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Clinical Profile and Localization of Lesions in Patients with Lacunar Stroke in a Tertiary Care Hospital

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Abstract

Background: Cerebral infarction of the lacunar type is also known as lacuna, which means cavity, hole or a small size. Dechambre first used the term “lacuna” in 1838 to describe softening in subcortical region of the brain found on autopsy[1]. Lacunar infarcts cause a clinically evident stroke when they occur at strategic sites where descending and ascending long tracts are concentrated in their course, subcortically or in the brainstem.

Method: A prospective observational study of patients with lacunar stroke was conducted over two years in Department of Medicine, Govt. Medical College, Srinagar. Detailed history, examination and investigations were done.

Results: Out of 92 patients, 52 had pure motor stroke and rest had either pure sensory stroke, sensori-motor or ataxic hemiparesis. The most common site of infarct was posterior limb of internal capsule.

Conclusion: Lacunar stroke is an ischemic stroke caused by various risk factors like smoking, hypertension, diabetes, dyslipidemia and has varied presentations.

Keywords: Lacunar stroke, dyslipidemia, subcortical, pure motor.

Introduction

A restricted area of infarction (2-20 mm) due to ischemia in the territory of a single perforating artery causes the classical ischemic lacune.[1] Lacunar strokes constitute a quarter of all ischemic strokes, and are generally attributed to lipohyalinosis.[2,3] The common lacunar syndromes (pure motor, sensori-motor, pure sensory, ataxia-hemiparesis, dysarthria-clumsy hand) can each be caused by lesions in different cerebral / brainstem areas, from anterior or posterior circulation territories.[1] Lacunes in lentiform nucleus may present as gradual cognitive decline.[4] The findings of widespread, ‘same-age’ lacunes and cortical lacunes, leads to consideration of embolisation as the etiology of these lacunes.[5,6] Although a recognized stroke subtype for over 50 years, the cause of lacunar ischaemic stroke[7] and whether it is different to cortical ischaemic stroke, remains under debate.[5,6] Furthermore, lacunar stroke is not benign; 30% of patients are left dependent, [8] and scant long term data suggest that up to 25% of patients have a second stroke within 5 years.[9] Therefore, the prevention and treatment of this common stroke subtype may be less than ideal.

As a general rule, lacunar syndromes lack findings such as aphasia, agnosia, neglect, apraxia, or hemianopsia (so-called "cortical" signs). Monoplegia,

stupor, coma, loss of consciousness, and seizures also are typically absent. Asymptomatic lacunar infarctions generally occur in hypertensive patients, and are usually multiple and due to lipohyalinosis. Silent lacunes are the most common clinical form, accounting for 52% of cases in a clinicopathological series[10] and 77% of cases in the series of Fisher.[11]

Lacunar infarcts may present as TIAs, that is focal neurological deficits lasting less than 1 h. Using the classical definition of TIA 'a cerebral dysfunction of an ischemic nature lasting no longer than 24 h', lacunar infarctions may account for 29–34% of all TIAs.[12,13,14] More than 20 lacunar syndromes have been described. Five have been validated as being highly predictive for the presence of lacunes radiologically: Clinical syndromes in patients with lacunar stroke include pure motor hemiparesis, pure sensory syndrome, sensorimotor stroke, ataxic hemiparesis and dysarthria–clumsy hand, in decreasing order.[15,16]

The diagnosis of lacunar infarction in vivo relies upon finding a clinical syndrome that is consistent with the location of a small noncortical infarct seen on CT or MRI. Brain imaging with CT or MRI is also useful to exclude other potentially life-threatening diagnoses such as intracerebral hemorrhage or subdural hematoma. Investigation of the underlying stroke mechanism (e.g, thrombosis versus embolism) is still indicated to exclude potentially correctable causes of recurrence. Currently, MRI is the imaging technique of choice for the demonstration of lacunar infarcts. In a study carried out in 227 patients with lacunar stroke, MRI yielded positive findings in 78% of cases compared with 44% for CT scan. MRI was significantly better than CT for imaging lacunes, especially those located in the pons or the internal capsule[17]. Occasionally, the use of an intravenous contrast agent (gadolinium) allows differentiation of recent cerebral infarct from an old or residual ischemic infarction.[18] The most sensitive and specific imaging method to detect acute subcortical ischemic lesions is diffusion weighted imaging (DWI), which can differentiate acute from non-acute lesions.

