Thrombus Features in Hyperacute Ischemic Stroke: A Perspective on Using Length and Density Evaluation

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Background: More insights in the etiopathogenesis of thrombi could be helpful in the treatment of patients with acute ischemic stroke (AIS). One of the most confident and early imaging findings of stroke includes arterial hyperdensity. The purpose of this study was to determine whether thrombi's density and length would be useful for predicting their origin. *Methods:* We evaluated 68 consecutive patients with AIS to correlate the presence of thrombi and their imaging features with the stroke subtype. *Results:* After excluding patients with small-artery occlusion mechanism and undetermined and other causes, the stroke etiologic subtypes were large-artery atherosclerosis (LAA) in 59.0% of the patients, cardioembolism in 31.0%, and cervical artery dissection (CAD) in 10.0%. CAD more often caused thrombi with the longest length and highest attenuation, while thrombi that originated from the LAA had the smallest length and lowest attenuation. The mean Hounsfield unit (HU) values of all thrombi (with and without hyperdensity) on noncontrast computed tomography were 62.4 (50.0-70.0) in CAD, 53.8 (42.0-65.0) in cardioembolism, and 48.6 (27.0-65.0) in LAA. The length measurements were 28.5 mm (12.0-52.0) in CAD, 13.7 mm (5.0-31.0) in cardioembolism, and 10.8 mm (3.0-25.0) in the LAA subtype. The minimum cutoff value of 60 HU and a length greater than 20 mm were able to discriminate the CAD thrombi with an accuracy of 86.8% and 92.6%, respectively. *Conclusion:* Our study findings show how important thrombus analysis is in patients with AIS. Thrombus analysis can allow early suspicion of CAD before dedicated imaging of the cervical arteries is performed.
Key Words: Thrombi density—thrombi length—stroke subtypes—cervical artery dissection—computed tomography angiography—hyperacute stroke.

Introduction

Accurately classifying the etiology of ischemic stroke is essential for research, stroke outcomes, recurrent stroke rate, and strategies for secondary stroke prevention, which vary according to different stroke subtypes.[1-3] Ischemic stroke of the arterial origin is caused by cardioembolism in 18%-30% of cases, large-artery atherosclerosis (LAA) in 13%-36%, or small-artery occlusion in 14%-27%.[2,4] Dissection of a vertebral or carotid artery is not often the cause of ischemic stroke, as it accounts for approximately 15% of strokes in younger patients.[5]

Noncontrast computed tomography (NCCT) of the brain has been accepted for the initial assessment when stroke

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is suspected in a patient.[6] One of the most confident and early NCCT finding in the stroke setting is arterial hyperdensity, which represents an intraluminal thrombus.[7]

A false increase in the attenuation may be related with high hematocrit (Hct) levels that increase the X-ray attenuation.[8] Likewise, a previous study, evaluating the use of CT density measurement and Hounsfield unit (HU)/Hct ratio in diagnosing acute cerebral venous sinus thrombosis, found only a minor difference between the accuracy of the HU/Hct ratio and the accuracy of the HU measurement alone.[9] Also, patients with hypertension and diabetes mellitus had a statistically higher attenuation in cerebral arteries, sometimes mimicking intraluminal thrombus.[10] The higher Hct may have increased the density of the blood, while both diabetes mellitus and hypertension are associated with calcification within blood vessel walls.[10]

Postmortem studies have demonstrated that thromboembolic stroke can be caused by white, red, or mixed blood cell clots. It is assumed that hemoglobin has an important concentration of iron and is responsible for the clot attenuation in NCCT scans.[11] Conversely, as white cell thrombi contain variable amounts of atheromatous and cellular debris, fibrin, and platelets with only few red cells, these peculiar thrombi might present with lower HU counts (hypodense).[12,13]

The reliable analysis of thrombus composition may provide important prognostic information and may affect treatment decisions in the acute phase, whereas according to some studies, the HU quantification of thrombus in NCCT correlates with recanalization and good clinical outcome.[14,15]

The length and location of the clot has also been used to predict treatment response, suggesting that more proximal and longer clots may be resistant to intravenous thrombolysis.[16,17] A recent randomized trial concluded that depending on the location of intracranial occlusion, the intraarterial treatment was effective and safe.[18]

Despite the importance of clot characteristics there are few reports in the literature correlating it with a specific stroke etiology.[14,19] This study evaluated the possible correlation among the attenuation and length of intracranial thrombus to predict different stroke subtypes in a series of patients.

Methods

Patient Selection

The institutional review board and local ethics committee approved the present study, which retrospectively reviewed the records of a series of patients with hyperacute stroke evaluated using multidetector computed tomography from July 2011 to May 2014.

Adult subjects (≥18 years old) who presented with neurological hyperacute symptoms (<6 hours) consistent with focal acute ischemia of the middle cerebral artery (MCA) territory and who had undergone computed tomography angiography (CTA) were considered eligible. The patients and/or their guardian signed the informed consent. Patients with known renal failure and iodinated contrast allergy, and those who refused to participate in the study and in examinations with inadequate image quality were excluded.

For the present study, only patients with a later defined etiologic cause of the ischemic stroke due to cardioembolism, LAA, or cervical artery dissection (CAD) were included. To classify the final etiologic cause, the Stop Stroke Study Trial of Org 10172 in Acute Stroke Treatment criteria was used.[20]

According to the adopted criteria, a cardioembolic stroke was diagnosed if at least 1 cardiac source for an embolus had been identified in the absence of significant ipsilateral stenosis (>50%) and atherosclerosis. Conversely, LAA was defined after potential sources of cardiac embolism exclusion, when imaging findings of either significant stenosis (>50%) or occlusion of a major cerebropetal artery on CTA ipsilateral to the symptomatic hemisphere were presumed to be due to atherosclerosis.[20] CAD on CTA images was defined as a narrowed eccentric lumen surrounded by a crescent-shaped mural thickening or a tapered occlusion with an associated increase in external vessel diameter.[21] Patients with small-artery occlusion, undetermined causes, or other causes were excluded from this analysis.

Protocol

All examinations were conducted using a previously defined protocol with a minimum dose of ionizing radiation and intravenous iodinated contrast using a Philips Brilliance CT 64-channel scanner (Philips Medical Systems, Eindhoven, The Netherlands), which included the NCCT and cervical and intracranial CTAs.

The scan parameters of the NCCT were as follows: 120 kVp, 300 mAs, and a 1-mm reconstructed slice thickness. CTA (120 kVp, 80 mAs, and a 1 mm reconstructed slice thickness) was obtained after the intravenous administration of 50 mL of nonionic iodinated contrast at a rate of 7 mL/s followed by a 20 mL saline flush at the aforementioned rate. CTA source images were separately analyzed and postprocessed to obtain the maximum intensity projection and tridimensional views of the intracranial and extracranial arteries.

Data Analysis

All data were postprocessed using commercially available software on a workstation (Extended Brilliance Workspace v3.5.0.2250; Philips Medical Systems). Two neuroradiologists with experience in vascular imaging who were blinded to the outcomes analyzed all the exams separately. In case of disagreement, consensus was reached with a third reader.