

Balloon-like Mobile Plaque in the Innominate Artery: Ultrasonographic and Pathological Perspectives of Repetitive Embolism

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Mobile plaque in the innominate artery is extremely rare and difficult to diagnose, especially in its acute stage. Its diagnosis is often delayed in many cases, resulting in delayed treatment and poor prognosis. Herein, we report the case of a 69-year-old patient with multiple cerebral infarction only in the right internal carotid artery and vertebrobasilar territories. No embolic sources were found until arterial ultrasonography detected a large balloon-like mobile plaque in the IA. Mobile plaque consisted of high- and low-echoic components and showed balloon-like plaque. Despite sufficient antiplatelet therapy, recurrence of cerebral embolism could not be prevented. IA replacement was eventually performed by cardiac surgeons. Pathological examinations showed that organized mobile plaque could have existed previously and acute thrombi, generated after the atheromatous plaque rupture caused by the mechanical burden of organized mobile plaque, could expand along with the organized mobile plaque and caused balloon-like plaque and related with repeated embolism. The IA should be explored immediately in cases of repetitive right-sided cerebral embolisms to prevent further recurrence.

Key Words: Mobile plaque—innominate artery—ultrasonography—acute thrombus—surgical treatment—right-sided cerebral embolism

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Introduction

Mobile plaque in the innominate artery (IA) is extremely rare and difficult to diagnose.^{1,2} Herein, we describe the case of recurrent cerebral embolisms due to the IA mobile plaque detected by ultrasonography and successfully treated.

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Case Presentation

A 69-year-old right-handed man was admitted to our hospital because of left hemiparesis. Neurological examination revealed slight left hemiparesis with NIHSS score of 2. Brain magnetic resonance imaging showed multiple acute and old cerebral embolisms in the right middle cerebral artery and vertebro-basilar territories (Fig 1A). MR angiography showed no abnormalities in major arteries. Laboratory findings showed elevated LDL-cholesterol (212 mg/dL). Although no embolic sources were detected by routine ultrasonography and electrocardiogram, carotid ultrasonography (LOGIQ E9, GE Healthcare) detected a large balloon-like mobile plaque attached to the wall of IA. Mobile plaque consisted of high- and low-echoic components (Fig 1B,C). Contrast-enhanced computed tomography angiography detected a mass lesion suggesting the plaque in the IA (Fig 1D).

