

Differential diagnosis of cardiomyopathies: Utility of cardiac magnetic resonance imaging

Kardiyomiyopatilerin ayırıcı tanısı: Kardiyak manyetik rezonans görüntülemenin işlevselliği

To the Editor,

We read with great interest the article by Kurtul et al. (1) entitled "Acute coronary syndrome with intraventricular thrombus after using erythropoietin," which is published in the current issue of The Anatolian Journal of Cardiology.

These authors (1) presented a case of acute coronary syndrome (ACS) with intraventricular thrombus associated with erythropoietin (EPO). They discussed that increased erythropoietin levels and intracardiac/intracoronary thrombus might be associated with abruptly increased hematocrit values, enhanced platelet activation and endothelial stimulation, and reduced coagulation inhibitors. Some comments may be of interest.

Increased EPO dose was significantly associated with the composite endpoint of death, heart failure, stroke, or myocardial infarction and the effect of EPO on blood pressure in normal humans is independent of its effect on hematocrit. Increment in plasma noradrenaline, endothelin-1, thromboxane-B2 concentrations, reactivity to exogenous noradrenaline and platelet α_2 -adrenoceptor densities can be observed in patients using EPO (2, 3).

Takotsubo cardiomyopathy is a cause of reversible left ventricle (LV) systolic dysfunction in mid to apical segments. It presents with a myocardial infarct-like clinical syndrome and it is often preceded by emotional stress or exacerbation of an existing medical condition. LV systolic function and patients' symptoms tend to normalize approximately in a week (4).

Increment in sympathetic activity associated with high dose of erythropoietin can result in Takotsubo cardiomyopathy or coronary ischemia via vasospasm. On cardiac magnetic resonance (CMR) imaging, whereas Takotsubo cardiomyopathy is characteristic in no or minimal late gadolinium enhancement (LGE), myocardial infarction is characteristic in subendocardial LGE, which extends variably transmurally to the epicardium (4, 5).

When etiology remains unclear, CMR appears to be a useful imaging modality for documenting the extent of the regional wall motion abnormality and differentiating cardiomyopathies from each other.

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Author's Reply

To the Editor,

We reported a case report titled "Acute coronary syndrome with intraventricular thrombus after using erythropoietin" in the May 2013 issue of The Anatolian Journal of Cardiology (1). We read with interest the letter to the editor about our case report. We thank for comments and we would like to reply this important comments on our article.

We reported a case of acute coronary syndrome (ACS) with intraventricular thrombus associated with erythropoietin (EPO). We discussed that prothrombotic effect of EPO might associated with abruptly increased hematocrit values, enhanced platelet production or reactivity, stimulation of endothelial cells or reduced coagulation inhibitors (1).

We supported the underlying mechanisms about erythropoietin worse outcome which put forward by the authors such as increasing of noradrenaline, endothelin-1 and thromboxane-B2 (2, 3). As they pointed out, increment in sympathetic activity associated with high dose of erythropoietin can result in Takotsubo cardiomyopathy (TCM) or coronary ischemia via vasospasm. TCM is a rare clinical entity, having clinical and electrocardiographic findings very similar to those found in acute myocardial infarction (4). Kim et al. (4) reported a case of Takotsubo cardiomyopathy with apical thrombus as a source of cardioembolic cerebral infarction. However, Haghi et al. (5) evaluated 63 consecutive patients with TCM with intravascular ultrasound (IVUS) and they did not find intracoronary thrombus in that patients. We examined the literature and we did not find any evidence that intracoronary thrombosis in these patients.

As they pointed out cardiac magnetic resonance (CMR) may be used to imaging modality for documenting the extent of the regional wall motion abnormality and differentiating different etiologies. But unfortunately, in our case, we did not evaluate the patient with CMR.

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The protection of therapeutic lifestyle change in individuals with prehypertension; a valuable study

Terapotik yaşam tarzı değişikliğinin prehipertansif bireylerdeki koruyuculuğu; dikkate değer bir çalışma

To the Editor,

We read the article "Effect of lifestyle modifications on diastolic functions and aortic stiffness in prehypertensive subjects: a prospective cohort study" published-written by Alpsoy et al. (1) with great interest. Recent studies have shown that hypertension has a very important role on atherosclerosis, cardiovascular disease (CVD) and deaths. Hypertension has an increasing prevalence and is one of the leading causes of preventable deaths (2, 3). Prehypertension has been defined on JNC-7's latest report, and was shown to be associated with increased MI and coronary artery disease (CAD) rates (3). The development of CVD is mainly caused by endothelial dysfunction, vascular inflammation and atherosclerosis (4). Atherosclerosis is characterized by a decrease in the elasticity and diffuse thickening of the vessel wall. Studies have shown that patients with prehypertension have increased atherosclerosis with increased systemic inflammation (3). Therapeutic lifestyle changes (TLSC) are recommended today in almost all guidelines (JNC 7, the ATP III and so on.), and has been replaced as the main treatment in hypertension and other CVDs.

The study deserves emphasizing in terms of the design and presentation, and we would like to thank to the authors. However, we would have a few matter of criticism, especially in methods section of the study, the demographic data of the patients have been given a bit superficially. For example, the data seems missing about how much of individuals take alcohol, or how long; how long have they smoked (pack/year will be more

accurate), liver and renal function test results, and so on. At the end of the study it is understood that there is not a decrease in an expected level, such as weight and BMI. It is not fully specified why this occurs and why participants could not comply with TLSC fully. Should it be considered in the form of a continued exercise of 180 hours a week because of the lack of an illuminating data at the introduction and results of the study about the exercise of all patients? Again, a proper exercise should increase HDL levels. Should the lack of a significant amount of increase in HDL levels show the existence of a problem with the alignment of exercise? Perhaps the effectiveness of weight loss and exercise could be more easily interpreted if the insulin resistance (HOMA-IR) were executed (5).

Na restriction (100 mmol/day) have been conducted to individuals participating in the study. It was not fully specified how it was evaluated quantitatively, with 24-hour urinary Na values at the beginning and at the end of study. As you know, our country ranks high in salt consumption (SALTURK 1-2). It would be more meaningful if the quantitative reduction of salt intake was presented. The basic benefit in this study is thought to arise because of the restriction of salt intake. It should be taken into account that consumption of high amounts of salt especially leads to increase in the preload and diastolic overload. It is understood that a portion of the individuals are smokers, but how much of these individuals reduced the amount of cigarettes during the study period, or was a recommendation performed for the stopping smoking? Salt intake and smoking play a role in atherosclerosis and hypertension directly as well as indirectly.

Finally, we think that the study would become stronger if data about systemic inflammation (hsCRP, CRP, STWEAK, etc.) and insulin resistance were added to the study.

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