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Right ventricular functions in obstructive nasal polyposis

To the Editor,

We have read with great interest the article entitled "Evaluation of right ventricular functions in patients with nasal polyposis: an observational study" by Şimşek et al. (1) as it highlighted an important issue about the effect of nasal polyposis and nasal obstruction on cardiac functions specially the right ventricle and stress on the idea that hypoxia resulted from nasal obstruction has harmful effects on cardiac functions.

We have some considerations;

First regarding study design in the methods section, authors had informed that the type of study was cross sectional prospective study, however prospective study is a kind of study where an outcome or event is studied and measured for its occurrence in a specific period or time and as far as we read the article authors did not measure an outcome in a specified period of follow up.

The correlation between hypoxia due to upper airway obstruction and deterioration of cardiac functions had been already proved, however we have a great interest to know if there is a correlation between the degree of obstruction and the peak systolic pulmonary artery pressure as the authors choosed different levels of nasal obstruction in their study population, Stage 2: Intermediate polyposis, and Stage 3: Severe polyposis.

This study gains its importance from being the first which discuss the relation between the presence of nasal polyposis and its grades with the right ventricular functions where it concludes that patients with nasal polyposis who are clinically asymptomatic and have normal right ventricular functions with conventional echocardiography have subclinical right ventricular longitudinal dysfunction with strain and strain rate echocardiography. We would prefer to know the arterial blood gases results regarding arterial oxygen tension and hypoxia level along with its correlation with nasal polyps grading. Finally we propose the multivariate regression analysis as a statistical method to know if nasal polyps are dependent or independent from hypoxia as a cause of subclinical right ventricular functions deterioration along with if surgical removal of nasal polyps has a good prognostic effect on the right ventricular functions or not.

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Author's Reply

To the Editor,

We thank for interest and positive reviews in our article published in the Anatolian Journal of Cardiology (1).

Nasal poliposis is a chronic inflammatory disease and the most common cause of nasal mass which leads to nasal obstruction. The most commonly used staging method is made by endoscopic appearence. For this staging method, Stage II defined as the polip which protrudes under the middle concha and could be seen without an endoscope, and Stage III defined as massive poliposis (2). In our article comparison of Stage 2 and Stage 3 NP patients revealed that only the SR value for the RV mid segment was significantly different (p=0.02); other segments did not show a significant difference in S and SR values (1).

Hypoxi and hypercapnia reported in various studies with patients who had nasal obstruction arised from a disease or an anterior and/or posterior nasal packing (3, 4). Despite the fact that arterial blood gas analysis is an objective method for determining hypoxemia, but also this is an invasive method. The studies which evaluated the cardiac effect before and after the operation in nasal poliposis patients reported significant improvement in pulmonary arterial pressure after the operation (5). We thought there is a need to the extensive studies for detailed evaluation of the subject.

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Does bilirubin level have an effect on cardiac parameters?

To the Editor,

We read the article "The effect of Gilbert's syndrome on the dispersions of QT interval and P-wave" written by Cüre et al. (1) published in The Anatolian Journal of Cardiology with great interes.

The authors aimed to investigate the effects of bilirubin levels on noninvasive electocardiograhic parameters. They concluded that increased bilirubin levels are associated with decrease in HR, Pd and QTd in Gilbert's syndrome (1). Thanks to the authors for their contribution.

We know that P wave and QT disperion are used for the prediction of atrial and venticular arrhythmias (2). Increased P wave dispersion gives us information about intraatrial and interatrial conduction delaying (3). Several studies have shown that an increased QT dispersion and/or QTc dispersion could be a marker for arrhythmic events, myocardial infarction, and sudden death.

P-wave and P-wave dispersion measurement of distances must be very sensitive. While some centers can measure automatically measurements are usually made manually. Therefore, analysis of intraobserver and interobserver differences are important. Increase of P wave duration resulting from the increase of interatrial and intraatrial conduction time due to atrial expansion causes a predisposition for arrhythmias. In a study, a mathematical formula has been developed associated with left atrial expansion and P wave duration as left atrial diameter (cm)= $2.47\pm0.29 \times p$ wave duration (mm) (4). So P and QT measurements would be supported by echocardiographic measurements of the left atrium and left ventricle.

It is also important to measure the ΩT duration and ΩT dispersion but the calculated ΩT corrected according to the heart rate is able to provide more accurate information. The authors should pay attention to these issues.

The cardioprotective effect of bilirubin is well known but the relationship between cardioprotection and bilirubin levels are unknown (5). It would be useful evaluating the correlation between bilirubin levels and Pd, HR and QTd in this study.

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Author`s Reply

To the Editor,

We thank Authors for the interest they have shown in our article published the Anatolian Journal of Cardiology (1).

Firstly, all electrocardiographic measurements were performed by a cardiologist and an internist who were not aware of the diagnosis of the patients. Clearly, the measured values of the QT interval were based on the shape of the descending part of the T wave. T wave offset determined by manual method is very unreliable. Unfortunately, available automatic methods have not been shown to have any advantage (2). In our study, echocardiography (echo) was not performed in the patients. So we can not make a comparison between P wave dispersion (Pd) and QT dispersion (QTd) with echo findings. In fact, echo findings might have enriched the study. Our study was performed as a pilot study.