The initial mortality rate in lacunar infarction is low (case–fatality ratio of 0–2% at 30 days), recovery of deficits is generally good in the first few weeks after onset, and the risk of early stroke recurrence is also low (average rate of recurrent stroke of 7.7%; range 2–12%).[16,19,20,21] For this reason, lacunar infarction has been traditionally considered a 'benign' or 'innocent' vascular entity in comparison with other subtypes of cerebral infarction with higher in-hospital mortality, greater neurological deficit and higher rate of stroke recurrence. The short-term prognosis of lacunar infarction is favorable because in-hospital mortality rate is very low and case–fatality at 1 year is less than 2.8%, a similar percentage to that of the general population.[16]

Materials And Methods

A prospective observational study of patients with lacunar stroke was conducted over a period of 2 years in Department of Medicine, Govt. Medical College, Srinagar.

After obtaining written informed consent and ethical clearance from institutional ethics committee, patients who developed lacunar stroke were analyzed.

Inclusion Criteria

1. All age groups with lacunar stroke irrespective of sex and ethnicity were included in the study.
2. Patients with old ischemic strokes, now presenting with lacunar stroke were also included in the study.

Exclusion Criteria

All patients with hemorrhagic stroke were excluded from the study

For each case, a detailed history, thorough physical examination was recorded. Relevant investigations such as computed tomography and magnetic resonance imaging were done in addition to complete blood counts, kidney function tests, liver function tests, lipid profile and Serum electrolytes.

Lacunar stroke was diagnosed as small noncortical infarcts made in setting of appropriate clinical syndromes and radiological tests.

Results

A total of 95 patients with lacunar stroke were included in the study. The mean age of presentation was 69.27 years. Lacunar stroke was most common in age group of 65 to 80 which constituted 61 patients (64.2 percent).

Table 1: Age distribution

Age group	Frequency	Percent
50-64	24	25.3
65-80	61	64.2
>80	10	10.5
Total	**95**	**100.0**

Out of the 95 patients, we found that 57 patients (52.29%) were males and 38 (47.71%) were females

Overall smoking was significant in 51 (53.68%) patients, hypertension in 64 (67.37%), diabetes in 36 (37.89%), atrial fibrillation (AF) in 19 (20%), dyslipidemia in 29 (30.53%), and ischemic heart disease (IHD) in 25 (26.32%) patients.

Table 2: Etiologic factors

Etiologic factor		Frequency	Percent
Smoking	Yes	51	53.68
	No	44	46.32
Hypertension	Yes	64	67.37
	No	31	32.63
DM	Yes	36	37.89
	No	59	62.11
AF	Yes	19	20.00
	No	76	80.00
Dyslipidemia	Yes	29	30.53
	No	66	69.47
IHD	Yes	25	26.32
	No	70	73.68

Out of total 95 study subjects 52 (54.7%) patients had pure motor stroke, 14 (14.7%) had sensorimotor stroke, 13 (13.7%) had pure sensory stroke, 12 (12.6%) had ataxic hemiparesis and 4 (4.2%) had dysarthria clumsy hand syndrome.

Table 3: Lacunar stroke syndromes

Lacunar stroke	Frequency	Percent
Pure motor type	52	54.74
Sensorimotor type	14	14.74
Pure sensory type	13	13.68
Ataxic hemiparesis type	12	12.63
Dysarthria-clumsy hand syndrome	4	4.21
Total	95	100.0

Internal capsule was the most common site of lacunar infarcts with clinically significant lacunar syndromes.35 (37%) patients had lesion in posterior limb internal capsule, 16 (17%) had lacunar infarct in other regions of internal capsule, 16 (17%) had lesion in thalamus,

11(11%) had infarct location in posterolateral thalamus, 14 (15%) had pontine infarct and 3 (3%) had infarct in corona radiate.

