence of important extracranial carotid stenosis (greater than or equal to 50% diameter reduction) in the lacunar stroke group was 13% (seven of 55) in the ipsilateral and 4% (two of 55) in the contralateral carotid artery. In 30 patients of non-lacunar infarct, 6 (20%) cases had less than 40% of carotid artery stenosis, 4 (13.3%) patients had 40 to 70% of carotid artery stenosis, and 3 (10%) patients had carotid artery stenosis more than 70%. No carotid artery stenosis was found in 17 (56.66%) patients. This observation was found to be statistically insignificant (p value > 0.05).

According to the NASCET classification, more than 70% of the carotid artery stenosis is considered significant. Out of 20 patients with lacunar infarct, only 1 (5%) had significant carotid artery stenosis (>70%). In 30 patients with non-lacunar infarcts, 3 (10%) had significant carotid artery stenosis. It was seen that significant carotid artery stenosis was more common in non-lacunar infarcts as compared with that of patients with lacunar infarct, although there was no statistically significant difference between two groups (p value > 0.05). A similar study conducted by Warlow[13] on 726 patients with anterior circulation transient ischemic events or infarcts revealed a negative association of carotid stenosis with lacunar infarction. Mead et al.[15] studied 259 patients with recent lacunar stroke without prior other stroke and found that there was no difference between the severity of ipsilateral and contralateral carotid artery disease. An observational study of NASCET data showed that with 70 to 99% of carotid artery stenosis, lacunar infarcts composed of just 21.6% of the total number of infarcts. Homburg et al.[14] recently published the first study to actually look at the relationship between atherosclerotic carotid plaque rupture and stroke subtypes. In this study, 750 patients with anterior circulation stroke symptoms were evaluated for the presence of atherosclerotic plaque rupture in symptomatic carotid artery using multi-detector CT angiography. Plaque ulcerations were found to be independently associated with non-lacunar stroke compared with lacunar stroke.

CONCLUSION

The findings in our study are consistent with the hypothesis that severe carotid artery stenosis in lacunar infarction is an incidental finding. So data from our study suggest that lacunar strokes may not warrant investigations for carotid stenosis. Hence, routinely evaluating for carotid artery disease is not required in patients presenting with radiologically confirmed lacunar infarcts.

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