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Editorial

Aortic Calcium Score and Vascular Atherosclerosis in Asymptomatic Individuals: Beyond the Coronary Arteries

Cuantificación del calcio aórtico y arteriosclerosis vascular en individuos asintomáticos: más allá de las arterias coronarias

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INTRODUCTION

Atherosclerosis is a generalized, progressive, chronic condition that can affect the entire vascular tree.[1] The clinical manifestations of atherosclerosis are ischemic heart disease, cerebrovascular disease, and peripheral arterial disease. Despite the current prevention strategies and a prompt diagnosis (through diagnostic imaging techniques), atherosclerosis is a major cause of morbidity and mortality in Western societies,[2,3] responsible for 84.5% of cardiovascular deaths and 28.2% of deaths due to any cause.[4]

From a pathophysiological viewpoint, atherosclerosis is a dynamic process that begins with deposition of lipid particles on the arterial wall, followed by apoptosis of the vascular smooth muscle cells and release of matrix vesicles. This process, together with macrophage infiltration, leads to calcification of the arterial wall intima. Fragments of the calcified plaque spread through the surrounding collagen tissue matrix and converge, forming fibrocalcific plaques.[5] Thus, arterial wall calcification is considered a direct marker of atherosclerotic disease. Over the last 2 decades, there has been a growing interest in detecting vascular calcification by various diagnostic imaging techniques in asymptomatic individuals with cardiovascular risk factors. The aim is to achieve improved risk stratification and reduce atherosclerotic disease-related morbidity and mortality.

Atherosclerosis is a complex phenomenon, involving numerous factors. An important one is increased blood concentrations of low density lipoprotein cholesterol, which causes changes in capillary permeability and gradual alterations in the arterial walls. Atherosclerosis is more prevalent in older individuals, those with hypertension, smokers, and patients with diabetes mellitus or insulin resistance.[6] Atherosclerotic plaques in the aorta have been associated with elevated homocysteine concentrations, prothrombotic markers (prothrombin), proinflammatory markers, (eg, leucocyte count, C-reactive protein concentration), and left ventricular hypertrophy. Although the white population shows a lower prevalence of hypertension and diabetes than the black population, paradoxically, it has a greater atherosclerotic burden.[7] There is also some controversy regarding the distribution by sexes: although coronary disease is less prevalent in women than men, there is evidence that women have a higher incidence of aortic atherosclerosis, even at earlier ages and regardless of the presence of cardiovascular risk factors.[8]

STRUCTURAL AORTIC WALL CHANGES ASSOCIATED WITH ATHEROSCLEROSIS

Atherosclerotic plaque formation in the aortic wall causes wall erosion and degeneration of the normal media layer. Subsequently, wall atrophy and wall thinning occur, and the vessel dilates in parallel with increases in the volume of plaque.[9] Dilation is considered a compensatory mechanism to prevent or delay stenosis of the vessel lumen, but in arteries having a medium-sized or small diameter and a large atherosclerotic burden, it is sometimes insufficient and stenosis ensues. In larger vessels, however, dilation maintains the vessel lumen open for a longer time, avoiding stenosis but producing a risk of aneurysm formation (Figure 1).

Changes in the aortic media layer are also associated with a decrease in the elastic properties and distensibility of the vessel. In this regard, non-oscillatory shear stress on the vessel wall has been linked with greater fat infiltration and cholesterol-rich plaques, which perpetuate the dilation process. Aortic calcifications originate in vessel segments where the wall stress is less intense, but highly ocillatory. The lesser curvature of the aortic arch and the posterior wall of the descending aorta are the most commonly affected areas.

Another factor associated with vascular dilation is the vessel wall tension. Wall tension is proportional to the radius of the vessel; thus, with gradual thinning of the arterial wall, the tension produced suffices to cause a slow increase in the vessel diameter. This increase then leads to an increase in wall tension, resulting in a vicious circle of vascular atrophy and dilation. This mechanism explains why the descending aorta and abdominal aorta are more prone to develop aneurysms than the coronary or carotid arteries.

The medial layer of the vessel segments most proximal to the aorta contains vasa vasorum. These structures, which vascularize

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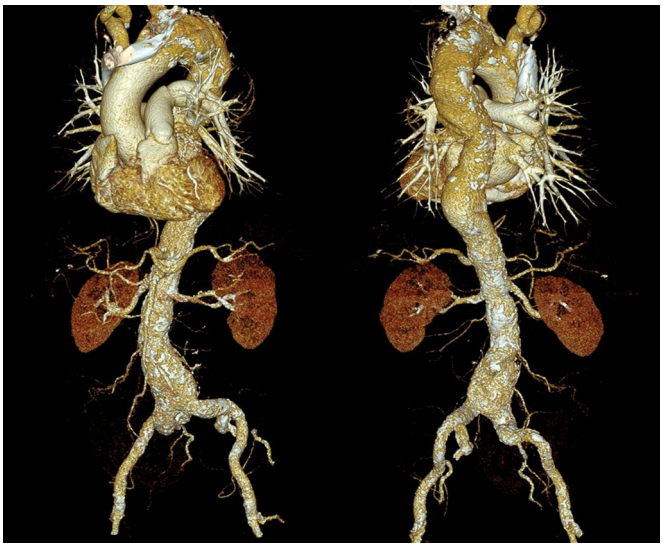

