Lacunar Strokes: Current Concepts

Kent W. Davidson, M.D.

Abstract: Lacunar strokes result from occlusion of penetrating arteries in the deeper, subcortical parts of the cerebrum and brain stem. Approximately 19 percent of all strokes are of the lacunar variety with lacunar strokes representing the most common cerebrovascular complication of chronic hypertension. Four major clinical syndromes are pure motor hemiparesis, pure sensory stroke, ataxic hemiparesis, and the dysarthria-clumsy hand syndrome. The advent of computed tomography (CT) has allowed the antemortem study of lacunar disease and has shed new light on the pathogenesis and clinical course of lacunar strokes. Recently, it has been demonstrated that lacunar strokes may be embolic or hemorrhagic in causation, are not invariably associated with hypertension, and may be larger and associated with neurological manifestations that do not conform to the classic patterns. In most instances, however, recognition of the characteristic clinical presentation and confirmation of the diagnosis with noninvasive studies spare many patients unnecessary risks associated with anticoagulation, arteriography, or vascular surgery. (JABFP 1988; 1:57-62.)

It has been estimated that 19 percent of all strokes result from occlusion of penetrating arteries in the deeper, subcortical parts of the cerebrum and brain stem.[1] These cerebrovascular events are termed lacunar strokes in reference to the small cavities or lacunae that are left behind after infarction of brain tissue with subsequent cavitation.[2] The size of the infarct is generally small, in the range of 2 to 15 mm in diameter. The most common sites for lacunar infarction include the putamen, caudate, thalamus, pons, internal capsule, and convolutional white matter.[3] Lacunar disease is a common neurological sequela of chronic hypertension and frequently produces characteristic clinical syndromes. In many instances, through recognition of the characteristic clinical presentation and confirmation of the lacunar stroke with noninvasive studies, patients may be spared the unnecessary risk associated with anticoagulation, arteriography, or vascular surgery.[3]

Although the occurrence of lacunar infarctions was noted as early as 1901,[4] years passed until the pathological processes underlying the lesions and the clinical features of lacunar strokes were intensively investigated. The greatest contribution to modern understanding of lacunar strokes was made by Fisher and colleagues beginning in the mid-1960s.[5] Due to the small size of the infarcts and the lack of appropriate imaging equipment available at the time, Fisher's work was done through meticulous brain dissection. Although Fisher's contribution was considerable, several gaps in the understanding of lacunar disease resulted from the inherent shortcomings of postmortem histologic study. With the advent of computed tomography (CT), which facilitated the antemortem study of lacunar strokes, a new body of data has accumulated regarding the pathogenesis and the clinical features of lacunar disease. This new information has augmented rather than refuted Fisher's work and has provided a clearer understanding of lacunar disease and its management.

From the Department of Family and Community Medicine, College of Medicine, University of Arkansas for Medical Sciences, Little Rock. Address reprint requests to Kent W. Davidson, M.D., Department of Family and Community Medicine, College of Medicine, University of Arkansas for Medical Sciences, 4301 W. Markham—Slot #530, Little Rock, AR 72205.

Pathophysiology

The pathological lesions responsible for lacunar strokes vary depending on the size of the vessel involved and the size of the resulting infarct. Infarcts in the range 3-7 mm in diameter involve arteries 400 to 200 μm in diameter. The pathological process responsible for these tiny infarcts is called segmental arterial disorganization or lipohyalinosis and is thought to be the result of long-standing hypertension.[6,7] These vascular lesions result from thickening of the arterial wall through replacement of the normal muscular and elastic elements with connective tissue and giant macrophages. Eventually, the arterial lumen is occluded, and infarction occurs. Because of the small size of the lacunae resulting from this proc-

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ess, many of these infarcts are thought to remain asymptomatic.[8]

A second pathological process that involves larger arteries in the 400 to 900 μm range also results from chronic hypertension.[8,9] Atheromata that develop in the cerebral vessels of normotensive individuals usually occur in the extracranial distribution of the carotid and basilar arteries. In individuals with chronic hypertension, the atheromata are more widespread and, additionally, involve intracranial arteries and arterioles.[10] When these vessels become occluded due to atherosclerotic plaque, infarction results. Infarcts occurring via this process are generally larger and more symptomatic.

