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RESEARCH ARTICLE

LACUNAR INFARCTS – 'THE BLACKHOLES OF THE BRAIN' A STUDY ON ASSESSMENT OF VARIOUS RISK FACTORS, CLINICAL PRESENTATIONS AND RADIOLOGICAL CORRELATION OF LACUNAR INFARCTS

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ABSTRACT

Background: This study was aimed to evaluate various clinical presentations of lacunar infarcts and correlating their localization with Radiological imaging
Method: This Cross sectional study was conducted on 100 patients who admitted and attended at Dr. PSIMS & amp; RF which is a rural tertiary health care centre from July 2015 to November 2016. Patients presenting with various clinical Lacunar syndromes, were evaluated for etiopathogenesis, confirming the lesion localization with radiological imaging.
Results: 100 patients were studied out of which 83 were hypertensives and 63 were smokers, 56 were diabetics. 63 patients were asymptomatic and 37 were symptomatic pure motor hemiparesis was seen 22 in symptomatic patients, most of them located in internal capsule region on radiological localization. Sensorimotor lacunar syndrome was seen in 10 patients, half of them having a thalamic lesion. 3 patients with ataxia hemiparesis had lesion at cortical and capsule ganglionic regions. 6 patients have lesions at multiple sites' had lesions mostly located in cortical regions with different presentations.
Conclusion: In our study, Hypertension and smoking were two most common risk factors for Lacunar infarct. Many of launar infarcts were asymptomatic. Pure motor hemiparesis is the most common type of lacunar syndrome with lesions commonly localized in internal capsule region.

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INTRODUCTION

Lacunar infarcts are small infarcts (2–20 mm in diameter) in the deep cerebral white matter, basal ganglia, or pons, presumed to result from the occlusion of a single small perforating artery supplying the subcortical areas of the brain (Bamford and Warlow, 1988), constituting a quarter (25%) of all ischemic strokes (Besson *et al.,* 2000; Chen *et al .,* 2009) . Although a recognised stroke subtype for over 50 years, the cause of lacunar ischaemic stroke, and whether it is different to cortical ischaemic stroke, remains under debate (Futrell, 2004; Norrving, 2004). Although occasionally result from mechanisms of brain ischemia such as cardiogenic embolism or carotid-artery stenosis, most result from intrinsic disease of the small penetrating arteries (Boiten and Lodder, 1993; Ross *et al.,* 1999; Tatemichi *et al.,* 1993). Two major vascular pathologies underlie brain damage in patients who have abnormalities involving small size penetrating cerebral arteries and arterioles are thickening of the arterial media with encroachment on the arterial lumens and obstruction of the origins of penetrating arteries by parent large intracranial artery intimal plaques resulting in lipohyalinosis mostly attributing secondary to Hypertension, diabetes, smoking and other risk factors (Wardlaw *et al.,* 2003; Louis, 2015). The global prevalence of hypertension and diabetes has risen dramatically over the past three decades, about 29 per cent of strokes are attributable to hypertension underlining the huge impact effective hypertension prevention and control can have on reducing the rising burden of cerebro vascular disease (Devi *et al.,* 2011). It is estimated that a 2-mmHg decrease in blood pressure (BP) population wide such as that easily achievable by modest salt reduction, can prevent 151,000 strokes deaths in India with larger blood pressure decreases yielding higher reductions (Rodgers *et al.,* 2000; Mohan and Prabhakaran, 2012). The findings of wide spread, multiple 'same-age' lacunes, and cortical lacunes, leads to consideration of embolization as the etiology of these lacunes (Samuelsson *et al.,* 1996). Recent studies have shown that the prognosis after lacunar infarcts is not benign; the risk of recurrent stroke is no lower than for other ischemic stroke subtypes, up to 25% of patients have a second stroke within 5 years. (Arboix *et al.,*

