DOI: 10.1590/0004-282X20130166

Lacunar strokes: does shape matter?

Infartos lacunares: o formato da lesão importa?

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Conflict of interest
There is no conflict of interest to declare.

Received 12 August 2013
Accepted 20 August 2013

Up to one in every four ischemic strokes can be lacunar infarcts. Traditionally, deep brain infarctions with a maximum diameter of 15-20 mm have been attributed to occlusion of a single penetrating artery by lipohyalinosis or microatheromatosis, but it has been suggested that other etiologies such as embolism may be responsible for up to one third of the cases. Lacunes can be detected by neuroimaging in asymptomatic individuals and are associated with greater risk of cognitive decline[1,2].

Lacunar infarcts can have a diameter greater than 15 mm on axial sections and greater than 20 mm on coronal or sagittal MRI sections[3,4]. It has been argued that shapes of lacunar infarcts may be linked to pathogenesis: lesions with an irregular shape may result from occlusion of largest perforating arteries, or from confluence of lesions, or even from secondary tissue degeneration. Lacunes with ovoid or spheroid, regular shapes may reflect involvement of smallest arteries[5].

In this issue of ***Arquivos de Neuro-Psiquiatria***, Feng et al.[6] evaluated clinical and imaging features as well as prognosis of 204 Chinese patients admitted with acute lacunar infarcts, defined as hypointense focal lesions in T1-weighted images and hyperintense focal lesions in T2-weighted, FLAIR, and DWI images with a diameter ranging from 3 mm to 20 mm. Radiologists (unaware of clinical characteristics) classified infarcts as regular or irregular by visual inspection of MRI images. The authors hypothesized that pathogenesis, clinical symptoms, and prognosis would differ between patients with acute regular or irregular infarcts. Sizes of the infarcts and extent of leukoaraiosis were also evaluated.

Blood pressure, blood glucose, hemoglobin A1c and lipids, as well as blood pressure variability (BPV) were checked within the first 24 hours after stroke. The authors did not mention criteria used to define risk factors for vascular disease such as arterial hypertension, diabetes mellitus, dyslipidemia (history? medical records? measurements/tests performed only before stroke, or either before or after stroke?). Logistic regressions were used to test for associations between risk factors for vascular disease, variation in systolic blood, shape and size of lacunar lesions, extent of leukoaraiosis, neurological deterioration (increase in NIH stroke scale scores at two weeks after stroke), and prognosis (modified Rankin scale at 3 months).

The only variable independently associated with shapes of lacunar infarcts was BPV within the first 24 hours. The authors concluded that "BPV is an independent risk factor for irregularly shaped lacunar infarction". However, because BPV was measured only ***after*** stroke, this variable cannot be undoubtedly considered a "risk factor" for irregular infarcts.

Variability in systolic blood pressure has been recognized as a predictor of stroke, and early variability in systolic blood pressure has been associated with worse outcomes after stroke[7-9]. There may be a causal link between BPV and pathogenesis of lacunar infarcts. Still, it is also possible that greater BPV may have occurred ***only after*** stroke and, in particular, in more severe strokes. Prospective studies are necessary to clarify this point. Furthermore, locations of infarcts were not depicted, and no information was provided about anti-hypertensive drugs chronically used before stroke, or administered within the first 24 hours after stroke, in patients with regular or irregular infarcts.

The authors also described a significant association between irregular shapes of lacunar infarcts and neurological deterioration two weeks after stroke. More severe leukoaraiosis, infarction size and irregular lacunes were found to be independently associated with worse

outcomes at 3 months. Since NIH stroke scales within the first 24 hours were not presented, it is uncertain whether or not severities of neurological impairments were comparable at baseline in patients with regular and irregular infarcts. It would be interesting to know whether shapes of the infarcts would remain independently associated with neurological deterioration at 2 weeks or with prognosis at 3 months, if baseline NIH stroke scores had been added to the model.

Efforts have been made to standardize definitions and methods to analyze/report imaging findings in patients with small vessel disease[3]. The work of Feng et al. highlights the importance of searching for distinguished features among patients with "lacunar infarcts". Such lesions should not be underestimated, or considered either "benign" or "homogeneous" strokes[2]. Research addressing the roles of small vessel disease on cognitive decline and neurodegeneration has gained great prominence, in parallel with the escalating increase in life expectancy worldwide, the "epidemics" of vascular disease, and the role played by vascular risk factors on development of small infarcts and leukoaraiosis. Understanding determinants and consequences of different patterns of lacunar infarcts will be valuable to shape preventive, diagnostic and therapeutic measures in order to decrease the burden of small vessel disease worldwide.

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