Síndrome aterotrombótico hemodinámico sistémico: ¿es causa de enfermedad arterial coronaria no estenosante en el joven?

Systemic hemodynamic a atherothrombotic syndrome: is it the cause of non-stenosing coronary artery disease in the young?

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Cardiovascular diseases continue to be one of the main global health problems today [1,2]. The burden of disease they generate is very high and the impact they have on the quality of human life is catastrophic, especially because there is an increase in the incidence of cardiometabolic disorders from the earliest ages [1,2]. Non-stenosing coronary artery disease in the young is a lethal condition that must be managed rapidly to control hypoxemic and oxidative mechanisms at the cardiac tissue level and prevent myocardial necrosis [1]. Although the pathophysiology of ischemic heart disease and acute myocardial infarction are generally associated with established atherosclerotic disease, non-stenosing coronary artery disease is a dilemma in cardiology and emergency departments, due to the large number of different diagnoses and the difficulties in proper management [2]. One of the described causes of coronary vasospasm may be an anaphylactic reaction or Kounis syndrome [3]. However, in those patients with a sudden decrease in coronary blood flow, and in the absence of obvious causes and traditional risk factors, what should be suspected?

Systemic hemodynamic atherothrombotic syndrome (SHATS) is a recently described pathological entity, which is based on the hypothesis of resonance of blood pressure variability, and the impact it has in generating vascular disease and causing major cardiovascular events [4-6]. Kario K [7] states that from a neurobiological disorder, a vicious cycle of hemodynamic stress is generated that causes target organ damage, mainly heart, kidney and brain [7]. In SHATS, the pulse flow wave passes from the large arteries to the small arteries without slowing, causing inflammation by mechanical injury to the endothelium and blood vessel. In those patients with previous atherosclerotic disease, this damage is greater and allows progression to cardiovascular disease to occur rapidly [7-9].

The synergistic risk of blood pressure variability together with other risk factors that cause subclinical vascular disease is what could really explain the cardiovascular complications [10,11]. Finally, this hemodynamic stress results in arterial stiffness and altered baroreceptor sensitivity, as well as an inability to react adequately to physiological processes that control coagulation and oxidation, which can precipitate any type of vascular event, such as non-stenosing coronary artery disease [3-7].

Pepine et al [1] propose that within the mechanisms of non-stenotic coronary artery disease, there are large and small vessel disease, and within the latter group, endothelial dysfunction, microvascular dysfunction, endothelial cell-x cell "crosstalk", inflammation, and other thromboembolic causes not associated with atheroma plaques [1]. However, SHATS correlates on the basis of its molecular and inflammatory mechanisms with the possibility of producing non-stenosing coronary artery disease due to thromboembolic disorders unrelated to atherosclerotic plaque [3-7]. SHATS has several phenotypes and is influenced by epigenetic factors to which young people are exposed, such as: shortest beat-by-beat variability, diurnal variability changes modified by position, diet, alcohol, psychological stress, and physical activity; day-by-day variability, visit-to-visit variability, seasonal variability, and the longest yearly variability of BP, which are risk factors that may go unnoticed [7,9,10]. Therefore, it is feasible that even SHATS may be the first non-invasive, clinically detectable manifestation of established cardiovascular disease.

We propose the need for progress in both clinical and translational research in the understanding of SHATS and its different phenotypes such as the surge morning and nocturnal blood pressure, especially in young people, who increasingly present with major cardiovascular events at younger ages, may compromise their functional capacity and die. It is necessary to carry out studies of the highest quality to collect sufficient data to define cut-off points and new treatment schemes that facilitate the strict control of blood pressure disorders and control the incidence of cardiovascular morbidity.

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