EDITÖRE MEKTUP / LETTER TO THE EDITOR

Neurobehavioral, language, and speech disturbances in caudate infarction and recurrent artery of Heubner occlusion

Kaudat enfarktüsünde ve tekrarlayan Heubner arter tıkanıklığında nörodavranış, dil ve konuşma bozuklukları

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To the Editor,

Ischemic strokes in the territories of the anterior cerebral arteries are rare and account for 0.5 to 3.0% of all ischemic strokes. The recurrent artery of Heubner and medial striate arteries arise near the origin of the anterior communicating artery and supply the anteromedial portion of the caudate nucleus as well as the anterior limb of the internal capsule. The caudate nucleus receives an overlapping blood supply from the lateral lenticulostriate arteries (middle cerebral artery), anterior lenticulostriate arteries (anterior cerebral artery), and recurrent artery of Heubner (anterior cerebral artery).

We report on the case of a 71-year-old hypertensive woman, who was brought to the Emergency Room by her family. Before 1 day, she developed sudden confusion and restlessness. Examination revealed disorientation to time and place (but not to person), severe dysarthria, and mild right facial-arm weakness. The power of the right lower limb was normal. The right planter reflex was equivocal. Non-contrast CT brain scan (figures 1 and 2) revealed ischemic infarction of the left caudate nucleus. The family declined doing cranial MRI/MRA.

Caudate damage is usually ischemic rather than haemorrhagic and is more common in males (mean age of 62±9 years). There is no predilection side; the right and left sides are somewhat equally affected and bilateral involvement occurs in 12% of patients.

The majority of cases are due to atherosclerotic small vessel occlusion; hypertension and hypercholesterolemia are the commonest risk factors.

Because of the variable and overlapping blood supply, the clinical presentation is also very variable and depends on the occluded supplying artery, side (right or left) of infarction, and damage to the adjacent structures (anterior limb of internal capsule and putamen). Caplan and coworkers analyzed 18 patients with caudate infarctions; extension into the anterior limb of the internal capsule was observed in 9 patients while only 5 patients demonstrated anterior putaminal involvement. Neuro-psychological and behavioral manifestations, cognitive dysfunction, speech and language disorders, motor features (so-called non-pyramidal hemi-motor syndrome), and involuntary movements (chorea) can be encountered. Mendez and colleagues reported on 12 patients with caudate infarctions, who developed neurobehavioral abnormalities and cognitive dysfunction. They found that apathy, disinhibition, or major affective disturbances were the main symptomatology; they suggested that the pattern of personality change correlated with size and location of lesion within the caudate but not the laterality. Grönholm and coworkers analyzed language and speech disturbances in patients with caudate infarctions; a somewhat unexpected finding was that the region of most convergence of lesions was subcortical, in the
body of the left caudate nucleus. Robes and colleagues\textsuperscript{7} suggested that the head of the caudate forms an epicenter of cognitive control, including control over the language network, in particular selection/inhibition. Kumar and colleagues\textsuperscript{3} found that language and speech disturbances were observed in 23\% of their patients; dysarthria was more common than aphasia. Non-fluent type of aphasia (the commonest), mild trans-cortical motor aphasia, and global aphasia were found. Kumar also found that more prominent aphasia and dysarthria were associated with extension into the anterior limb of the internal capsule.

In summary, caudate infarction may result in a multitude of behavioral changes, psychological abnormalities, and cognitive dysfunction in addition to a constellation of language and speech disturbances, involuntary movements, and weakness.

Figure 1. Non-contrast CT brain scan of the patient. Note the left triangular hypodense lesion abutting on the frontal horn of the left lateral ventricle. In addition, there are multiple bilateral periventricular white matter ischemia of small vessel disease.

Figure 2. Non-contrast CT brain scan at the level of basal ganglia and thalami. The head of the left caudate nucleus is infarcted, together with the anterior limb of the left internal capsule. The overall depiction is highly suggestive of occlusion of the left recurrent artery of Heubner. The normal right caudate (within the white lines), anterior limb of internal capsule (within the red lines), and putamen (within the yellow lines) were demarcated for comparison.

REFERENCES