Figure 1. 3D reconstruction of the aorta showing severe, diffuse atherosclerosis associated with formation of aortic aneurysms and increased tortuosity of the vessel. Anterior view (left panel) and posterior view (right panel) of the aorta.

the vessel wall layers, are not found in the more distal segments. Hence, when atherosclerotic plaques are present, impeding proper diffusion and irrigation of the vessel wall from the lumen, hypoperfusion can occur in this region. This reduction in wall perfusion also facilitates atrophy and progressive dilatation, and helps to explain the greater frequency of aneurysms in the abdominal aorta.[10]

The aortic flow pattern is another factor that has an influence on aortic dilatation. A clockwise helicoidal flow pattern has been described at the level of the aortic arch that protects against plaque formation in the distal arch and the proximal descending aorta. In addition, the aorta shows a gradual physiologic narrowing known as *aortic taper*, in which the ascending aorta has a larger diameter than the descending aorta. This results in an acceleration of blood flow in the descending aorta, which avoids sluggish flow and the development of atherosclerosis. However, when the descending aorta progressively dilates as a consequence of diffuse aortic atherosclerosis, the blood flow slows and this favors progression of the atherosclerotic plaques.[11] Atherosclerosis, together with changes in the aortic wall composition and elasticity, lead to elongation and tortuosity of the aorta.[12] These alterations in the morphology and configuration of the vessel are associated with wall tension and flow distribution changes, which enhance the degenerative process.

Thus, there is a relationship between atherosclerosis and changes in aortic morphology. In a recent study in *Revista Española de Cardiología*, Craiem et al. were the first to describe a correlation between aortic calcification and the diameter of the aortic arch and descending aorta. These authors also found that the ascending aorta diameter did not correlate with the total aortic calcium, suggesting that aortic dilation may have different pathophysiologic mechanisms and require different prevention strategies.[13]

Despite considerable therapeutic advances in secondary prevention of atherosclerotic disease, achieving a prompt diagnosis of the condition in asymptomatic patients (ie, primary prevention) remains a challenge. That is why huge efforts have been made in recent years to develop and apply noninvasive diagnostic imaging techniques to promptly detect atherosclerotic disease.

DIAGNOSTIC IMAGING TECHNIQUES FOR EARLY DETECTION OF ATHEROSCLEROSIS

Based on the relationship between vascular calcification and atherosclerotic disease, several diagnostic imaging techniques have focused on detection and measurement of vascular calcification as a marker of the degree and severity of atherosclerosis. Radiologic study of vascular calcification by fluoroscopy was described at the end of the 1950s, and soon after, an association was established between vascular calcification and episodes of cardiovascular disease.[14] The later development (in the 1980s) of electron beam computed tomography and mutidetector computed tomography enabled vascular calcification to be precisely diagnosed and measured (Figure 2A).

Vascular calcium is measured using the Agatson score, which is the product of the area of each calcification (in pixels, mm^2) having computed tomographic attenuation of at least 130 Hounsfield units and the density score, obtained by rating the lesion on a scale.[15] The Agatston score has been widely used for studying coronary atherosclerosis[16] because it provides important prognostic information, as will be seen later on.

In an effort to establish prognostic factors and markers of cerebrovascular and peripheral vascular disease (in addition to

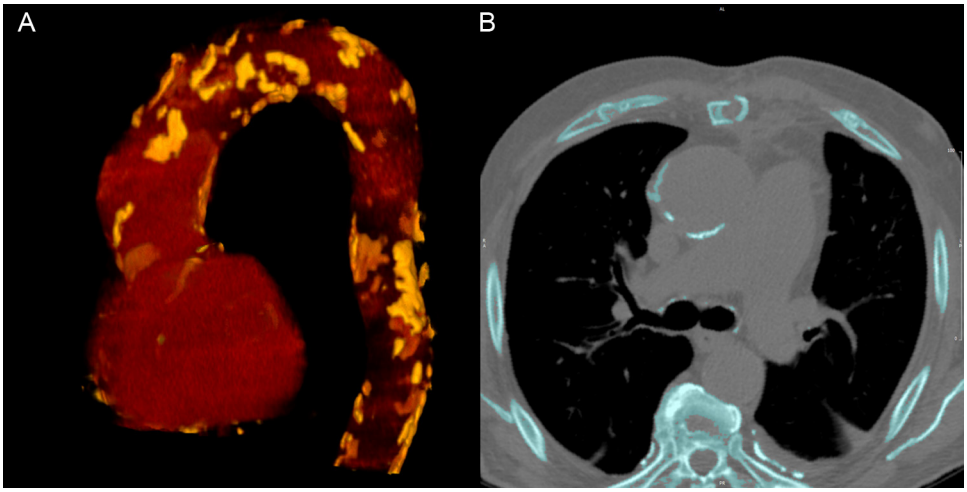

Figure 2. 3D reconstruction of an unenhanced computed tomography study showing severe, diffuse aortic calcification (A). Axial tomographic image depicting aortic calcification determined using the Agatston score (B).