Revista Portuguesa de Cardiologia 41 (2022) 1009–1010

Revista Portuguesa de
Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

EDITORIAL COMMENT

Thromboembolic risk in mitral stenosis: Are we underestimating the potential role of inflammation?

Risco trombo-embólico na estenose mitral: estaremos a subestimar o papel da inflamação?

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Available online 3 August 2022

The presence of spontaneous echo contrast (SEC) in the heart and great vessels and has been long recognized as an independent predictor of thromboembolic risk.[1,2]

In this issue of the Journal, Kelesoglu et al., from Kayeseri, Turkey[3] describe an association between the presence of spontaneous echo contrast (SEC) in the left atrium on the transoesophageal echo performed before percutaneous mitral valvuloplasty in patients with mitral stenosis and the value of the systemic immune inflammation index (SII). Patients with SEC had more severe disease: lower mitral valve area, higher LA volumes and higher degree of the estimated pulmonary artery systolic pressure and still multivariate logistic regression analysis demonstrated that an high value of SII was an independent risk factor for the occurrence of SEC. Somehow surprisingly the prevalence of atrial fibrillation (AF) was not different between the SEC positive and the SEC negative group and this raises the awareness of the other prothrombotic/inflammatory factors that may be involved.

In non-valvular AF, in addition to the hemodynamic changes associated with the arrhythmia, there are multiple causes to explain its unfavorable thromboembolic risk. These include malfunctioning of the clotting cascade, inappropriate/excessive platelet activation, abnormal hemostasis process and aberrant blood stasis and presence of structural heart disease.[4] The classic components of the Virchow's triad for thrombus formation: ''abnormal blood flow, abnormal vessel structure and abnormal blood constituents'' are present in non-valvular AF and probably play a role in patients with mitral stenosis even in the presence of sinus rhythm.

Systemic immune inflammation index is calculated as total peripheral platelets count (P)×neutrophil-to-lymphocyte ratio (N/L) (SII=P×N/L ratio). It is a simple and easily available inflammatory parameter. By including peripheral neutrophil, lymphocyte and platelet counts in one index, it may grossly reflect the balance between inflammation and immunity.

The prognostic impact of SII has been more extensively studied in oncologic diseases. Inflammatory responses have been confirmed to play decisive roles at different stages of cancer development and SII has been associated with poor outcomes in various types of cancer.[5]

Also in several studies, SII was found to be a prognostic marker in coronary heart disease, improving risk prediction.[6] It has also been recently described as an independent prognostic factor for persistent LV systolic dysfunction in patients with peripartumcardiomiopathy.[7]

Inflammation and thrombosis are physiological processes with an intense interdependence.

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https://doi.org/10.1016/j.repc.2022.07.008

In 2012 Engelmann et al.[8] introduced the concept of immunothrombosis, a physiological process that consists in an inflammation-dependent activation of the coagulation system, as a component of the host response to pathogens, aiming to limit their systemic spread in the bloodstream, thereby protecting host integrity without inducing major collateral damage. This response is achieved through an interplay between innate immune cells and platelets, triggering the activation of the coagulation system.

However, thromboinflammation — the aberrant and excessive activation of immunothrombosis — can be a trigger of non-infectious cardiovascular diseases, causing atherosclerotic plaque rupture or erosion, such as in acute myocardial infarction and stroke or stagnant blood flow, such as in venous thromboembolism and may contribute to the formation of thrombi in the left atrium, potentially causing embolic stroke or peripheral thromboembolism. A detailed list of the mechanisms involved in thrombo-inflammation is beyond the scope of this editorial, but the mutual activation of platelets and neutrophils resulting in clot formation and vessel occlusion plays a crucial role.[9]

In ischemic cardiopathy, the role of inflammation in the pathophysiology, disease progression and event occurrence is already strongly considered and increasingly taken into account in patient management.

The present study has the merits of raising awareness of the potential role of inflammation also in the setting of disease and specifically mitral stenosis.

Targeting inflammation to prevent cardiovascular events is a valuable current concept as an adjuvant therapy for ''classic'' antithrombotic medication and may also play an increasingly important role in valvular disease.

Conflicts of interest

The author has no conflicts of interest to declare.

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