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2015). Recurrent lacunar ischemic stroke may be associated with a more severe clinical picture and it is one of the major factors involved in producing lacunar state and vascular subcortical dementia (Jacova *et al.,* 2012). Lacunes determine longitudinal cognitive impairment in small vessel disease. Although the individual contribution of lacunes on cognition was modest, they cannot be considered benign findings, but indicate a risk of progressive cognitive impairment (Voisin *et al.,* 2002). These observations suggest that most lacunar strokes are the clinically focal manifestation of what is actually a diffuse abnormality of the small cerebral arterioles, which, if extensive enough, can also manifest clinically as cognitive decline and dementia. In the acute phase, response to intravenous thrombolysis appears to be similar to other subtypes of ischemic strokes (Warlow *et al.,* 2013; Do Lacunar Strokes Benefit from Thrombolysis, 2013). Hence "Black holes in the brain" can no longer be considered as benign in view of their recurrence and their effect on future cognitive and execution dysfunction. Hence a detailed study on lacunar infarcts is needed for assessing the risk factors, clinical presentation in relation to their radiological localization, management and prognostic implications in the near future

MATERIALS AND METHODS

This study was carried out at the Dr. Pinnamaneni Siddhartha Institute of Medical Sciences and Research Foundation (which is a tertiary care, teaching hospital which extends health care facilities to the rural population. The patients who attended the Department of general Medicine and neurology during July 2015 to November 2016, 100 patients were included in the study. Serially recruited patients with lacunar infarcts, detected by CT/MRI imaging to have an infarct of 2 cm size or less, were evaluated for demographic and epidemiological factors, presence or absence of risk factors, their clinical presentations along with localization on imaging and the presence of additional simultaneous lacunes. Infarcts that involved the cerebral cortex or extensive areas of the subcortical white matter or that were >2 cm in greatest diameter were excluded. The lacunar infarcts were localized by means of an atlas comparing anatomic slices with standard CT slices.' 5 We distinguished the following locations: internal capsule (anterior limb, posterior limb), corona radiata, lentiform nucleus, thalamus, and caudate nucleus. We distinguished four vascular territories using Damasio's brain templates16: medial striate arteries, lateral striate or lenticulostriate arteries, anterior choroidal artery, and thalamoperforant arteries. Infarct volume was estimated according to Nelson *et al.,* (1980). The length and width of the infarct was measured at right angles. The product was multiplied by thickness and number of affected slices and divided by 2. In asymptomatic lacunar infarction, the lacunar infarct was compatible with the clinical signs and symptoms, not pertaining to classical lacunar syndromes but presented with symptoms like headache, giddiness, neckpains etc. Symptomatic lacunar infarction was defined as a case of a lacunar syndrome in which CT findings were compatible with infarction due to occlusion of one single perforating artery, i.e., a subcortical, small, sharply marginated hypodense lesion with diameter smaller than 20 mm, or if no specific lesion was visible on CT. Lacunar syndrome (LI) was defined as one of the following syndromes: pure motor stroke (unilateral pure motor deficit involving at least two of three areas-face, arm and leg); pure sensory stroke (unilateral pure sensory deficit involving at least two of three areas face, arm and leg); ataxic hemiparesis (ipsilateral corticospinal and cerebellar-like dysfunction without other features clearly localising to the posterior circulation); dysarthria- clumsy hand syndrome (severe dysarthria with slight weakness and clumsiness of the hand).

RESULTS

Out of 100 patients included in the study 83 were hypertensives, 56 were diabetes, 63 were smokers, 24 were alcoholics, 14 had history of ischemic heart disease and 12 had both ischemic and Hypertensive Hear diseases. 8 had previous history of stroke, 6 had their family members suffered from stroke. Analysis of clinical features showed 63 were asymptomatic, 37 were symptomatic among which 22 had pure motor stroke, 10 had sensorimotor stroke, 3 had ataxia hemiparesis, 1 pure dysarthria, remaining 1 had dysarthria clumsy hand syndrome. Localization of symptomatic infarcts showed 20 patients had lesion in internal capsule region, 4 had cortical location of infarcts, 5 had infarcts in multiple sites, 6 had lesions in thalamus and 1 in brainstem

Table 1. Demographic factors

Parameter	No.of patients (Frequency)	Percentage
AGE		
<55	23	23
>55	77	77
SEX		
Male	52	52
Female	48	48
BMI		
<25	36	36
>25	64	64

Table 2. Risk factor profile in lacunar strokes (n=100)

