

High-Resolution Magnetic Resonance Imaging Confirmed Atherosclerosis of an Intracranial Penetrating Artery: A Case Report

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Intracranial branch atheromatous disease is an atheromatous pathophysiological process associated with occlusion or stenosis at the orifice of a deep penetrating artery. However, apart from autopsy, atherosclerosis in the penetrating artery has not been confirmed in vivo. In this case, we confirmed that the ostium was located in the parent artery plaque and demonstrated the atherosclerosis of the proximal penetrating artery by using high-resolution magnetic resonance imaging. HR-MRI can be used to identify the pathological changes in penetrating arteries and provides more reliable basic information for the clinical subclassification and optimal medical treatment of penetrating artery infarction. **Key Words:** Branch atheromatous disease—high-resolution magnetic resonance imaging—penetrating artery—atheromatous plaque—middle cerebral artery—ischemic stroke.

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Introduction

In 1989, Caplan¹ first used the term intracranial branch atheromatous disease (BAD) to describe a type of ischemic stroke associated with occlusion or stenosis at the orifice of a branching artery supplying blood to the internal

capsule or pontine. Three different BAD pathologies were described^{1,2}: (1) obstruction of the branching artery mouth by an atherosclerotic plaque originating from the parent artery, (2) a junctional plaque in the arterial trunk and branches, and (3) microatheroma originating from the orifice of an arterial branch (Fig 1). These changes were differentiated from intrinsic disease of the penetrating artery itself. This pathology was called lipohyalinosis by Miller Fisher.³ However, apart from autopsy, direct evidence of an acute infarction in the penetrating artery has not been confirmed in BAD. In previous BAD cases, infarction was reported in the penetrating artery supply area, and the ipsilateral atherosclerotic plaque was detected in the parent artery, but there was no direct radiological evidence to demonstrate the penetrating artery was occluded by the parent plaque. Furthermore, to date, atherosclerosis has not been reported in the penetrating artery.^{4,5} Neuroimaging technology has advanced in recent years and high-resolution magnetic resonance imaging (HR-MRI, Discovery MR750 3.0T; GE Medical Systems, Milwaukee, WI) can now be used to accurately evaluate the morphology of intracranial vascular walls, including atherosclerotic plaques,

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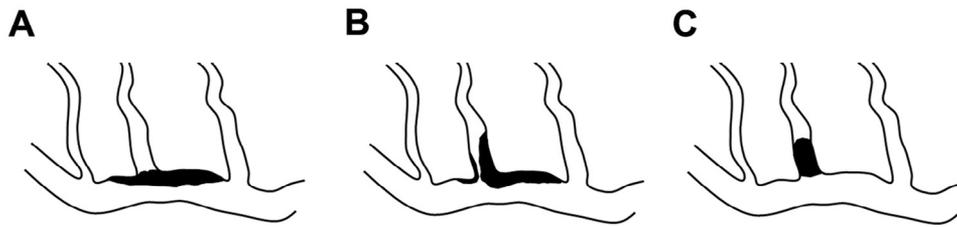


Figure 1. Schematic diagram of 3 different types of branch atheromatous disease. (A) Penetrating artery orifices obstructed by an atherosclerotic plaque originating from the parent artery; (B) the atherosclerosis unit with the parent artery trunk and the penetrating artery; (C) microatheroma originating from the orifice of the artery branch. Black represents the atherosclerotic plaque. (Adapted from Caplan.²)

plaque hemorrhage, dissection, and vasculitis.⁶ In this case report, we demonstrated the pathomechanism of ischemic stroke by using HR-MRI; the penetrating artery was occluded by the parent plaque and the circular enhancement of atherosclerosis lesion in the proximal penetrating artery.

Background

A 62-year-old Chinese man awakened with weakness of his left face and left limbs. The man had type 2 diabetes mellitus and hyperlipidemia, and was recently found to be hypertensive. On arrival to the hospital, the man's National Institutes of Health Stroke Scale (NIHSS) score was

7. An emergency computed tomography scan revealed a low-density lesion in the right basal ganglia (Fig 2, A). A high signal from 1.5-T magnetic resonance imaging was observed in the right lenticulostriate artery (Fig 2, B), and severe stenosis was detected in the proximal segment of the right middle cerebral artery (MCA) (Fig 2, C).

