HYPERTENSIVE BRAIN DISEASES.
III. LACUNAR SYNDROMES

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THE TERM lacunar stroke has been used to indicate small cavities in the deep cerebral tissue as early as 1843 and was revisited at the turn of the century in 1901 by Marie.1 It was not until recently that certain clinically separate lacunar syndromes were identified and their etiopathogenesis precisely described by Fisher. 2

The distinction of lacunar syndromes as an entity separate from other cerebrovascular strokes arises from: (1) their close relationship to arterial hypertension, (2) their restriction to a subcortical deep area in the territory of a single perforating (penetrating) artery, and (3) the typical lipohyalinosis found at autopsy. 2-4

**Definition**

A lacunar syndrome is defined as a constellation of symptoms and signs at the time of maximum deficit, caused by a single vascular event, which has been associated with restricted subcortical deep areas of infarction due to primary disease of a single perforating artery of the brain. 3-5 Therefore, symptoms and signs related to cerebral cortex like aphasia, apraxia, agnosia, vision-spatial deficit and hemianopia exclude the diagnosis of lacunar syndrome.

**Epidemiology**

Lacunar strokes account for 13% to 23% of all cerebral infarcts. 6-8 In the recently published Oxfordshire Community Stroke Project 1987, the incidence was 19.8% of all first ever strokes, and 24.6% of all cases of cerebral infarctions. 9 The incidence of lacunar infarcts was found to rise steeply with advancing age. The male-to-female ratio was reported to be equal in this study, but other studies reported male preponderance. 10,11 In a hospital stroke study at King Khalid University Hospital (KKUH) in Riyadh, the incidence of lacunar syndromes was 21%.11 A report from Japan gives the incidence of lacunes as high as 40% of all ischemic strokes. 12

**Etiopathogenesis**

Lacunar strokes are attributed to small perforating vessel disease. There is mainly segmental arterial disorganization with focal loss of the normal architecture of the vessel wall, and replacement by connective tissue, fatty macrophages, subintimal hyaline and fibrin containing material (lipohyalinosis).13 These eventually lead to occlusion of the vessel causing the lacunar stroke.

These changes do not necessarily occur at the bifurcation of the artery. In the proximal parts of the vessel supplying the lacunes, occlusion occurs by plaques of foam cells subsequently termed "microatheroma." 14 Lipohyalinosis is also associated with areas of focal dilatation and may be the underlying cause of Charcot-Bouchard aneurysm,15,16 though thrombosis within an embolus from these areas of dilatation might equally well occlude the vessel. The size of a lacunar infarct obviously depends upon the diameter of the affected vessel, ranging from 40 to 200 μm for small infarcts, and 300 to 500 μm for large lacunes.5
From the limited pathological evidence available, microatheroma seems to be the most frequent cause of single symptomatic lacunes. However, the exact relationship between these small-vessel arteriopathies and large-vessel atheromatous plaques remains uncertain. Mohr et al.6 reported that among patients undergoing angiography, 63% of those with lacunar syndromes were normal, compared with only 4% among those with cerebral thrombosis. Olsen et al.7 reported 73 consecutive and unselected patients with nonhemorrhagic infarcts, all of whom had angiogram and CT within 2 days from admission; virtually all cases with small deep infarcts on CT had minimal changes in their carotid arteries and had significantly less internal carotid disease than those with larger infarcts involving the cortex. Ringelstein et al.8 reported stenosing lesion in the extracranial vessels in 22% of patients with small deep infarcts compared with 71% in other types. However, in the series of Loeb et al,9 41% of lacunar cases and 69% of other ischemic cases had significant carotid artery stenosis or occlusion, and Donnan et al.10 reported ipsilateral carotid abnormalities in 35% of their lacunar infarcts.

**Relationship to Hypertension**

Microatheroma and lipohyalinosis have been considered to be the reaction of different parts of the arterial system to different grades and/or duration of hypertension.20 Fisher reported on 114 patients with lacunes. All except 3 were hypertensive. In other studies the frequency of hypertension in patients with lacunes ranges from 57% to 75%.6,10,21-23 Cole and Yates24 found a total of 128 lacunae in 23 hypertensive patients compared to 59 in normotensive groups. This may reflect the strict definition of hypertension adopted by some of these authors.

In our study at KKH hypertension was found in 75% of cases.11 Hughes25 hypothesized that hypertension leads to an elongation of the basilar artery with resultant displacement of the ostia of the small paramedian arteries. This would cause a retrograde blood flow and kinking of the fine vessels resulting in ischemia. He also postulated that coiling occurred in the small vessels which allowed the transmitted arterial pulsations to traumatize the surrounding neural tissue, thus leading to cavitation.

