

Lacunar Syndromes

Jacques De Reuck*

Emeritus Professor, Department of Neurology, University Hospital, Ghent, Belgium

*Corresponding Author: Jacques De Reuck, Emeritus Professor, Department of Neurology of the Ghent University Hospital, Belgium.

Received: June 27, 2020; Published: July 03, 2020

Miller Fisher defined lacunes as small deep infarcts generally considered as the hallmark of small-vessel disease [1]. They are mostly asymptomatic or give rise to aspecific or a less specific lacunar syndrome [2]. Only pure motor strokes correlate significantly with the presence of the responsible lacune in the internal capsule and with the stroke severity [3].

Lacunar infarcts are a frequent cause of vascular cognitive impairment and predominate in the deep white matter of vascular dementia [4].

On positron emission tomography, patients with white matter changes and lacunar infarcts who are cognitively impaired have a global more severe decrease in blood flow and oxygen consumption [5] as well as a more pronounced loss of vasoreactivity in the cerebral cortex, compared to those with preserved mental status [6].

Cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy (Cadasil) is an inherited microangiopathy with subcortical lacunar and white matter lesions, caused by mutations in the Notch3 gene. The clinical presentation is variable between and within affected families and is characterized by symptoms including migraine with aura, subcortical ischaemic events, mood disturbances, apathy and cognitive impairment. The mean age at onset of symptoms is 45 years with a variable duration of the disease ranging from 10 to 40 years.

The pathological hallmark of cerebral autosomal dominant arteriopathy with subcortical infarcts and leukoencephalopathy is the presence of electron-dense granules in the media of arterioles [7].

Although the initial description of Binswanger's disease was poor the neuropathological characteristics have been clearly described afterwards. They consist of enlarged ventricles, diffuse myelin loss in the deep white matter, sparing the subcortical arcuate fibres and the presence of many lacunar infarcts in the white matter and in the basal ganglia. A main characteristic is the fact that the cerebral cortex is completely spared. However, Binswanger's disease remains a rare and questionable cause of pure vascular dementia [8].

The most widely accepted approach to treatment is to intensively control well-established vascular risk factors, of which hypertension is the most important [9]. Aspirin significantly reduce stroke recurrence in patients with a lacunar stroke [10].

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