To identify the cause of stenosis in the right MCA, HR-MRI was performed after 2 days. Cube T1 is a relatively new 3-dimensional T1-weighted fast spin-echo sequence that uses a variable flip angle technique and a higher echo train length to reduce acquisition time and specific absorption rate.⁷ The atheromatous lesions of vessel wall could be enhanced on Cube T1 sequence. The junctional

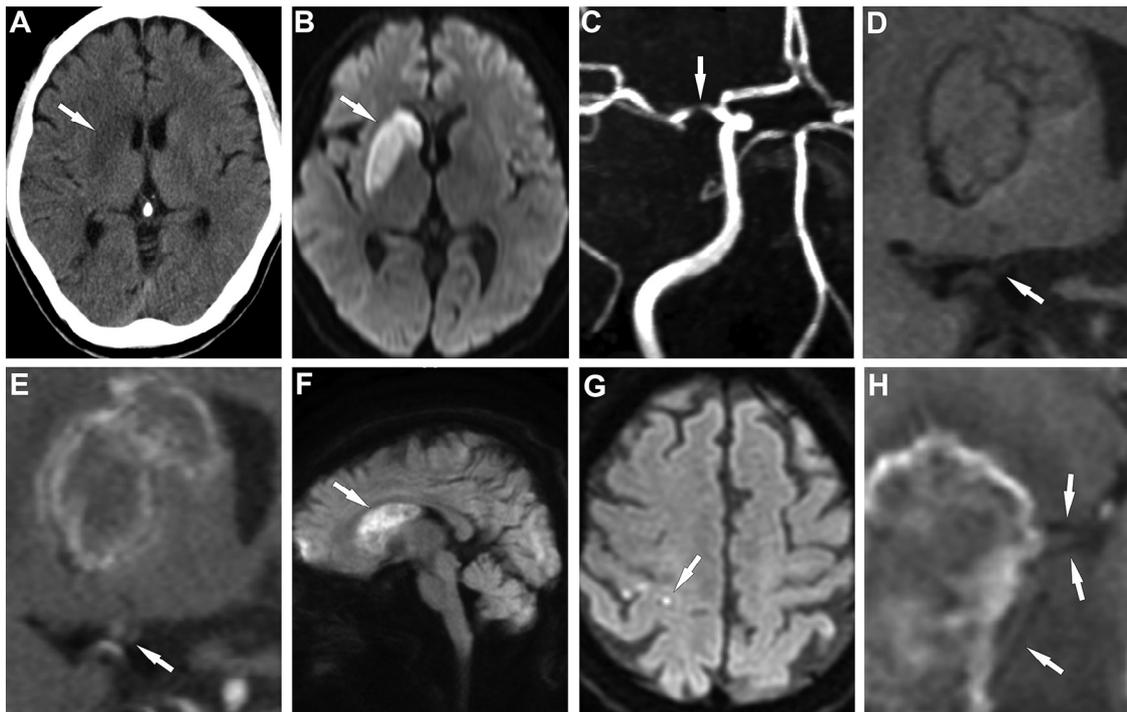


Figure 2. (A) Computed tomography scan showing a hypodensity signal in the right basal ganglia (white arrow). (B) Magnetic resonance imaging showed a hyperintense signal in the right lenticulostriate artery (white arrow). (C) Magnetic resonance imaging showing severe stenosis at the proximal segment of the right middle cerebral artery (white arrow). (D) Coronal Cube T1-weighted HR-MRI showed equal plaque signal in the middle cerebral artery stenosis (white arrow). (E) Enhanced Cube T1-weighted HR-MRI showing irregular enhancement of the atheromatous plaque (white arrow). (F) Sagittal diffusion-weighted imaging showing the infarction extending from the basal ganglia to the deep area. Basal ganglia appeared as hemorrhagic transformation (white arrow). (G) Diffusion-weighted imaging showing several small infarction lesions in the right parietal cortex (white arrow). (H) Sagittal Cube T1-weighted HR-MRI showing several penetrating arteries extending from the plaque of the middle cerebral artery to the infarct region (white arrows). Abbreviation: HR-MRI, high-resolution magnetic resonance imaging.

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