The absence of hypertension in 25% to 43% of patients suggests that hypertension cannot be the only cause of lacunar syndromes. Other causes such as embolism and carotid occlusive disease may play a role. Interestingly, these alternative causes are more commonly seen in large lacunar infarcts. Thus, it may be that emboli and carotid occlusive diseases tend to produce larger lacunae with more complex symptoms.3 This would be in accord with Fisher's findings of hypertension in 97% of patients with small frequent asymptomatic lacunae, whereas other series of larger, mostly symptomatic lesions report only 57% to 75%.6,10,22,23

Gorselink et al.26 reported 12% of cases with small deep infarcts as having potential cardioembolic source similar to 10% in the Harvard study, 2 though there were no such cases in Weisberg's study.23 Overall about 13% of lacunar infarcts will have a potential cardioembolic source,27 though as with angiographic findings may be coincidental and must not be assumed to have caused the small deep infarcts.28

**Clinical Picture**

There are five distinct lacunar syndromes: 

**Pure Motor Stroke:** Acute unilateral motor deficit involving face, arm and leg, or two of them (face and arm, arm and leg) without any sensory deficit, aphasia, agnosia, apraxia, or homonymous hemianopia. Subjective minor sensory symptoms may be present at the onset of the stroke but should not be found on objective clinical examination. The pure motor hemiparesis may develop after transient ischemic attacks (TIAs) or could be of sudden onset. Deep tendon reflexes may become quickly brisk and the plantar response is usually upgoing on the affected side. Monoplegia is usually not considered to be a lacunar syndrome. Abulia (lack of initiative) may also be an associated symptom. The infarct is topographically located at the posterior limb of the internal capsule, at the medullary pyramids or at the base of the pons (where the pyramidal tract converge together to form the pyramids).29 The occluded perforating artery is supplied either from the middle cerebral artery or from the lower basilar artery.

**Pure Sensory Stroke:** The syndrome takes the
form of numbness, dysesthesia, and paraesthesia which may occur in attacks (like TIA's) in the absence of motor weakness, aphasia, agnosia, or hemianopia. It involves the face, arm and leg on the same side, but may spare one of them. The sensory abnormality could even be diagnosed without clear-cut objective findings at the time of clinical examination. The sensory deficit may involve all modalities of sensation or may spare proprioception. The lesion is usually located at the posteroventral nucleus of the thalamus.

Sensorimotor Stroke: The symptoms comprise of motor weakness and objective sensory loss involving face, arm, and leg or two of them (brachiofacial or brachio-crural) on the same side, in the absence of aphasia, apraxia, agnosia, and hemianopia. The sensory loss could involve all modalities or may spare proprioception. Usually the whole limb is involved. The lacunae can be located at the internal capsule (thalamo-capsular) or corona radiata.

Ataxic Hemiparesis: In this syndrome motor hemiparesis is associated with cerebellar-like dysfunction of the affected limbs. The motor weakness is usually not so severe, so that the dysmetria of the limb could be detected on clinical examination. There may be some degree of dysarthria or mild nystagmus. Subjective sensory symptoms (numbness) may be present initially, but are not detected on clinical examination. The lesion is located at the base of the pons (where the pyramidal tract runs from the cerebral peduncles to condense later and from the pyramids), at the posterior limb of the internal capsule, or the adjacent corona radiata. Occlusion of the paramedian branches of the basilar artery causes these lacunar infarcts.

Dysarthria Clumsy Hand Syndrome: Sometimes this syndrome is considered as a part of the previous one (ataxic hemiparesis). There is slight weakness and clumsiness of the arm associated with weakness of the face on the same side, without any sensory loss. The clumsiness is apparent on action (taking a glass of water, writing, etc.). The speech is dysarthric. There are brisk, deep tendon reflexes, and an upgoing plantar on the same side. The lesion (i.e., the infarct) is located at the genu of the internal capsule or at the base of the pons.

Other lacunar syndromes have been suggested, including pure motor hemiparesis with contralateral gaze paresis, pure dysarthria, hemichorea, unilateral asterixis, and as many as 15 more with varying combination of brainstem syndromes. The utility of these is limited and they are uncommon and nonspecific for lacunar disease and in some cases lack adequate pathologic substantiation.

Status Lacunaris - Lacunar State
It is a clinical picture attributed to multiple lacunes characterized by chronic progressive neurological symptoms, eventually leading to dementia, pseudobulbar palsy, pyramidal and extrapyramidal signs, dysarthria, gait abnormalities, and incontinence. At postmortem examination, the brain is found to have multiple lacunae in the nuclei. The widespread use of antihypertensive drugs made this lacunar state less common.

Diagnosis
Diagnosis of lacunar syndrome depends upon the clinical picture of the five major syndromes described. However, neuroradiological techniques are invaluable in helping to establish the diagnosis. Of the brain is good in detecting small infarcts in the area of the internal capsule, but may sometimes fail to demonstrate small infarcts at the base of the pons. Magnetic resonance imaging (MRI) has been shown to be superior to CT scan in detecting lacunar infarcts and also useful in distinguishing between acute and chronic lesions. MRI can also detect a small, acute intracerebral hemorrhage, giving similar picture of a lacunar syndrome.

Management and Prognosis
Although lacunar infarcts as a separate subgroup constitute about one fifth of all strokes, the treatment remains basically the same as for other strokes, i.e., treatment and prophylaxis of the major risk factor, mainly hypertension, or any other associated risk factors. If the hypertension is not treated adequately, patients may eventually develop progressive neurological deterioration culminating in the status lacunaris with a variable degree of dementia.
Since the vascular process is lipohyalinosis which is typically associated with hemosiderininfilled macrophages, indicating slight extravasation of erythrocytes, heparin and warfarin may be harmful or even contraindicated, but aspirin and dipyridamole should be tried.

Patients with lacunar infarction are likely to survive their lacunar stroke. In most patients the prognosis for recovery from a lacunar deficit is excellent and a prolonged rehabilitation will be rewarding. Occasionally, pure motor stroke fails to follow the rule and about one third will be, to some extent, dependent on other persons in a year's time.

9 References