Table 4: Infarct location on imaging

Infarct location on imaging	Frequency	Percent
Posterior limb internal capsule	35	37
Thalamic infarct	16	17
Internal capsule	16	17
Pontine infarct	14	15
Posterolateral thalamus	11	11
Corona radiata infarct	3	3
Total	95	100.0

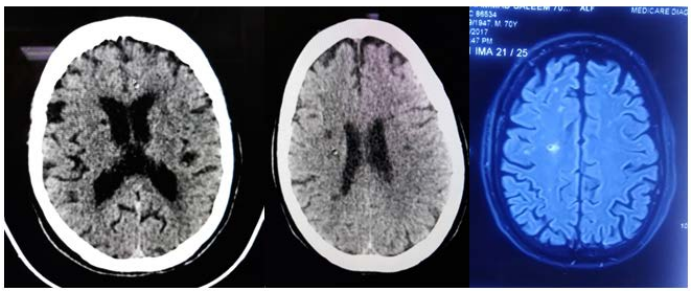

Figure 1 : CT and MRI showing lacunar infarcts

Discussion

Lacunar stroke constitutes a homogeneous subgroup of cerebral infarctions due to the small size of the lesion (maximal diameter <20 mm) and favorable functional prognosis at hospital discharge, characterized by a lower in-hospital mortality and functional deficit compared with other types of ischemic infarctions, such as those of cardioembolic or atherothrombotic cause.[22,23]

The present study was done to detect clinical profile, risk factors and localization of lacunar stroke. The established risk factors for lacunar infarcts are age, sex, hypertension, diabetes mellitus, ischemic heart disease, TIA, and current cigarette smoking. In our study, the frequencies of these risk factors are very similar to data collected in previous studies.[24]

In our hospital based study which included 95 subjects with lacunar stroke, we found 60 subjects belonging to age group of 65 to 80 years, which constituted 63.15 % of the study subjects. Of the rest 24 belonged to age group of 50-64 and rest 11 above 80 years. Overall the mean age of presentation was 69.27 years. This is similar to findings of other studies. The more advanced age coincides with results reported by Asplund et al.[25] with a mean age of 66.5 years in male and 73.2 in female patients. Advancing age is an important risk factor for lacunar infarction.[26,27,28,29,30] In a study conducted by Gérard Besson et al. the mean age of presentation was 69.7 years for pure motor stroke, 67.5 years for pure sensory stroke, 59.8 years for sensory motor stroke and 65.7 years for ataxic hemiparesis.[2]

In our study 52 study subjects were smokers which constituted 54.73% of the study subjects. In our study 78% of the study population had a smoking history of more than 10 year duration. Smoking is a risk factor for lacunar infarction, with incidence rates between 28[31] and 68%.[32] In a study conducted by C. Gandolfo et al.[33] smoking increased the risk of lacunar infarction by 2.3 times where as in the study conducted by R.You et al.[34] smoking increased the risk by 6.6-times. In a study conducted by Uma Sundar and Vijay Ghuge, smoking was a significant risk factor in 62% of cases with lacunar stroke.[35]

In our study, 65 study subjects were found to have a history of hypertension which account for 68.42 % of the patients. Of the total 65 hypertensive patients 42 had uncontrolled levels of blood pressures which account for

64% of the hypertensives. In a study conducted by Adria` Arboix et al. where the study cohorts of patients with lacunar stroke included 51 patients aged <55 years and 813 aged >55 years. Hypertension was significant in 32 (62.7%) patients below age 55 and in 586 (72.1%) patients above age 55 years.[36]

In our study 36 study subjects were found to have diabetes mellitus (DM) which comprised of 37% of study subjects. The prevalence of stroke was higher in subjects with greater duration of DM. 25(71%) diabetics had more than 10 year history of diabetes, 9 (25%) had 5 to 10 year history and 2 (4%) had history of less than 5 years. A. Arboix et al. conducted a study where they prospectively analyzed 286 consecutive patients with lacunar syndromes. Diabetes was present in 28% of patients.[37]

