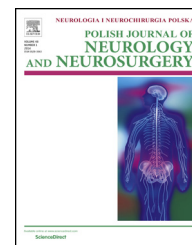


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Letter to the Editor

Hyperdense artery sign in middle cerebral and basilar arteries: A catastrophic stroke



Keywords:

Hyperdense artery sign
Middle cerebral artery occlusion
Basilar occlusion
Acute stroke
Computed tomography

A 60-year-old hypertensive man, complaining of fatigue and dyspnea, was admitted to the cardiology service for a workup. The cardiac catheterization was performed, showing 40% stenosis of the right coronary and left circumflex arteries; while transthoracic echocardiography showed an aneurysm in the ascending aorta (59 mm), with aortic valve insufficiency. The patient was submitted to an aneurysmectomy, including reconstruction of the ascending aorta, lasting 7 h (9 am to 4 pm). He was transferred to the intensive care unit following the procedure, without complications. Twenty-four hours after admission, his Glasgow Coma Scale was 3/15 without sedation. Due to the absence of adequate awakening (24 h post-surgery), the patient was submitted to non-enhanced cranial computed tomography (NECCT), which showed hyperdense artery signs (HAS) in the left middle cerebral artery (MCA)—longer than 8 mm in length, 62 Hounsfield units (HU)—and basilar artery (BA)—58 HU; with extensive hypodensity in the left frontal lobe, brainstem, occipital lobes and cerebellum (Fig. 1). In a few hours, his pupils were unreactive (5 mm) and corneal and oculocephalic reflexes were absent. The patient died 36 h after surgery. We used a 4-detector, multislice, CT scanner, model Asteion Super 4 (Toshiba, Japan) to perform standard brain studies with the following parameters: tube current, 200 mAs; tube voltage, 120 kV; iterative 3D volume reconstruction; matrix, 512 × 512; and reconstruction section thickness, 5 mm. Analyses were performed in the axial section, parallel to the orbitomeatal line from the base of the skull to the vertex, with multiplanar reconstruction techniques (MPR) in the coronal and sagittal plane.

HAS is the earliest alteration in acute ischemic vascular events, visible before changes in brain parenchyma [1,2]. When this sign is local, asymmetric and associated with

clinical signs of an acute cerebrovascular event, it predicts thrombus or embolus in brain arteries [2]. As an early marker of MCA ischemia, HAS occurs in 5–75% of patients with acute ischemic stroke [3]. BA and MCA occlusive disease are a devastating disorder, commonly resulting in death or severe disability [1,2,4]. BA occlusion is responsible for approximately 1% of all strokes [4]. A diagnosis of posterior circulation stroke with hyperdense BA sign is very rare [2]. To the best of our knowledge, this is the first description of HAS concomitant in the MCA and BA.

A shorter period between symptom onset and an NECCT is the main factor for increased HAS detection [3], while reducing section thickness in image reconstruction is another predictor of increased sensitivity, between 80 and 100% [1]. The MCA is 2–3 mm in diameter, while the section thickness used in most NECCT is 5 mm [1]. Given the perpendicular orientation of the BA in relation to the axial NECCT, we postulate two factors that increase HAS detection sensitivity: longitudinal clot size and thinner sections in image reconstruction.

The histopathological correlate is vessel occlusion due to thrombus or embolus coagulates [1,2]. The hyperattenuating component is due to plasma extrusion with increased haematocrits and subsequent organization of cells and debris during clot formation [1,2].

Since it characterizes an early sign, in the proximal portion of brain arteries, HAS should alert neurologists and neurointerventionalists to the urgent need for reperfusion therapy. The most common procedure is currently intravenous thrombolysis, up to 4.5 h after onset, with recanalisation in around 10% of cases when occlusion occurs in the internal carotid (terminal portion) and 30%, in the MCA (proximal segment) [5]. Favorable results have been achieved with mechanical thrombectomy, within 8 h of symptom onset, with successful recanalisation in 61% and 86%, respectively [5]. When NECCT sections show occlusions longer than 8 mm in the anterior circulation, recanalization with intravenous tissue plasminogen activator (IV tPA) is rarely performed [6]. Promising results have been reported for BA occlusion with thrombectomy, even beyond 6 h of symptom onset, that are more effective than intra-arterial (up to 6 h after symptom onset) and venous thrombolysis, with recanalisation rates of

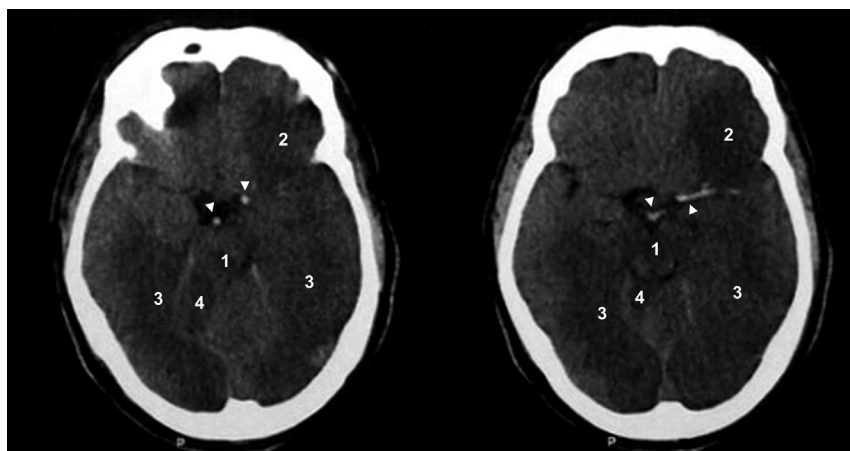


Fig. 1 – Non-enhanced cranial computed tomography, axial view: signs of hyperdense artery in the basilar and middle cerebral arteries (arrow head); observe the hypodensity over the brain stem (1), left frontal lobe (2), occipital lobes (3), and cerebellum (4).

81.2%, 65% and 53%, respectively [7]. Favorable outcomes (Rankin scale ≤ 2 , 3 months) were reported in 56.2%, 24% and 22% of cases, respectively [7].

According to a study by Moftakhar et al., the composition of the thrombus is an essential factor for the success of recanalization [8]. Thrombi with lower HU have a higher proportion of components (platelets, atheroma and cellular debris) that are resistant to fibrinolytic agents and mechanical thrombectomy [8].

In the case described, a catastrophic cardioembolic stroke occurred. The late diagnosis (24 h) of MCA and BA occlusions in the presence of HAS, associated with extensive areas of hypodensity become impossible any attempt at revascularisation in search of a favorable outcome. After cardiac surgery, when inadequate awakening is suspected, an emergency NECCT (fastest method) should be performed in order to identify evidence of ischemic stroke. The size of the thrombus and analysis of the radiological image of its composition in the NECCT, can help select patients who would probably not respond to IV tPA, but could achieve better results with endovascular therapy, such as intra-arterial thrombolysis or mechanical thrombectomy [6,8].

Conflict of interest

None declared.

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Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involv-

ing humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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