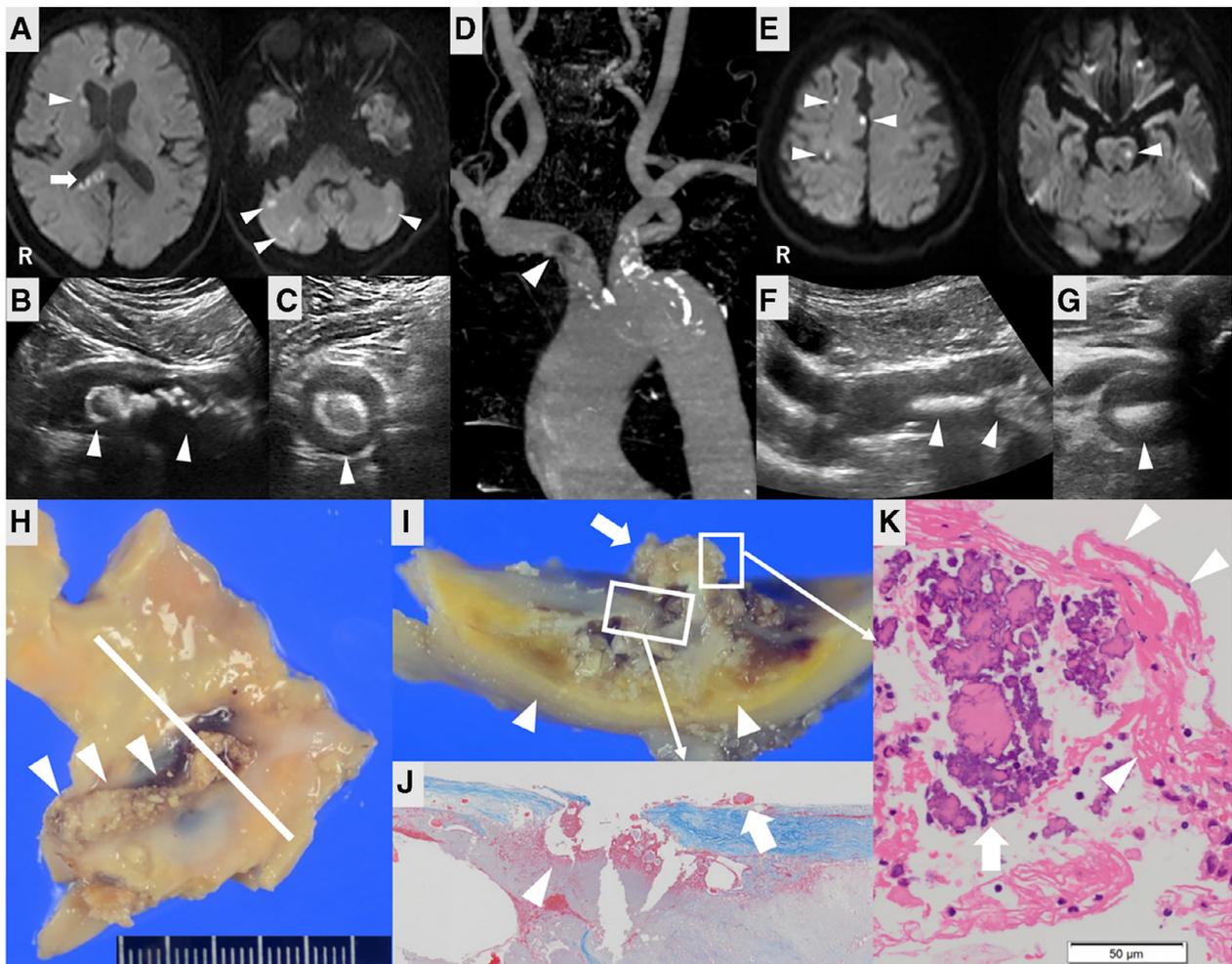


Figure 1. MRI, Ultrasonographical and pathological findings of mobile plaque. (A) Axial diffusion-weighted imaging showing multiple hyperintense signals in the right internal carotid artery territory and cerebellar region. Hyperintense signal lesions include acute (arrowheads) and old ischemic stroke (arrow). (B and C) Carotid ultrasonography showing a large mobile plaque attached to the innominate arterial wall (arrowheads) in longitudinal (B) and cross-sectional (C) views. (D) Contrast-enhanced computed tomography angiography showing a mass lesion suggesting the plaque in the innominate artery (arrowheads). (E) Follow-up magnetic resonance imaging showing multiple recurrent hyperintense signals in the right middle cerebral artery territory and pons (arrowheads). (F and G) Follow-up ultrasonography showing mobile plaque regression in the innominate artery (arrowheads) in longitudinal (F) and cross-sectional (G) views. (H) The mobile plaque (arrowheads) consisted of calcified plaque attaching to the intra-arterial atheromatous plaque surface. (I) Cross-sectional view (cut along the line in Panel H) showing mobile plaque (arrow) attached near the ruptured site of the intra-arterial atheromatous plaque (arrowheads). (J) Cross-sectional view of the mobile plaque with Masson's trichrome stain also showing ruptured intra-arterial atheromatous plaque at the site where the mobile plaque was attached (arrowhead), and many acute thrombi were observed around the mobile plaque surface (arrow). (K) Cross-sectional view of the mobile plaque (arrow of the panel I) with hematoxylin and eosin stain showing calcified plaque (arrow) covered by many acute thrombi (arrowhead).

Despite dual antiplatelet therapy, follow-up magnetic resonance imaging showed cerebral embolism recurrence in the right middle cerebral artery and vertebro-basilar territories (Fig 1E) and ultrasonography revealed mobile plaque regression (Fig 1F,G). Brain CT did not show calcified cerebral emboli. IA replacement was performed, and no recurrent cerebral embolism were noted. Pathological examination showed an organized calcified mobile plaque which was attached to the ruptured site of the atheromatous plaque surface in the IA (Fig 1H,I) and acute thrombi were observed

both of the atheromatous plaque rupture site and organized mobile plaque surface (Fig 1J,K). We concluded acute thrombi, generated after the atheromatous plaque rupture, could expand along with the organized mobile plaque and showed balloon-like plaque.

Discussion

Mobile plaques are associated with recurrent cerebral embolisms.³ In our case, atheromatous plaque rupture at

the root of the mobile plaque could result in fresh thrombi progression along with organized mobile plaque, and these fresh thrombi could cause balloon-like plaque and caused recurrent cerebral embolisms. Considering old cerebral infarction was seen in the right hemisphere, atheromatous plaque rupture could have repeated by the mechanical burden of the organized mobile plaque. There are only 3 cases of ischemic stroke caused by the IA mobile plaque detected by ultrasonography.⁴⁻⁶ Most of these cases showed early stroke recurrence or plaque enlargement despite sufficient antithrombotic therapies. Therefore, early detection of the IA mobile plaque by ultrasonography and surgical removal could be essential to prevent further recurrence.

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