In our study which included 95 patients with radiologically proven lacunar infarct, the pure motor stroke was found in 52 (54.7%) patients, pure sensory type in 13(13.7%) patients, sensorimotor type in 14 (14.7%) patients, and ataxic hemiparesis in 12 (12.6%) patients and dysarthria clumsy-hand in 4 (4.2%) patients. The most common site of infarct was the posterior limb of internal capsule. It was found in 35(37%) of patients. Other sites of lacunar infarct were thalamus in 16(17%) patients, internal capsule (anterior limb and genu) in 16(17%) patients, Posterolateral thalamus in 11(11%) patients, and pons in 14 (15%) patients and corona radiata in 3 (3%) patients. In a study conducted by Uma Sundar, Vijay Ghuge**,** pure motor stroke (PMS- 58/82-70.7%) was the commonest lacunar syndrome, with the lesion in the internal capsule (IC-31/58- 53.4%). Pontine strokes accounting for the PMS was seen in 6 patients, and a combination of pontine with capsular strokes in 3 patients. Clear localization to carotid territory was seen in 49/58 patients (84.4%), localization to vertebrobasilar territory in 6/58 patients (10.3%), and localization to both in 3/58 patients (5.1%).Multiple simultaneous lacunes were seen in 8 patients with PMS, 5 having an ipsilateral small cortical lacune, and 3 having an ipsilateral lacune (2 pontine). Sensorimotor stroke was seen in 12/82 (14.6%) patients, half of them having the lesion in contralateral thalamus. 'Ataxia hemiparesis' (AH) was diagnosed in 5 patients, and 'dysarthria-clumsy hand' in 3 patients where the lesions were found in ganglionocapsular region and Internal Capsule.[35]

Conclusion

Lacunar stroke is an ischemic stroke caused by factors like smoking, hypertension, diabetes, Dyslipidemia and has varied presentations. This study helped in understanding clinicoetiological profile in lacunar stroke. More studies need to be done to understand pathophysiological aspects of the lacunar strokes.

References

1. Fisher CM. Lacunar strokes and infarcts: a review. Neurology 1982; 32:871.
2. Besson G, Hommel M, Perret J. Risk factors for lacunar infarcts. Cerebrovasc Dis 2000; 10:387-390.
3. Chen X, Wen W, Anstey K, Sachdev PS. Prevalence, incidence and risk factors of lacunar infarcts in a community sample. Neurology 2009; 73:266-272.
4. Voisin T, Rous de Feneyrols AR, Pavy Le Traon A, et al. Cognitive impairment after first lacunar stroke: clinical features and risk factors. Cerebrovasc Dis 2002; 13(suppl 3) 297.
5. Futrell N. Lacunar infarction. Embolism is the key. Stroke 2004; 35:1778–9.
6. Norrving B. Lacunar infarction. Embolism is the key: Stroke 2004; 35:1779–80
7. Bamford JM, Warlow CP. Evolution and testing of the lacunar hypothesis. Stroke 1988; 19:1074.

