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LETTER TO THE EDITOR

Atheromatous Plaque Vulnerability — the Neglected Vulnerable Carotid Plaques

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Dear Editor,

I read with great interest the article written by Benedek T *et al.*, entitled "Assessment of Coronary Plaque Vulnerability in Acute Coronary Syndromes using Optical Coherence Tomography and Intravascular Ultrasound. A Systematic Review", published in a recent issue of the Journal of Cardiovascular Emergencies.[1] The authors present a comprehensive description of multiple imaging-derived biomarkers that can characterize coronary plaque vulnerability.

The role of the vulnerability of an atheromatous plaque has been recognized in the last years as a key determinant of a major cardiovascular event. However, atheromatous plaques are present in the entire arterial system, without being limited to coronary circulation. A particular location where plaque vulnerability is of major importance is represented by the carotid arteries. Vulnerable carotid plaques have been demonstrated to be associated with a significantly higher risk of stroke resulting from the rupture and embolization of an unstable carotid plaque.[2] Therefore, identification of patients with vulnerable carotid lesions, even asymptomatic, remains an essential goal for stroke prevention.

Approximately 90% of carotid interventions, either surgical or interventional, are performed worldwide for asymptomatic carotid stenosis, in order to prevent the risk of further progression or plaque embolization with consecutive stroke.[2]

The presence of vulnerable components within a carotid plaque has been demonstrated to be associated with an increased risk of cerebrovascular events.[3] Therefore, assessment of vulnerability markers in the atheromatous plaques located in the carotid circulation could be extremely important. Such vulnerability markers of carotid plaques could be represented by ulceration, the amount of lipid core, intraplaque hemorrhage, or plaque inflammation. While ulceration could be easily detected by computed tomography, advanced technologies of magnetic resonance imaging (MRI) and positron emission tomography are required to certify the intraplaque hemorrhage and inflammation.[3] In a recent study, the presence of carotid intraplaque hemorrhage documented on MRI was independently associated with the size of cortical infarcts at brain MRI.[3] At the same time, new molecular MRI probes have been tested and validated as new imaging tools for assessment of inflammatory activity within an atheromatous plaque as a direct indicator of plaque vulnerability.[4]

Besides these sophisticated techniques, an alternative, easy-to-use method could be represented by transcranial Doppler, able to detect the presence of atheromatous microemboli in the carotid circulation, microemboli resulting from the rupture of a vulnerable carotid plaque.

In the above mentioned manuscript by Benedek T *et al.*, I believe that the relevance of plaque vulnerability in other territories than the coronary arteries is not sufficiently addressed. Taking into consideration the devastating impact of a stroke on a patient's health and socioeconomic status, a significant effort should be dedicated to detection of vulnerable carotid plaques. The significant progress encountered in the recent years in the

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field of detection and treatment of vulnerable coronary plaques, in order to prevent an acute coronary event, should be translated to assessment of vulnerable carotid plaques, too, in order to prevent a large number of ischemic strokes and reduce the number of unnecessary carotid interventions.

I believe that addressing both coronary and carotid plaque vulnerability could further help to elucidate the role of invasive and noninvasive imaging techniques in the complex assessment of pan-circulatory plaque vulnerability, at the same time putting patient vulnerability in the center of the pathophysiologic process related to acute cardiovascular events.

CONFLICT OF INTEREST

Nothing to declare.

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