Case Report

Bilateral hyperdense middle cerebral arteries: Stroke sign or not?

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ABSTRACT

Hyperdense middle cerebral artery (MCA) is a classic sign of acute thromboembolic disease. Simultaneous bilateral occurrence is uncommon and traditionally attributed to physiological hemoconcentration or attributable to imaging artifact. We present the case of a 71-year-old man whose admission noncontrast computed tomography (CT) demonstrated bilateral hyperdense middle cerebral arteries without other radiographic evidence of acute stroke. CT angiography confirmed bilateral MCA, M1 segment vascular occlusion and follow-up non-contrast CT demonstrated MCA territory infarctions.

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1. Introduction

The advent of computed tomography (CT) in the 1970s revolutionized the evaluation of acute stroke. Initially, clinical utility was limited to evaluation of hemorrhagic etiologies; however, several classic findings are now known to be indicators of acute ischemic stroke: loss of lentiform nucleus or insular ribbon outline, sulcal effacement, and dense middle cerebral artery (MCA) [1,2].

A unilateral hyperdense MCA is defined as a segment with Hounsfield units (HU) greater than 43 and 1.2 times the contralateral segment. It is considered an insensitive, yet highly specific, indicator of acute ischemic stroke and negative correlate for clinical outcome [2,3]. Although unilateral pathology correlates well with thromboembolic disease, bilateral pathology is considered, in almost all cases, due to underlying physiological dysfunction or image artifact. This occurs, for physiological dysfunction, with elevated hematocrit, and with image volume averaging artifact, either from vascular calcification or proximate parenchymal hypodensity [2,4].

To the best of our knowledge, there are only three reported cases in literature of bilateral hyperdense MCAs in acute ischemic stroke [5–7]. In only one of these cases, is CT angiography (CTA) confirmation of vascular occlusion documented [7]. This report describes a unique case of acute, embolic, bilateral, hyperdense MCA in the setting of poorly managed atrial fibrillation. In this instance, tissue plasminogen activator (t-PA) was contraindicated due to coagulopathy.

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2. Presentation of the case

A 71-year-old man with a history of chronic atrial fibrillation, managed with Coumadin, presented following a syncopal event. Physical examination demonstrated Glasgow Coma Scale and National Institute of Health Stroke Scale scores of 3 and 31, respectively. Noncontrast CT demonstrated bilateral increased MCA, M1 segment, density concerning for thromboembolic disease in the setting of an otherwise unremarkable imaging study and laboratory data (Fig. 1). Due to the patient’s clinical presentation and abnormal imaging study, a CTA was performed. CTA revealed bilateral MCA, M1 segment, hypodensity without distal contrast flow consistent with acute thromboembolism (Fig. 2). Intubation was performed for airway protection and the patient transferred to a medical intensive care unit for further management. The subsequent day, ventilator spontaneous breathing trials were unsuccessful and a follow-up noncontrast CT demonstrated classical MCA territory infarctions (Fig. 3) with diffuse cerebral edema. The patient consequently died from brainstem herniation.

3. Discussion

Stroke is a major cause of morbidity and the third leading cause of mortality in Western culture. Despite the advent of CT in the 1970s and improved recognition of acute ischemic stroke, the traditional outcome maxim of one thirds; distributed amongst full recovery, significant morbidity and mortality, persisted [8]. It was not until development of t-PA in the 1990s that an intervention existed to improve outcome. The inaugural study demonstrated a near 30% increase in the full recovery cohort compared to placebo [9].

On CT, physiological vascular flow corresponds to a density of 40 HU [10]. Several reported conditions can mimic both unilateral and bilateral thromboembolic disease. Partial volume averaging artifact is a result of the discrepancy between standard CT slice thickness, 5 mm, and MCA cross-sectional...
Delayed recognition of bilateral thromboembolic disease is commonly described in the few published reports of bilateral hyperdense MCAs [5–7], however no outcome data exists due to the paucity of reported cases. The outcome of a stroke patient with a unilateral hyperdense vessel, however, has been well established and is an ominous sign. It has consistently been demonstrated to predict a larger stroke volume, greater degree of neurologic deficit, and mortality [12]. As outcomes are so poor, early recognition is critical to initiating aggressive thrombolytic and neurointerventional therapy.

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Fig. 3 – Bilateral middle cerebral artery territorial infarcts. Axial non–contrast 5 mm image of the head. Cerebral edema in the frontal, parietal, temporal lobes, and midbrain.