Cognitive Impairment in Ischaemic Lacunar Stroke

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Abstract

Ischaemic cerebral small-vessel disease (SVD) can be visualised on brain magnetic resonance imaging (MRI) scans as lacunar infarcts, white matter hyperintensities and cerebral microbleeds, and has been recognised as the most frequent cause of cognitive impairment of vascular origin. Lacunar infarction is the most common manifestation of SVD and accounts for approximately 25 % of all brain infarctions. In patients with first-ever lacunar infarction, more than half of cases show impairment of the executive functions and meet criteria of mild vascular cognitive impairment. Lacunar stroke is an important predictor of post-stroke cognitive decline and vascular dementia. In the well-characterised cohort of 1,636 lacunar stroke patients of the Secondary Prevention of Small Subcortical Strokes (SPS3) trial, mild cognitive impairment was present in nearly half of participants and was an important complication of lacunar stroke more prevalent than physical disability. The cognitive profile identified to be associated with ischaemic cerebral SVD includes preserved memory with attention deficits and executive functioning impairment. However, in a recent systematic review of cognitive impairment in lacunar ischaemic stroke, impaired cognition appears less selective than previously thought, involving episodic memory and all major cognitive domains. Brain atrophy is also a feature of ischaemic cerebral SVD. Cognitive dysfunction in lacunar stroke sufferers is frequently overlooked in routine clinical practice and may be as common and clinically relevant as motor and sensory sequelae.

Keywords

Lacunar infarct, brain atrophy, prognosis, cognitive impairment, neuropsychology, cerebral small-vessel disease, brain atrophy

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Lacunar ischaemic infarctions, white matter lesions (WML) (hyperintensities) or leukoaraiosis and cerebral microbleeds constitute the spectrum of ischaemic cerebral small-vessel disease (SVD) documented on magnetic resonance imaging (MRI) studies. History of ischaemic SVD is frequently present in patients with cognitive impairment of vascular origin.[1–4] Cerebral lacunes are small infarctions of less than 20 mm in diameter localised in the vascular territory of penetrating arterioles, and represent the most-frequent manifestation of cerebral SVD (see *Figure 1*).[5,6]

Clinically, ischaemic lacunar stroke presents five well-recognised features, including pure motor hemiparesis,[7] pure sensory syndrome,[5,8] sensorimotor syndrome,[5,9] dysarthria clumsy-hand[10] and ataxic hemiparesis.[5,11] Atypical lacunar syndrome is occasionally observed.[12] Hypertension and diabetes are well-known cardiovascular risk factors for lacunar stroke.[2,5] Other presenting forms of cerebral infarction of the lacunar type are silent lacunar infarcts and transient cerebral ischaemia.[2]

Deep or penetrating arterioles 100–400 µm in diameter that originate directly from a large cerebral artery are typically involved in the pathogenetic mechanism of lacunar infarct.[13] These arterioles, without terminal anastomoses or collateral branches, provide blood supply to the deepest and nearest territories to the middle cerebral hemispheres and the brainstem. Lacunes are frequently found at the level of the lenticular branches of the anterior and middle cerebral arteries, the thalamoperforating and thalamogeniculated branches of the posterior cerebral artery and the paramedian branches of the basilar artery.

Symptomatic lacunar infarctions are commonly related to microatheromatosis or branch atheromatous disease.[13] Patients with hypertension frequently present multiple asymptomatic lacunar infarctions usually due to lipohyalinosis. In different series, silent lacunes have been reported to occur in 52 % and 77 % of cases.[5,13]

Also, silent lacunes have been detected by MRI in about 30 % of patients with first-ever lacunar stroke.[1,14] Contributing factors to vascular cognitive impairment in patients with lacunar infarction are summarised in *Table 1*.

This review is focused on the clinical evidence and mechanisms underlying the relationship between cognitive impairment and lacunar stroke. The description of neuropsychological consequences of haemorrhagic lacunar stroke[15] or the role of ischaemic cerebral SVD to the development of Alzheimer disease[16] is not discussed in detail, although this remains an intriguing area of research and overlaps with the topics covered in this review.