A 34-year-old woman was admitted for sudden onset of left-sided hemiparesis. Diffusion-weighted magnetic resonance imaging (MRI) 12 hours after symptom onset was positive, with hypersignal in the territory of the right middle cerebral artery consistent with acute cerebral infarction. Magnetic resonance angiography confirmed occlusion of the right middle cerebral artery and found a 30% stenosis (North American Symptomatic Carotid Endarterectomy Trial criteria) of the origin of the right internal carotid artery. Carotid ultrasound (Figure, A–D) evidenced the presence of a noncalcified plaque of the right carotid causing a stenosis of the origin of the internal carotid artery evaluated at 40% (North American Symptomatic Carotid Endarterectomy Trial criteria). Results of electrocardiography, Holter monitoring, transthoracic and transesophageal echocardiography, and laboratory investigations were all normal.

To rule out the presence of a prothrombogenic cancer, combined positron emission tomography and computed tomography was performed 1 hour after intravenous injection of F18-fluorodeoxyglucose (FDG; 4 MBq/kg); results were considered normal except for the presence of an intense uptake of FDG in the area of the right carotid bulb. Complementary positron emission tomography acquisition focused on the supra-aortic trunks (35 mL of Iomeron 400; Bracco, Milan, Italy). A pedicule thrombus appended to a noncalcified, nonstenotic plaque of the right carotid artery ipsilateral to the stroke (Figure, E) was identified with computed tomography angiography. In addition, intense FDG uptake with positron emission tomography was located along the right carotid bulb (Figure, E–G), which suggested a high density of inflammatory cells. The presence of a thrombus in contact with a ruptured carotid plaque (Figure, H–J) was confirmed by high-resolution MRI. With black-blood T1-weighted images acquired 5 minutes after intravenous injection of gadolinium chelate (0.1 mmol/kg gadoterate meglumine; Dotarem; Guerbet, Villepinte, France), this plaque appeared to be formed of a hypointense area covered only partially by a thin hyperintense band, consistent with the presence of a large lipid-rich necrotic core and a thin disrupted fibrous cap. The patient was treated with antiplatelet drug and statins and did not present with any recurrent stroke. Six months later, according to high-resolution MRI, only a nonstenotic atherosclerotic plaque persisted in the right carotid artery.

In up to 40% of patients presenting with ischemic stroke, no definite cause can be established despite an extensive workup. Interestingly, Freilinger et al detected with MRI the presence of nonstenotic carotid atherosclerotic plaques ipsilateral to the stroke in one third of patients admitted for an ischemic stroke considered cryptogenic. These atherosclerotic plaques were often complicated by intraplaque hemorrhage, fibrous plaque rupture, and luminal thrombus evidenced by high-resolution multicontrast MRI. The present case report confirms that thrombus can be generated by the rupture of nonstenotic carotid atherosclerotic plaques and can embolize distally, leading to ischemic stroke. Interestingly, atherosclerotic plaque had similar imaging aspects with high-resolution MRF and FDG–positron emission tomography, as described previously for symptomatic carotid plaques that caused luminal stenosis >50%. This observation emphasizes the potent role of nonstenotic carotid plaques in patients presenting with an ischemic stroke classified as cryptogenic and suggests that imaging could assist in the evaluation of the risk associated with these plaques.

Disclosures

None.

References


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Figure. A–D, Carotid ultrasound of the right carotid showed the presence of a noncalcified plaque with gray-scale 2-dimensional imaging (white arrow; A, longitudinal view; C, axial view) that caused a stenosis of the origin of the right internal carotid artery evaluated at 40% (North American Symptomatic Carotid Endarterectomy Trial criteria) with color Doppler imaging (B, longitudinal view; D, axial view). E–G, Combined F18-fluorodeoxyglucose (FDG) positron emission tomography/computed tomography angiography (PET-CTA) of carotid arteries. A pediculate thrombus (white arrowhead) appended to a noncalcified and nonstenotic plaque of the right carotid artery (E, white arrow) ipsilateral to the stroke was identified with CTA of the carotid arteries. In addition, intense uptake of FDG was detected along the right carotid bulb (F, white arrowhead) with PET, which suggested a high density of inflammatory cells (G, fusion PET-CTA). H–J, Successive black-blood T1-weighted axial views starting from the right carotid bulb (H) and extending into the origin of the internal carotid artery (I and J) obtained with high-resolution magnetic resonance imaging 5 minutes after injection of gadolinium chelates. Magnetic resonance imaging confirmed the presence of a thrombus (H, white arrowhead) in contact with a ruptured atherosclerotic plaque formed of a hypointense area covered only partially by a thin hyperintense band, consistent with the presence of a large lipid-rich necrotic core and a thin disrupted fibrous cap (H, white arrow).
Rupture of Nonstenotic Carotid Plaque as a Cause of Ischemic Stroke Evidenced by Multimodality Imaging
Fabien Hyafil, Isabelle Klein, Jean-Philippe Desilles, Mikael Mazighi, Dominique Le Guludec and Pierre Amarenco

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