A third mechanism involves the obstruction of the penetrating artery due to atherosclerosis of the parent artery. Atherosclerotic plaques blocking the penetrating arteries from the middle cerebral and basilar arteries have been documented.[11,12] As would be expected, these infarcts are generally larger because the entire region of the brain supplied by the involved penetrating artery is affected.

In Fisher's work, the possibility of embolic phenomena from the heart or carotid systems causing lacunar strokes was postulated.[8] Additional data obtained through the study of patients with deep cerebral lesions utilizing CT scanning have confirmed this association.[13,14] It is thought the emboli block the origin of the penetrating arteries after lodging in their parent arteries and produce larger and more symptomatic lesions. This is similar to the third mechanism previously described. This mechanism is generally thought to be less common than the other three in the pathophysiology of classic lacunar stroke syndromes.

In each instance, obliteration of the lumen results in infarction of adjacent brain tissue. Macrophages eliminate the infarcted tissue, and the characteristic cavity or lacuna remains. In general, the smaller lacunae, 3-7 μL, result from segmental arterial disorganization and lipohyalinosis, whereas the larger lacunae, 1-2 cc, are the result of atheromatous or embolic occlusion of a penetrating vessel. The larger lacunae are usually symptomatic, whereas smaller lacunae may be asymptomatic unless situated in a sensory or motor tract.

At one time, lacunar strokes were thought to be virtually always associated with longstanding hypertension. Fisher reported a 97 percent incidence of hypertension in one series.[5] Others have reported the incidence of hypertension with lacunar infarction in only 57 percent to 65 percent of cases.[14,15] Reports noting higher associations between lacunar infarction and hypertension involve primarily patients with one of the classically described syndromes with limited neurological involvement and small deep infarcts.[16] Infarction in these cases was due to lipohyalinosis or microatheroma in a penetrating artery. In series with a lower association between hypertension and lacunar infarction, one usually finds larger lacunae with more complex neurological events and a higher frequency of emboli responsible for the infarct or hypoperfusion due to extensive extracranial atherosclerosis.[14,15]

Clinical Features

As noted earlier, many lacunar strokes are asymptomatic. This is because the vessels involved are of extremely small size, the area of infarcted tissue is small, and the infarction occurs in a relatively silent part of the brain. When lacunar strokes are symptomatic, however, they are often distinctive in their clinical presentations. Fisher originally described four clinical syndromes, referred to as lacunar syndromes, that he thought were specific to these infarcts. Subsequent to Fisher's original descriptions, there have been several other neurological syndromes described that have been attributed to lacunar infarction, although these cases are few in number and in some instances not confined to the deeper, noncortical parts of the brain.

There are a number of clinical features that are common to all of the lacunar syndromes. Lacunar stroke syndromes characteristically evolve in a "leisurely" fashion, with as many as 30 percent developing over a period up to 36 hours.[16] Lacunar syndromes characteristically do not embody certain neurological manifestations, including visual field defects, aphasia, stupor, coma, loss of consciousness, or seizures.[17] In only approximately 30 percent of patients with one of the lacunar syndromes, the stroke will be preceded by a transient ischemic attack.

Pure Motor Hemiparesis

The most common lacunar syndrome involves pure motor hemiparesis of the face, arm, and leg on one side of the body.[18] The stroke may involve various combinations of these three body parts, and the extent of the paralysis is variable. Dys-

arthria is frequently present with this syndrome. Occasionally, mild sensory symptoms occur without demonstrable impairment. This syndrome is notable for its lack of certain neurological manifestations, including visual field defects, aphasia, apraxia, cortical sensory deficits, ataxia, and cranial nerve abnormalities. Patients usually maintain full mental acuity. Increased deep tendon reflexes and an extensor plantar response are usually present on the hemiparetic side. The site of the lesion resulting in pure motor hemiparesis is in either the anterior or posterior limbs of the internal capsule, corona radiata, or in the base of the pons involving the corticospinal motor tract.