Risk Factor	No.of patients (Frequency)	Percentage
Hypertension	83	83
Diabetes	56	56
Smoking	63	63
Alcohol	24	24
TIA	18	18
Past h/o stroke	8	08
Family h/o stroke	6	06
Ischemic heart disease	14	14
Hypertensive heart disease	15	15
Both ischemic and hypertensive heart disease	12	12

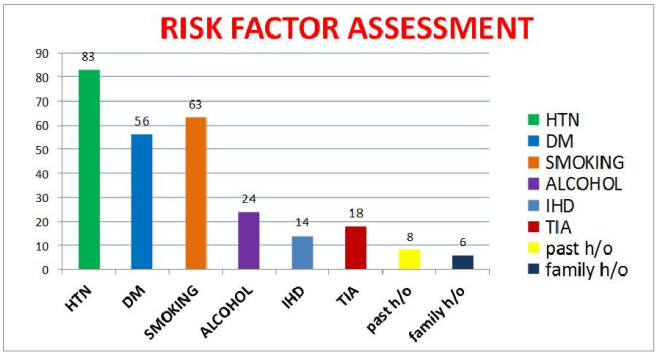

Table 3. Clinical presentations of various lacunar infarcts

Presentation	No.of patients	Percentage
Asymptomatic	63	63
Pure motor	22	22
Sensorimotor	10	10
Ataxia hemiparesis	03	03
Dysarthria clumsy hand	01	01
Pure dysarthria	01	01

Table 4. Radiological Localization of lacunar syndromes

	Capsulo gangionic	Corona radiata	Cortical	Multiple	Thalamic	Brainstem
Pure motor	14	2	1	5	--	--
Sensorimotor	1	1	1	1	06	--
Ataxia- hemiparesis	1	0	2	--	--	--
Dysarthria -clumsy hand	1	--	--	--	--	--
Pure dysarthria	--	--	--	--	--	01

DISCUSSION

As the prevalence of hypertension is being increased in developing countries like India which is the most common risk factor for lacunar infarcts as seen in present study (83%). The incidence of lacunar infarcts is also increasing with primary pathology being Lipo hyalinosis. Other risk factos like smoking (63%), diabetes, alcohol, old CAD increase the risk of lacunar infarct with advancing age and atheromatous vessel wall changes. Most of the lacunar infarcts are silent, with most of them are asymptomatic (63%), as in our studypresenting with non specific symptoms like headache, neck pains, giddiness etc. But 37 out of 100 patients presented with symptomatic lacunar infarcts with features of classical lacunar syndromes, pure motor stroke being the most common type identified in about 22 patients and most of them were located in the internal capsular region. In fact, more restricted deficits, such as face+arm, or arm+leg, localise to lacunes in the corona radiata or its junction with the internal capsule. Sensorimotor (SM) strokes, seen in 10 out of 100 patients in present series, have been attributed to lesions in the posterior limb of the Internal capsule interrupting the thalamo cortical fibres, or the thalamus medially. Six out of 100 patients had multiple infarcts, having cortical infarcts in addition to a more standard lacunar lesion. Multiple lesions were seen most often in pure motor stroke; 5/22 (23%) Pure mtor strokes having multiple lesions, and 5 of 6 'multiple lesions' belonging to pure motor category. Traditionally, lacunar strokes are not investigated aggressively with angiography, as the risk of recurrence is considered to be low, and the pathology, well-delineated.The importance of multiple lacunes, especially pertaining to 'possible' proximal embolic source (Wessels *et al.,* 2005; Takahashi *et al.,* 2002). Other authors have suggested that multiple lacunes represent a more severe small arterial disease. Hence "Black holes in the brain" can no longer be considered as benign in view of their recurrence and their effect on future cognitive and execution dysfunction. Interventions through life style modifications like cessation of smoking and alcohol, Dietary modifications low salt intake have a significant role in preventing hypertension and diabetes leading to reduction in the incidence of lacunar infarcts. Antiplatelet drugs, careful blood pressure control, statins and modification of lifestyle risk factors are key elements in secondary prevention after lacunar infarcts

Conclusion

In our study, Many of lacunar infarcts are asymptomatic. Hypertension and smoking were two most common risk factors for Lacunar infarcts. Pure motor hemiparesis is the most common type of classical lacunar syndrome in symptomatic patients with lesions commonly localized in internal capsule region.

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