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LETTERS TO THE EDITOR

Carotid Artery OCT in Cerebral Infarction

In a recent issue of *iJACC*, Prati et al. (1) suggested that plaque characterization by newer imaging modalities such as intracoronary optical coherence tomography (OCT) may help optimize management strategies in acute coronary events. Although OCT imaging has been extensively performed for the characterization of culprit plaques in acute coronary events (2), the intravascular imaging data defining plaque composition in the acute cerebral events is rather sparse (3). We present anecdotal examples of internal carotid artery (ICA) OCT images of plaque morphology in the patients afflicted by acute cerebral infarction. An 83-year-old man presented with sudden hemiplegia while praying in a temple (2 METs) (Figs. 1A to 1D). Magnetic resonance angiography revealed an ipsilateral ischemic lesion with 60% stenosis of the right internal carotid artery. Ten days after onset, we first tried to perform carotid artery stenting (CAS) of the culprit lesion as recently proposed by the randomized controlled trial SAPPHIRE (Stenting and Angioplasty With Protection in Patients at High Risk for Endarterectomy) (4). During balloon occlusion of the right common carotid artery with distal protection, we performed OCT (M2 OCT Imaging system, LightLab Imaging, Westford, Massachusetts) using a 0.016-inch OCT catheter (Imagewire, LightLab Imaging) to characterize the tissue components of the culprit lesion (Fig. 1B). OCT revealed a ruptured fibrous cap with a thickness of 130 μm and thrombus formation distal to the site of rupture. The red thrombus was accompanied by optical signal attenuation, and suggested the plaque rupture and thrombus formation were of recent origin. Accordingly, CAS was deferred in favor of carotid endarterectomy to reduce the risk of embolic complications (5). Similarly, a 76-year-old man presented with sudden hemiplegia. Eleven days after onset, angiography revealed an ipsilateral ischemic lesion with 60% stenosis of the left ICA. The OCT,

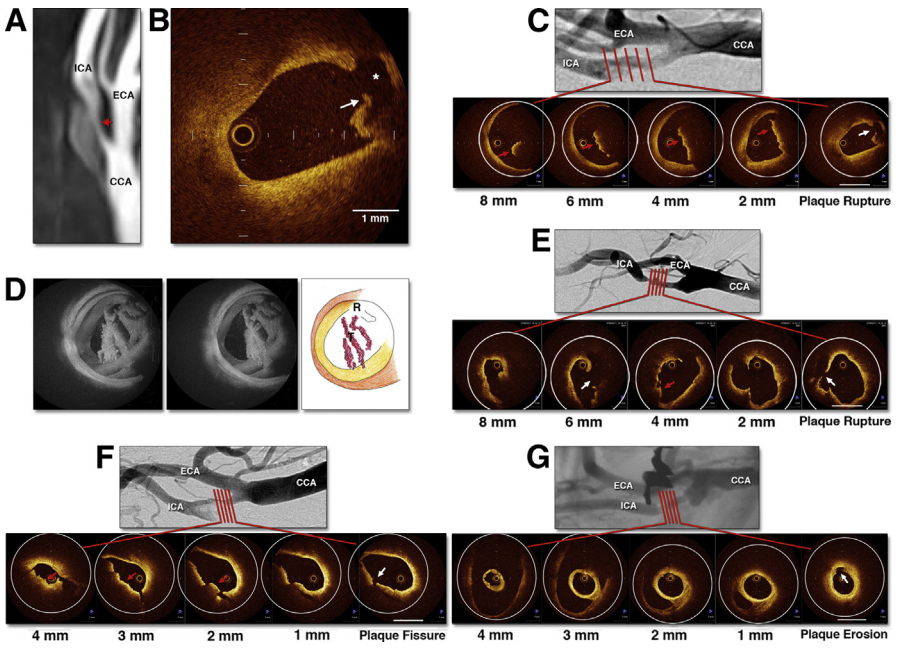

Figure 1. OCT Imaging of the Plaque Rupture in the ICA

(A) Magnetic resonance angiography revealed an ipsilateral ischemic lesion with stenosis of 60% in the right internal carotid artery **(red arrow)**. **(B)** Optical coherence tomography (OCT) revealed a residual fibrous cap identified as a flap **(white arrow)** between the vessel lumen and the cavity of the plaque **(asterisk)**, with a thickness of 130 μm. **(C)** Red thrombi with optical signal attenuation were detected distal to the site of rupture (scale bar = 2 mm). **(D)** The stereoscopic view proximal to the site of the lesion shows that thrombi were located at the center of the vascular lumen from proximal to distal to the lesion, although only the surfaces of the thrombi were detected due to the limited penetration depth of OCT. The rupture site was located at 1 o'clock on the proximal side. For a stereoscopic image with a 3-dimensional effect, please see the **right** image with your right eye and the **left** image with your left eye. At the same time, please focus as if you see the far side vaguely, and an image in 3 dimensions will be appreciable in the **center**. A schematic illustration of the proximal view is also provided on the **right side**. **(E)** Multiple plaque ruptures **(white arrow)** with thrombus formation **(red arrow)** distal to the site of plaque rupture. **F and G** show OCT imaging of the plaque fissure and plaque erosion. **(F)** Plaque fissure **(white arrow)** with thrombus formation **(red arrow)** distal to the site of the plaque fissure. **(G)** Plaque erosion **(white arrow)** with a double-lumen structure distal to the site of plaque erosion. CCA = common carotid artery; ECA = external carotid artery; ICA = internal carotid artery; R = ruptured site; T = thrombi.

however, revealed multiple plaque rupture sites with thrombus formation distal to the site of plaque rupture (Fig. 1E).

