STROKE

Retinal small-vessel changes and lacunar stroke

A large study of patients with acute stroke suggests that the substantial proportion who have acute lacunar stroke could have an underlying non-atherothrombotic, occlusive, small-vessel disease rather than a thrombotic disorder. This could explain why antiplatelet treatments have proved ineffective in large stroke-prevention trials. “In the MATCH trial published in 2004, for example, 53% of patients had acute lacunar stroke; it is, therefore, no surprise that antithrombotic therapy failed to have an impact,” notes lead author Richard Lindley, of the University of Sydney, Australia.

Investigating the underlying cause of lacunar stroke is difficult because of the low death rate, which limits autopsy findings, and because noninvasive brain imaging techniques such as standard MRI and CT scans have insufficient resolution to examine small cerebral arteries in detail. Lindley and colleagues designed their study to try to establish whether changes in the retinal microvasculature could be used to monitor cerebral microvascular disease and so act as an indicator of lacunar stroke.

The cross-sectional study recruited 1,321 patients with acute ischemic stroke presenting at two centres in Australia and one in Singapore. In addition to standard clinical assessment, each patient had either a CT scan or an MRI scan of the brain, and retinal photographs were taken of each eye and then graded for retinal microvascular signs. An accurate diagnosis of lacunar stroke was made in 410 patients by means of a modified version of the Treatment of Acute Stroke Trial criteria or the Oxford Community Stroke Project criteria, and according to the findings on MRI scans.

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“Patients with acute lacunar stroke were more likely to have a range of retinal microvascular signs than are patients with non-lacunar stroke,” explains Lindley. For example, people with acute lacunar stroke had a 3.55-fold increased chance of having focal arteriolar narrowing,

a 2.32-fold increased chance of having enhanced light reflex of the arteriolar wall, and a 1.96-fold increased chance of having arteriovenous nipping.

Lindley says that the study has two major implications: “firstly, we hope to trigger a paradigm shift on what could be causing lacunar stroke and make researchers more aware of the other potential non-thrombotic mechanisms that have been suggested in the literature. Secondly, we hope to increase the research activity in this neglected form of stroke, which affects millions of people each year”. Since clinical management and potential interventions for lacunar stroke have traditionally been based on the assumption of underlying thrombotic small-vessel disease, “our study emphasizes the need for distinct and possibly new approaches to tackle this important debilitating disease,” concludes Lindley.

Kathryn Senior

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