Pure Sensory Stroke

The second most frequent type of lacunar syndrome results in unilateral sensory symptoms.[19] In this variant, termed pure sensory stroke, there is numbness involving the face, arm, and leg on one side of the body. Axial structures including the scalp, neck, and trunk may also be involved, and the patient can have a sharp midline "plumb-bob" demarcation to the symptoms. The sensory symptoms may be described as tingling, numbness, heat or cold sensation, a pressure sensation, or alteration in size or weight of the involved regions. In general, the patient's sensory complaints are out of proportion to the objective findings noted on examination. Because tactile sensation is usually diminished rather than absent, Fisher suggested that a more appropriate designation for the syndrome would be "pure paresthetic stroke." This syndrome is unassociated with weakness, dysarthria, vertigo, diplopia, visual field defects, nystagmus, aphasia, or change in level of consciousness. The site of involvement with pure sensory strokes is in the sensory (postero-ventral) nucleus of the thalamus, and the most frequently responsible pathological process is lipohyalinosis.

Ataxic Hemiparesis

A third lacunar syndrome, also originally described by Fisher, is referred to as ataxic hemiparesis.[20] The site of this lacunar infarct is in the pons and results in ipsilateral extremity ataxia and hemiparesis of varying degrees on the side opposite the lesion. In most cases, the leg is involved more than the arm, and gait disturbance is a prominent symptom. Neurological exam will reveal dysmetria of the arm and leg with predominately a distal weakness on the same side with hyperreflexia and Babinski sign. Neurological signs characteristically absent include visual field defects, aphasia, intellectual impairment, dysarthria, dysphagia, and sensory loss.

Table 1. *Uncommon Lacunar Syndromes.*

Sensorimotor stroke
Generalized chorea
Ataxic tetraparesis
Bilateral paramedian thalamic infarction
Focal dystonia
Pure motor hemiparesis with contralateral gaze paresis
Isolated facial stroke
Pure dysarthria
Hemichorea-hemiballism
Unilateral asterixis

Dysarthria-Clumsy Hand Syndrome

A fourth common lacunar syndrome involves the combination of dysarthria and clumsiness of one hand.[21] With this syndrome, there is dysarthria, lower facial weakness on one side, and clumsiness with mild hand weakness on the same side. There may be associated dysphagia. The involved hand exhibits slight weakness with impairment in writing and fine movements. There are usually hyperactive reflexes of the involved upper extremity, and Babinski's sign may be present on that side. Visual field and cortical and cranial nerve deficits are not seen. The site of the lesion in this syndrome is in the pons or internal capsule.

Other Lacunar Syndromes

In addition to these four common syndromes, there have been a number of other less common lacunar syndromes described (see Table 1).[22-31] They are not associated with visual field defects, mental deficits, or other signs of cortical involvement. They usually occur in the setting of chronic hypertension and are associated with a good prognosis. The less common lacunar syndromes also typically evolve in a "leisurely" fashion rather than producing their neurological picture suddenly, as is characteristic of embolism, intracerebral hemorrhage, or subarachnoid hemorrhage.

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Diagnosis

Traditionally, the diagnosis of lacunar infarction has been made on clinical grounds through the recognition of the syndromes previously described.[1] This remains true in most cases of a classic lacunar syndrome. It should be noted, however, that in some series, the classic lacunar syndromes are manifested in only approximately 50 percent of cases,[15,32] with incomplete, complex, or atypical symptomatology present in the remainder of cases. Electroencephalography may be of some help in that it is usually normal with subcortical infarcts. Examination of the cerebrospinal fluid is generally not helpful in elucidating a diagnosis. Because of the small size of the vessels generally involved, conventional angiography does not possess the resolution necessary to demonstrate abnormalities responsible for lacunar strokes.

