Lacunar Strokes and Infarcts: A Review

Neurology, 1983 August. 32: 871-876

Lacunar infarcts are deep infarcts in non-cortical structures, occurring as a result of smaller occlusions to penetrating branches of larger cerebral arteries (i.e., MCA, PCA, basilar artery). Following resolution, lacunar infarcts result in small cavities ("lacunes") within deep white matter structures, commonly within the putamen, caudate, thalamus, pons, or internal capsule. Lacunar infarcts can be large (1.5-2cm) or small (3-4mm); larger lacunes are typically a result of smaller embolic particles from a larger, parent artery thrombus, and smaller lacunes (3-7mm) from lipohyalinosis (small vessel disease, or a chronic hypertensive vasculopathy). Lacunar infarcts are clinically distinct, and a foundational knowledge of lacunar syndromes helps with clinic-radiologic correlation. A host of different lacunar syndromes were outlined in this publication, based on case report review.

Five Major Lacunar Syndromes:

Pure motor hemiparesis: pure motor weakness of the face, arm, and leg on one side. Here, the author notes that the rules only apply when all three structures are affected. Weakness can develop acutely, in a step-wise fashion, or gradually over a few days. Brisk reflexes can develop w/in the first few hours. The most common locations include the juncture of the anterior and posterior limbs of the internal capsule, or the lower basis pontis. Etiologies include atherosclerosis or other small vessel disease of penetrating branches or embolisms from a larger parent artery.

Pure sensory stroke or TIA: fixed or transient face, arm, and leg numbness, though w/o the need to have all three regions every time. Sensory symptoms are also varied and can include paresthesias, though numbness is most commonly reported. The lesion typically lies in the posteroventral nucleus of the thalamus, with an etiology of small vessel disease.

Ataxic-Hemiparesis: combined hemiparesis with super-imposed ataxia in the ipsilateral leg and arm, as well as dysarthria and/or nystagmus. Sensory changes in the involved face or hand can develop if the medial lemniscus is involved. The lesion is typically at the junction of the upper and inferior basis pontis. The involved artery is typically the paramedian branch of the basilar artery, due to a small atheroma or an embolism from the parent artery.

Dysarthria-clumsy hand syndrome: commonly associated with dysarthria, dysphagia, and slight weakness of one hand, in the absence of any sensory deficit. The involved region is often the basis pontis, perhaps the internal capsule. Cortical infarcts can also present in this way, though often there will be a subtle sensory deficit, if so.

Sensorimotor syndrome: mixed hemi-sensory deficits and hemiparesis of the face, arm, and leg on one side, with involvement of the posterolateral nucleus of the thalamus and the posterior limb of the internal capsule, often secondary to small vessel disease.

Less common but often appreciated lacunar syndromes were also reviewed here, a few of which are described below:

<u>Mesencepahlothalamic syndrome</u>: involvement of the paramedian regions of the midbrain, subthalamus and thalamus, due to occlusions of the artery of Percheron originating from the top of the basilar artery.

Deficits often include 3rd nerve palsies, Parinaud syndrome, drowsiness, possible abulia. Per the author here, most are felt to be embolic in etiology.

Lower basilar branch syndrome: several deficits can occur, including dizziness, nystagmus, diplopia, INO, horizontal gaze palsy, dysphagia, ataxia. Often, these are due to an occlusion of a branch of the lower basilar or upper vertebral artery, with infarcts of the lower tegmentum, sparing the corticospinal tract. Partial syndromes were felt to be due to occlusions of the PICA, SCA, or AICA.

Lateral pontomedullary/medullary syndrome: facial weakness, vertigo and/or vomiting, dysphagia, dysarthria, tinnitus, ataxia, nystagmus, ipsilateral Horner's, ipsilateral facial numbness, contralateral body numbness. All are due to an infarct of the lateral medulla due to an occlusion of the upper vertebral artery or PICA.

Pure motor hemiparesis sparing the face: infarct of a medullary pyramid due to a vertebral artery occlusion. Vertigo or nystagmus may be present.

Thalamic dementia: infarct of the central region of the thalamus or subthalamus from an anterior paramedian artery (of the PCA) occlusion, leading to memory impairment, abulia, and a partial Horner's syndrome (hypothalamic involvement)

Hemiparesis with a 3rd nerve palsy (Weber): cerebral peduncle (midbrain) and 3rd nerve

Hemiparesis with a 6th nerve palsy: cerebral peduncle (inferior pons) and 6th nerve

Cerebellar ataxia and 3rd nerve palsy (Claude): cerebellar peduncles and 3rd nerve

Hemiballism: infarct of the subthalamic nucleus, due to a PCA or AchA occlusion

OTHER: isolated dysarthria was often encountered in hypertensive patients, though was w/o pathological correlation. Lacunes in the head of the putamen and caudate were rarely symptomatic unless the internal capsule was involved, and the author stated that atherosclerotic Parkinsonism could not be confirmed, per review.

As is accepted today, the author notes that many lacunar infarcts can occur haphazardly in different locations over time in a single patient, leading to more complex and neurologic deficits. However, only one or a few of these are typically symptomatic; accrual of purely silent lacunar infarcts over time is deemed less likely. Finally, in the authors review, the incidence of lacunar strokes appeared to be declining at the time of this publication, in conjunction with the more widespread anti-hypertensive therapy. This provided anecdotal support for long-standing hypertension as a major risk factor for all of the above.

Summary created by Elaine Sinclair, D.O.