On the other hand, cerebral infarction may also be associated with intact fibrous caps wherein only plaque fissure or erosion may constitute the substrate for thrombus formation (Figs. 1F and 1G). An 83-year-old man presented with sudden hemiplegia while he had his breakfast. On the day after the onset, angiography revealed an ipsilateral ischemic lesion with 80% stenosis of the right internal carotid artery. We performed OCT to characterize the tissue components of the culprit lesion. OCT revealed a plaque fissure with thrombus formation distal to the site of plaque fissure (Fig. 1F). Thrombus may also occur with an intact cap, which has been described in coronary arteries as plaque erosion (1). A 72-year-old man presented with stuttering hemiplegia. Fifteen days after onset, angiography revealed an ipsilateral ischemic lesion with 80% stenosis of the right internal carotid artery. The OCT revealed an intact fibrous cap with a double-lumen structure distal to the site of plaque erosion (Fig. 1G). Although only a few examples of ICA imaging are available in the literature, we expect that larger experience would help better the understanding of carotid plaques and allow more effective interventions in cerebral infarction.

Masanori Kawasaki, MD, PhD,* Shinichi Yoshimura, MD, PhD, Kiyofumi Yamada, MD, Arihiro Hattori, MD, Yoshiyuki Ishihara, MD, Kazuhiko Nishigaki, MD, PhD, Genzou Takemura, MD, PhD, Toru Iwama, MD, PhD, Shinya Minatoguchi, MD, PhD

*Department of Cardiology, Gifu University Graduate School of Medicine, 1-1 Yanagido, Gifu 501-1194, Japan. *E-mail: masanori@ya2.so-net.ne.jp*

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Myocardial Mechanics in Noncontiguous HCM

Marked phenotypic heterogeneity is typical of hypertrophic cardiomyopathy (HCM) with mostly localized and contiguous left ventricular hypertrophy. Rarely, noncontiguous but focal left ventricular hypertrophy is seen (1). A more comprehensive assessment of HCM, including segmental distribution of hypertrophy resulting in diverse patterns of HCM, has been described using cardiac magnetic resonance (CMR), and centerline chord method on CMR has been used to identify hypertrophied left ventricular segments that may otherwise go unidentified on conventional 2-dimensional echocardiography (2). A recent 3-dimensional CMR analysis showed that the majority of patients with asymmetrical septal HCM had a spiral pattern of hypertrophy, with extension toward complete concentric apical involvement seen in 10% of these patients (3,4). This finding suggests that asymmetric septal and apical HCM are not completely separate phenotypes. The continuum of disease involvement from base to apex can be underestimated in cross-sectional imaging, especially on a conventional 2-dimensional echocardiogram. However, the role of speckle tracking echocardiography in identification of such a continuum and in understanding the mechanics of myocardial dysfunction, especially in patients with noncontiguous HCM, is unclear.

We describe 5 cases of the noncontiguous left ventricular hypertrophy phenotype of HCM. All of these patients had localized hypertrophy involving the basal subaortic septal segment as well as circumferential hypertrophy of noncontiguous apical segments, with normal thickness (apparent sparing) of the intervening midventricular segments on conventional 2-dimensional echocardiography and CMR. However, speckle tracking–derived myocardial mechanics confirmed reduced longitudinal systolic strain not only of the hypertrophied segments of the apex and basal septum but also revealed abnormally reduced strain of the nonhypertrophied intervening midventricular segments (Fig. 1A). For comparison, we also describe patients with isolated basal septal hypertrophy, basal and midseptal hypertrophy, and apical hypertrophy. The myocardial mechanics of these patients showed a reduced strain in the areas of hypertrophy without involvement of the adjacent nonhypertrophied segments (Fig. 1B).

Age of the patients in the 5 cases ranged from 35 to 73 years; 4 patients were male. Dyspnea was the common symptom. Three patients had sleep apnea. Electrocardiograms of all patients revealed marked T-wave inversions in precordial leads, typical of apical HCM. Basal septal thickness ranged from 20 to 24 mm and apical thickness from 15 to 20 mm. The midventricular thickness in all of these patients was measured to be <12 mm in women and <13 mm in men. There were varying degrees of systolic anterior motion of mitral leaflets. Three patients had delayed gadolinium enhancement (DGE) on CMR involving the apex and basal septum. All patients received optimal medical treatment for symptoms attributable to left ventricular outflow tract obstruction. Three patients underwent alcohol septal ablation. Two patients received implantable cardioverter-defibrillators (ICDs) due to syncopal episodes and DGE; a third patient had sudden cardiac death prior to ICD implantation. Four patients underwent genetic testing, and no mutations were found.

The age of the 5 patients with HCM used for comparison ranged from 20 to 78 years; 2 were male. Three had basal septal hypertrophy, 1 had basal septal hypertrophy with some involvement of the midventricle, and 1 had localized apical hypertrophy. Ventricular wall thickness ranged from 19 to 22 mm, and mild left ventricular outflow tract obstruction was present in septal variants. Also included was a patient with genotype positive for a myosin heavy chain 7 gene mutation but phenotype negative, with normal global and regional longitudinal strain. One patient had a tropomyosin