The advent of computed tomography (CT) has allowed accurate localization of lacunar infarcts and has contributed greatly to the understanding of the pathogenesis and clinical course of lacunar disease. Computed tomography of the brain will either demonstrate no abnormality or may demonstrate the characteristic lesions. Normal CT scans can be compatible with lacunar infarcts that were missed because of their small size, because of being located in a difficult region of the brain to visualize via CT or because of improper timing of the study. In a large prospective study, positive CT scans were obtained in only 69 percent of the patients who exhibited one of the classic lacunar syndromes.[33] Fifty-five percent of these patients had positive CT scans within 10 days of the stroke or transient ischemic attack. By three months, an additional 13 percent of the study patients demonstrated CT changes consistent with lacunar infarction. Presumably, a period of time must pass for cavitation to occur to produce the characteristic lacuna. In the future, magnetic resonance imaging (MRI) with its superior resolution and capability for demonstrating the deeper noncortical parts of the cerebrum and brain stem may prove to be better than CT in the evaluation of lacunar strokes.[34]

A useful property of CT is its ability to distinguish between lacunar infarction and lacunar hemorrhage. Convention has held that it was unlikely for cerebral hemorrhage to cause lacunar syndromes. Recently, however, investigators have described the occurrence of all four classic lacunar syndromes due to small intracerebral hemorrhage.[35]

Recent data have suggested that emboli[14] and carotid occlusive disease[36] may be more frequently associated with lacunar stroke than originally appreciated. The usual source of emboli is the heart with atrial fibrillation, mural thrombus, prosthetic valves, and subacute bacterial endocarditis acting as predisposing factors. However, embolic infarction more commonly is seen as cerebral infarction of acute onset with cortical deficits. Clues to the presence of carotid occlusive disease include evidence of generalized atherosclerosis, bruits, and diminished carotid upstroke. When extracranial cerebrovascular disease or cardiac emboli are suspected clinically, additional procedures such as echocardiography, carotid doppler, and ultrasound or arteriography may be necessary to establish the diagnosis.

Management

Patients with lacunar strokes most commonly exhibit the triad of chronic hypertension, one of the four common lacunar syndromes, and a normal CT scan. It is generally agreed that patients with completed lacunar strokes should be treated expectantly without specific therapy. Vigorous treatment of mild to moderate hypertension, if present, should be avoided. Gradual reduction of blood pressure is preferred in order to avoid further deterioration in the neurological status due to relative hypoperfusion and exacerbation of ischemia in the tissue adjacent to the area of infarct.[8]

In patients with one of the uncommon lacunar syndromes, no specific therapy is indicated if one assumes the stroke is nonprogressive. However, since other diseases involving the central nervous system such as tumors or multiple sclerosis can mimic many of the uncommon lacunar syndromes, consultation with a neurologist is considered prudent.

The prognosis for survival and recovery in lacunar strokes is generally favorable. As would be expected, prognosis and degree of recovery have been found to be better with smaller lesions.[14] Following the acute phase, intensive physical therapy and rehabilitation should be encouraged, and control of hypertension, if present, should be attained.

Thirty percent to 50 percent of patients will have a progression of neurological symptoms after the occurrence of a lacunar stroke, or they will have lacunar transient ischemic attacks.[1,15,17,37] Treatment in these cases is controversial. Many

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authorities recommend treatment of evolving, cortical, thrombotic strokes in progression with anticoagulation when the progression is due to increasing ischemia rather than edema, cardiopulmonary complications, or hemorrhage.[38,40] Fisher and others have discussed the benefits (based on anecdotal or theoretical data) of anticoagulating patients with progressive lacunar strokes, specifically the hemiparetic variety, to decrease neurological deterioration.[8,32,36] Conversely, Fisher has advised against treatment of the pure sensory variety with anticoagulation since extravasation of erythrocytes is a characteristic feature.[17]

In a small prospective study involving anticoagulation in progressive lacunar stroke of the hemiparetic variety, no advantages to anticoagulation were demonstrated.[41] This study, which involved only four patients, however, was too small to draw definite conclusions. In a second uncontrolled, nonrandomized study, 15 patients treated with either anticoagulation or aspirin were felt to have had a more favorable outcome at one month than another group of 10 patients without either treatment.[42] There was no mention, however, of the type or severity of the lacunar strokes that their patients experienced, and the authors admitted that their results could have been skewed by a selection bias. Thus, the efficacy of anticoagulation in the treatment of lacunar stroke in progression has not been confirmed, although theoretical benefits with that form of therapy exist. If anticoagulation is considered, it is imperative that intracerebral hemorrhage first be excluded by means of computed tomography. Hopefully, prospective studies will appear in the future to address this important aspect of the management of lacunar stroke.

