Clinical stroke syndromes: how to recognize and classify lacunar syndromes

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From the Porto University Center of Medicine Stroke Update Course, Porto, Portugal. 26–27 June 2018.

Abstract

Lacunar syndromes are usually caused by small, noncortical ischaemic strokes resulting from the occlusion of a single penetrating branch of a large cerebral artery (circle of Willis, middle cerebral artery or the basilar artery) and mostly are localized in the basal ganglia, subcortical white matter and pons. It is estimated that lacunar strokes are responsible for 15-26% of first-ever strokes. There are several proposed aetiological mechanisms, but the two major causes are lipohyalinosis of the penetrating arteries and microatheroma of the origin of the penetrating arteries. They are associated with hypertension, diabetes mellitus and cigarette smoking, among others. Lacunar syndromes usually present with symptoms developing over a short period of time but can also fluctuate for several hours with posterior clinical worsening. There are six validated clinical lacunar syndromes: pure motor hemiparesis, pure sensory stroke, ataxic hemiparesis, sensorimotor stroke, dysarthria-clumsy hand syndrome and hemiballismus/hemichorea, with lesions in different brain regions. These syndromes are characterized by the absence of "cortical" stroke signs. Brain imaging has several limitations in the acute phase and early clinical recognition of this syndrome is essential for a proper approach.

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Citation: Freitas, E. Clinical stroke syndromes: how to recognize and classify lacunar syndromes. International Journal of Clinical Neurosciences and Mental Health 2018; 5(Suppl. 2):L5
Published: 25 Jun 2018

Open Access Publication Available at http://ijcnmh.arc-publishing.org

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