8. Bamford J, Sandercock P, Dennis M, et al. Classification and natural history of clinically identifiable subtypes of cerebral infarction. Lancet 1991; 337:1521–6.
9. Samuelsson M, Soderfeldt B, Olsson GB. Functional outcome in patients with lacunar infarction. Stroke 1996; 27:842–6.
10. Arboix A, Ferrer I, Martí-Vilalta JL. Clinico-pathologic analysis of 25 patients with lacunar infarcts. Rev. Clin. Esp. 1996; 196: 370–374.
11. Fisher CM. Lacunes: small, deep cerebral infarcts. Neurology 1965; 15: 774–784.
12. Arboix A, Martí-Vilalta JL. Transient ischemic attacks in lacunar infarcts. Cerebrovasc. Dis. 1991; 1: 20–24.
13. Vaxman SG, Toole JF. Temporal profile resembling TIA in the setting of cerebral infarction. Stroke 1983; 14: 433–437.
14. Kappelle LJ, van Latum JC, Koudstaal PJ, van Gijn J for the Dutch TIA Study Group. Transient ischaemic attacks and small vessel disease. Lancet 1991; 337: 339–341.
15. Fisher CM. Lacunar infarcts. A review. Cerebrovasc. Dis. 1991; 1: 311–320.
16. Martí-Vilalta JL, Arboix A, Mohr JP. Lacunes. In: Stroke. Pathophysiology, Diagnosis, and Management. Mohr JP, Choi DW, Grotta JC, Weir B, Wolf PhA (Eds). Churchill–Livingstone, PA, USA 275–299 (2004).
17. Arboix A, Martí Vilalta JL, García JH. Clinical study of 227 patients with lacunar infarcts. Stroke 1990; 21: 842–847.
18. Arboix A, Martí-Vilalta JL, Pujol J, Sanz M. Lacunar cerebral infarct and nuclear magnetic resonance. A review of sixty cases. Eur. Neurol. 1990; 30: 47–51.
19. Norrving B. Long-term prognosis after lacunar infarction. Lancet Neurol. 2003; 2: 238–245.
20. Baumgartner RW, Sidler C, Mosso M, Georgiadis D. Ischemic lacunar stroke in patients with and without potential mechanism other than small-artery disease. Stroke 2003; 34:653.
21. Jackson C, Sudlow C. Are lacunar strokes really different? A systematic review of difference in risk factor profiles between lacunar and nonlacunar infarcts. Stroke 2005; 36: 891–901.
22. Arboix A, Martı´-Vilalta JL. Lacunar stroke. Expert Review of Neurotherapeutics, 2009; 9: 179-96.
23. Rundek T, Sacco RL, Mohr JP, Wof PhA, Grotta JC, Moskowitz MA, Mayberg MR, von Kummer R, .Prognosis after stroke. Editors Stroke Pathophysiology, diagnosis, and management. Elsevier Saunders; Philadelphia: 2011. p. 219-41.
24. Lodder J, Boiten J. Incidence, natural history, and risk factors in lacunar infarction. Adv Neurol 1993; 62: 213–227.
25. Asplund K, Ha¨gg E, Helmers C, et al. The natural history of stroke in diabetic patients. Acta Med Scand 1980; 207: 417-24.
26. Wolf PH, Kannel WB. Epidemiology of Stroke. In: Mohr JP, Wof PH, Grotta JC, Moskowitz MA, Mayberg MR, von Kummer R, Editors Stroke Pathophysiology, diagnosis, and management. Elsevier Saunders; Philadelphia: 2011. p. 198-218.
27. Gorsselink EL, Peeters HPM, Lodders J: Causes of small deep infarcts detected by CT. Clin Neurol Neurosurg 1984; 86: 271-273.
28. Bamford J, Sandercock P, Jones L, Warlow C. The natural history of lacunar infarction: the Oxfordshire Community Stroke Project. Stroke 1987; 18:545.

29. Bogousslavsky J, Van Melle G, Regli F. The Laussane Stroke Registry: analysis of 1000 consecutive patients with first stroke. Stroke 1988; 19: 1083-1092.
30. Mohr JP, Caplan LR, Melski JW et al. The Harward Cooperative Stroke Registry: A prospective registry. Neurology 1978; 28: 754- 762.
31. Norrving B, Staaf G. Pure motor stroke from presumed lacunar infarct. Incidence, risk factors and initial course. Cerebrovasc. Dis. 1991; 1: 203–209.
32. Lodder J, Bamford JM, Sandercock PAG, Jones LN, Warlow CP. Are hypertension or cardiac embolism likely causes of lacunar infarction? Stroke 1990; 21: 375–381.
33. Gandolfo C, Caponnetto C, Del Sette M, Santoloci D, Loeb C. Risk factors in lacunar syndromes: a case–control study. Acta Neurol. Scand. 1998; 77: 22–26.
34. You R, McNeil JJ, O'Malley HM, Davis SM, Donnan JA. Risk factors for lacunar infarction syndromes. Neurology 1995; 45: 1483–1487.
35. Uma Sundar, Vijay Ghuge, Lacunar Syndromes – Where is the Lesion? JAPI 2015; 63(6): 41-4.
36. Arboix A, Estevez S, Rouco R, Oliveres M, Garcı´a-Eroles L, and Massons J. Clinical characteristics of acute lacunar stroke in young adults. Expert Review of Neurotherapeutics, May 2015.
37. Arboix A, Marti-Vilaltab JL. Lacunar syndromes not due to lacunar infarcts. Ccrcbrovasc Dis 1992; 2: 287-292.