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Caudate nucleus ischemia

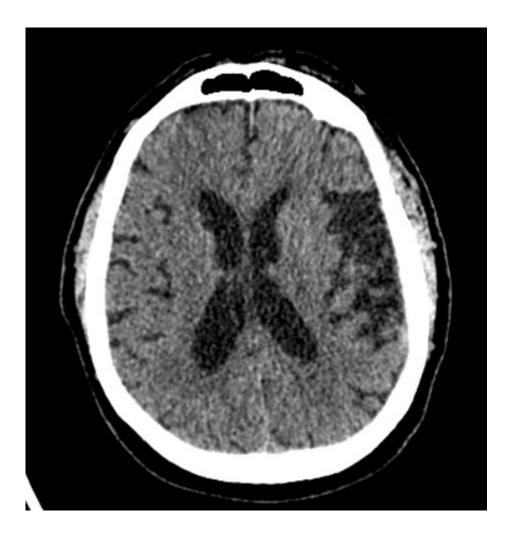
Etiology

Small vessel disease is the primary cause (59–66%) of ischemic stroke in CN, followed by cardioembolic etiology (20%), ipsilateral significant carotid stenosis and occlusion (8%). Furthermore, a single case of ischemic stroke involving the territory of Heubner's artery due to syphilitic vasculitis has been described ¹⁾.

Clinical features

Clinical features of both ischemic and hemorrhagic strokes included behavioral abnormalities dysarthria, movement disorders, language disturbances and memory loss. Most studies to date that have examined vascular CN pathologies have evidenced good outcomes ²⁾.

Diagnosis



Outcome

Kumral et al. ³⁾ have reported that 60% of patients with caudate infarct and 50% of those with CN hemorrhages recovered completely, even in the presence of hydrocephalus.

Case series

Eighteen patients had caudate nucleus infarcts (10 left-sided; 8 right-sided). Infarcts extended into the anterior limb of the internal capsule in 9 patients, and also the anterior putamen in 5 patients. Thirteen patients had motor signs, most often a slight transient hemiparesis. Dysarthria was common (11 patients). Cognitive and behavioral abnormalities were frequent, and included abulia (10 patients), agitation and hyperactivity (7 patients), contralateral neglect (3 patients, all right caudate), and language abnormalities (2 patients, both left caudate). The majority of patients had risk factors for penetrating artery disease. Branch occlusion of Heubner's artery, or perforators from the proximal anterior or middle cerebral arteries were the posited mechanism of infarction.

Chen ST, Liu YH, Hsu CY, Hogan EL, Ryu SJ: Moyamoya disease in Taiwan. Stroke 1988;19:53–59.

Villablanca JR. Why do we have a caudate nucleus? Acta Neurobiol Exp (Wars). 2010;70(1):95-105. Review. PubMed PMID: 20407491.

Kumral E, Evyapan D, Balkir K: Acute caudate vascular lesions. Stroke 1999;30: 100-108

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