In regard to prevention of lacunar strokes, treatment of hypertension and attention to other risk factors related to the development of cerebrovascular disease are central points. Because of the established effectiveness of low-dose aspirin in treating transient cerebral ischemia in men, this form of prophylaxis seems reasonable.[43,45]

Discussion

Much has been learned about lacunar strokes in recent years regarding their etiology, clinical presentation, and clinical course. Fisher's contribution to the understanding of this disease process is enormous; it appears that his "small deep infarcts," which were almost invariably associated with hypertension, represent a majority of lacunar strokes. Recent evidence demonstrates that some lacunar strokes can be embolic[14] or hemorrhagic[35] in etiology, are not invariably associated with hypertension,[14,33] may be larger,[13,15] and can be associated with neurological manifestations that do not conform to the classic patterns.[22-31]

In most cases of suspected lacunar stroke, a familiarity with the lacunar syndromes and performance of appropriate noninvasive studies will confirm the diagnosis. Patients who have the triad of chronic hypertension, one of the classical lacunar syndromes, and a compatible CT scan can generally be spared angiography or the consideration of cerebrovascular surgery.[43] Because of the pathological heterogeneity of lacunar disease, however, it is incumbent upon physicians to be critical in the evaluation of patients whose clinical picture deviates from the classic presentation.

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References

1. Mohr JP, Caplan LR, Melski JW, et al. The Harvard Cooperative Stroke Registry: a prospective registry. Neurology (NY) 1978; 28:754-62.
2. Adams RD, Victor M. Principles of neurology. New York: McGraw Hill, 1977:518-20.
3. Wanger SL. Lacunar strokes. Primary Care 1979; 6:757-69.
4. Marie P. Des foyers lacunaires de désintégration et de différents autres états cavitaires du cerveau. Rev Méd 1901; 21:281-98.
5. Fisher CM. Lacunes: small deep cerebral infarcts. Neurology (NY) 1965; 15:774-84.
6. *Idem.* The arterial lesions underlying lacunes. Acta Neuropathol (Berl) 1968; 12:1-15.
7. *Idem.* Cerebral miliary aneurysms in hypertension. Am J Pathol 1972; 66:313-30.
8. *Idem.* Capsular infarcts: the underlying vascular lesions. Arch Neurol 1979; 36:65-73.
9. Fisher CM, Cole M. Homolateral ataxia and crural paresis: a vascular syndrome. J Neurol Neurosurg Psychiatry 1965; 28:48-55.
10. Fisher CM, Gore I, Okabe N, White PD. Atherosclerosis of the carotid and vertebral arteries: extracranial and intracranial. J Neuropath Exp Neurol 1965; 24:455-76.
11. Fisher CM. Bilateral occlusion of basilar artery branches. J Neurol Neurosurg Psychiatry 1977; 40:1182-9.
12. Araki G. Small infarctions of the basal ganglia with special reference to ischemic attacks. In: Recent Advances in Gerontology 1978; 469:161-2. Proceedings of the XI International Congress of Gerontology, Tokyo, Aug 20-5, 1978.

