Letter to the Editor

Assessment of Natriuretic Peptides: The Heart Should Be Evaluated as a Whole

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Dear Editor,

We read with great interest the article by Rodrigues et al[1] entitled "Right Ventricular Assessment by Tissue-Doppler Echocardiography in Acute Pulmonary Embolism", which was published of the Arquivos Brasileiros de Cardiologia. The authors[1] aimed to evaluate the right ventricular (RV) systolic function using two-dimensional tissue Doppler echocardiography in patients with acute pulmonary embolism (PE) and to analyze its correlation with atrial natriuretic peptide (BNP) levels. The authors concluded that increased BNP levels (≥ 50 pg/mL) were associated with a higher prevalence of RV systolic dysfunction in patients with PE. Although the current article is well-managed, and we commend the authors for the detailed and valuable information that they have provided, some comments may be beneficial.

Natriuretic peptides are predominantly secreted by cardiac myocytes in response to myocardial stretch induced by volume load[2,3]. They act to increase natriuresis and decrease vascular resistance, thereby decreasing blood volume, systemic blood pressure and afterload[2]. Atrial natriuretic peptide is synthesized primarily by the atrial myocardium. Natriuretic peptide concentrations are associated with atrial and atrial appendage functions as well as with the severity of mitral and aortic valve regurgitation and stenosis[2-5].

In addition, natriuretic peptide levels have been shown to be higher in hyperthyroid, hypertensive and obese patients, due to the direct stimulatory effect of thyroid hormones, left ventricular hypertrophy and increased afterload and insulin resistance, respectively[5].

In conclusion, although natriuretic peptide levels are associated with ventricular functions, other factors which could increase peptide levels should be assessed. If information on thyroid hormone levels, body mass indexes and mean blood pressure levels of patients was provided, as well as on the assessment of mitral and aortic valves (regurgitation and stenosis) and of atrial volumes (left and right), which could alter the results, the study could be more valuable and the information obtained from the study could add more consistency to the results.

Keywords

Natriuretic Peptides; Ventricular Function, Right; Pulmonary Embolism.

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Reply

We would like to thank Yalcinkaya et al[1] and coworkers for the worthy commentaries on our recent article[1]. Brain natriuretic peptide (BNP) is produced primarily in ventricular myocytes, and elevated levels are found mainly in patients with increased left ventricular and pulmonary artery pressure. It has been regarded as a useful marker for both the diagnosis and severity of heart failure. We certainly agree that BNP is increased in a variety of conditions apart from heart failure, such as hypertension, left ventricular hypertrophy, pulmonary hypertension, thyroid disease and cirrhosis. However, our aim was to assess BNP levels in a selected group of patients with acute pulmonary thromboembolism. Because elevated plasma BNP is a powerful marker of left ventricular systolic dysfunction, we excluded patients with that condition. Furthermore, our population did not comprise patients with significant valve disease, which notably affects BNP levels. We did not test our population for thyroid disease, although, in experimental studies, thyroid function has a modest but significant effect on NT-proBNP concentration, suggesting thyroid hormones can enhance synthesis of atrial peptide hormones[2]. On the other hand, Kato et al[3] have stated that cardiovascular condition is the major factor responsible for serum BNP elevation in thyrotoxic patients, while thyrotoxicosis itself is a significant, but minor contributor. The prevalence of overt thyroid dysfunction is about 5% in the general population[4]; thus, testing thyroid function to check its influence on BNP would hardly be acceptable in patients with pulmonary thromboembolism. In fact, natriuretic hormones are affected not only by cardiac function but also by activation of neuro-hormonal and stress-related cytokines, in which case, all would have to be tested[5]. On reviewing key studies using BNP for the diagnosis of heart failure, we have found no citations on thyroid hormones[6].

Finally, we agree that the specificity of BNP is limited, because it is elevated in a large number of clinical situations that may coexist, and adjustments for the independent effects are possibly required.

Sincerely,

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