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13. Santamaria J, Graus F, Rubio F, Arbizu T. Peres J. Cerebral infarction of the basal ganglia due to embolism from the heart. Stroke 1983; 14:911-4.
14. Pullicino P, Nelson RF, Kendall BE, Marshall J. Small deep infarcts diagnosed on computed tomography. Neurology (NY) 1980; 30:1090-6.
15. Weisberg LA. Lacunar infarcts: clinical and computed tomographic correlations. Arch Neurol 1982; 39:37-40.
16. Mohr JP. Lacunes. Stroke 1982; 13:3-11.
17. Fisher CM. Lacunar strokes and infarcts: a review. Neurology (NY) 1982; 32:871-6.
18. Fisher CM, Curry HB. Pure motor hemiplegia of vascular origin. Arch Neurol 1965; 13:30-44.
19. Fisher CM. Pure sensory stroke involving face, arm and leg. Neurology (NY) 1965; 15:76-80
20. *Idem.* Ataxic hemiparesis. A pathological study. Arch Neurol 1978; 35:126-8.
21. *Idem.* A lacunar stroke. The dysarthria-clumsy hand syndrome. Neurology (NY) 1967; 17:614-7.
22. Mohr JP, Kase CS, Meckler RJ, Fisher CM. Sensorimotor stroke due to thalamocapsular ischemia. Arch Neurol 1977; 34:739-41.
23. Tabaton M, Mancardi G, Loeb C. Generalized chorea due to bilateral small, deep cerebral infarcts. Neurology (NY) 1985; 35:588-9.
24. Van Gijn J, Vermeulen M. Ataxic tetraparesis from lacunar infarction in the pons. J Neurol Neurosurg Psychiatry 1983; 46:669-70.
25. Guberman A, Stuss D. The syndrome of bilateral paramedian thalamic infarction. Neurology (Cleveland) 1983; 33:540-6.
26. Russo LS. Focal dystonia and lacunar infarction of the basal ganglia: a case report. Arch Neurol 1983; 40:61-2.
27. Fisher CM. Cerebral ischemia—less familar types. Clin Neurosurg 1971; 18:267-336.
28. Huang CY, Broe G. Isolated facial palsy: a new lacunar syndrome. J Neurol Neurosurg Psychiatry 1984; 47:84-6.
29. Caplan LR. Lacunar infarction: a neglected concept. Geriatrics 1976; 31:71-5.
30. Kase CS, Maulsby GO, deJuan E, Mohr JP. Hemichorea-hemiballism and lacunar infarction in the basal ganglia. Neurology (NY) 1981; 31:452-5.
31. Massey EW, Goodman JC, Steward C, Brannon WL. Unilateral asterixis: motor integrative dysfunction in focal vascular disease. Neurology (NY) 1979; 29:1180-2.
32. Nelson RF, Pullicino P, Kendall BE, Marshall J. Computed tomography in patients presenting with lacunar syndromes. Stroke 1980; 11:256-61.
33. Donnan GA, Tress BM, Bladin PF. A prospective study of lacunar infarction using computed tomography. Neurology (NY) 1982; 32:49-56.
34. Bydder GM, Steiner RE, Young IR, et al. Clinical NMR imaging of the brain: 140 cases. AJR 1982; 139:215-36.
35. Mori E, Tabuchi M, Yamadori A. Lacunar syndrome due to intracerebral hemorrhage. Stroke 1985; 16:454-9.
36. Aleksic SN, George AE. Pure motor hemiplegia with occlusion of the extracranial carotid artery. J Neurol Sci 1973; 19:331-9.
37. Rascol A, Clanet M, Manelfe C, Guiraud B, Bonafe A. Pure motor hemiplegia: CT study of 30 cases. Stroke 1982; 13:11-7.
38. Millikan CH, McDowell FH. Treatment of progressing stroke. Stroke 1981; 12:397-409.
39. Carter AB. Anticoagulant treatment of progressive stroke. Br Med J 1961; 2:70-3.
40. Baker RN, Broward JA, Fang HC, et al. Anticoagulant therapy in cerebral infarction. Neurology (Minneapolis) 1962; 12:823-35.
41. Dobkin BH. Heparin for lacunar stroke in progression. Stroke 1983; 14:421-3.
42. Lodder J, Gorsselink EL. Progressive stroke caused by CT-verified small, deep infarcts: relation with the size of the infarct and clinical outcome. Acta Neurol Scand 1985; 71:328-30.
43. Miller VT. Lacunar stroke: a reassessment. Arch Neurol 1983; 40:129-34.
44. Fields WS, Lemak NA, Frankowski RF, Hardy RJ. Controlled trial of aspirin in cerebral ischemia. Stroke 1977; 8:301-14.
45. A randomized trial of aspirin and sulfinpyrazone in threatened stroke. The Canadian Cooperative Study Group. N Engl J Med 1978; 299: 53-9.

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