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Evaluating the effect of nasal septoplasty on atrial electromechanical features

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ABSTRACT

Background: Recent studies have demonstrated that right ventricular (RV) dysfunction and increased pulmonary artery pressure may be frequent in patients with upper airway obstruction. In this study, we evaluated atrial conduction delays in patients with upper airway obstruction secondary to nasal septum deviation (NSD).
Methods: A total of 32 patients with upper airway obstruction secondary to NSD undergoing a septoplasty procedure were enrolled in this study. Preoperative electrocardiography and transthoracic echocardiography were performed in all patients who underwent surgery. The mean pulmonary artery pressure (mPAP) and atrial conduction time (ACT) were recorded before and 6 months after the surgical procedures.
Results: The PAP was significantly lower postoperatively than preoperatively (20.75 ± 4.83 vs. 24.68 ± 5.26; $P < 0.001$). The postoperative Electromechanical Delay of Mitral septal wall (EMD-MS) value was significantly lower than that preoperatively (46.20 ± 8.5 vs. 40.5 ± 9.9; $P < 0.001$). The postoperative Electromechanical Delay of Mitral lateral wall (EMD-ML) value decreased significantly compared to the preoperative period (46.3 ± 7.4 vs. 40.6 ± 9.3; $P < 0.001$). The postoperative Electromechanical Delay of Tricuspit lateral wall (EMD-TL) value was significantly lower than that preoperatively (43.8 ± 7.0 vs. 38.1 ± 9.1; $P < 0.001$). There was no significant change in pre- or postoperative measurements of inter- and intra-atrial conduction delays.
Conclusions: We demonstrated that upper airway obstruction secondary to NSD causes a significant increase in mPAP and a significant delay in ACT, which improved after nasal septoplasty. According to these results, we conclude that upper airway obstruction may be an important risk factor for pulmonary arterial hypertension, RV dysfunction, and atrial arrhythmias, especially in unoperated cases.

1. Introduction

Nasal septum deviation (NSD), adenoid hypertrophy, and obstructive sleep apnea syndrome (OSAS) may lead to upper airway obstruction (UAO) and cause a secondary increase in pulmonary artery pressure (PAP) [1,11]. UAO may reduce quality of life and worsen work efficiency. UAO is associated with pulmonary and cardiovascular dysfunction [1,9–11]. Severe UAO may cause hypoxia, hypercarbia, respiratory acidosis, and/or bronchospasm. This condition leads to an increase in PAP and subsequent right ventricular (RV) dysfunction long-term and is known as “cor pulmonale” [1,3,9,10]. On the other hand, atrial dilatation may impede atrial action potentials and thus affect atrial electromechanical delays [2,5,8]. The delay in atrial action potentials inhibits the transmission of atrial conduction homogeneously and leads to an atrial electromechanical delay (AEMD) [8].

The diagnosis of AEMD is made with transthoracic echocardiography (TTE) with one-lead electrocardiogram (ECG), and atrial conduction time (ACT) can be measured simultaneously with ECG monitorization. Additionally, TTE is the most common tool used to assess right heart chambers, and PAP can be easily and accurately estimated via TTE. Atrial EMD is considered an important risk factor for atrial tachycardia and atrial fibrillation (AF) long-term [6,7]. Previous studies have mainly focused on the association between AEMD and chronic obstructive pulmonary disease (COPD) and OSAS [2,12]. However, there is a lack of evidence on the effects of NSD on AEMD in adults. In this study, we aimed to reveal changes in PAP and AEMD in patients with UAO secondary to NSD undergoing a septoplasty procedure.

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2. Methods

This was a single-center prospective study. Patients who were diagnosed with UAO due to NSD (n = 32) were enrolled in the study in a consecutive manner. The age of the patients ranged from 15 to 65 years. The study was performed in cooperation with the cardiology and otolaryngology department. The duration of the study was 12 months (from January 2015 to December 2015). Ethical approval was obtained from the Ethics and Research Committee of our hospital. All participants provided written informed consent prior to their enrollment. The research was conducted in accordance with the principles of the Declaration of Helsinki.

All patients were examined by a trained cardiologist and otolaryngologist. Their previous health issues and clinical and demographic properties were recorded at the first examination. All patients were evaluated pre- and postoperatively in both departments. Airway examination was assessed via rhinoscopy and flexible transnasal endoscopy. Patients underwent a paranasal sinus computed tomography scan before the septoplasty procedure. Patients with conditions associated with upper and lower airway obstruction, such as COPD, adenoid hypertrophy, and asthma, were excluded from the study. Routine blood analyses and a chest x-ray were performed preoperatively. A 12-lead ECG was recorded pre- and postoperatively. All patients underwent the septoplasty procedure (Cottle method) under general anesthesia. Among the 32 patients included in the study, 23 were male and 9 were female.

2.1. Echocardiographic examination

Conventional echocardiographic and tissue Doppler imaging (TDI) studies (Vivid 7 Pro, 3-MHz transducer; GE, Horten, Norway) of all patients and control subjects were performed by a cardiologist blinded to the clinical details. A one-lead ECG was recorded continuously during the echocardiography examination. All echocardiographic measurements were averaged over three consecutive beats. The mean pulmonary arterial pressure (mPAP) was calculated using the following Mahan formula [9]: mPAP = 79 − 0.45 × PVAT (pulmonary velocity acceleration time) [4].

2.2. Atrial electromechanical – condition delay measurement

In this study, AEMD was defined as the interval from the onset of atrial electrical activity (P-wave on surface ECG) to the beginning of the mechanical atrial contraction (A-wave) (Fig. 1). AEMD was measured from the mitral lateral, mitral septal, and tricuspid lateral annulus from apical-4-chamber views and named AEMD-ML, AEMD-MS, and AEMD-TL, respectively. AEMD was measured from the same cardiac segments described earlier [19].

2.3. Statistical analysis

Categorical variables are expressed as numbers and percentages. Pearson's chi-square or Fisher's exact tests were used to evaluate differences between groups. Before evaluating the differences between the pre- and postoperative measurements, the Kolmogorov–Smirnov test was used to test for data normality. Descriptive statistics of the pre- and postoperative measurements of the variables with a normal distribution are presented as the mean ± standard deviation. A paired sample *t*-test was used to compare the pre- and postoperative measurements. Descriptive statistics of the pre- and postoperative measurements of variables with a skewed distribution are presented as the median (25th–75th percentile). The Wilcoxon test, a non-parametric test, was used to compare the pre- and postoperative measurements. $P < 0.05$ was considered statistically significant. Analyses were performed using Statistical Package for Social Sciences software, version 20.0 (SPSS; IBM, Armonk, NY, USA).

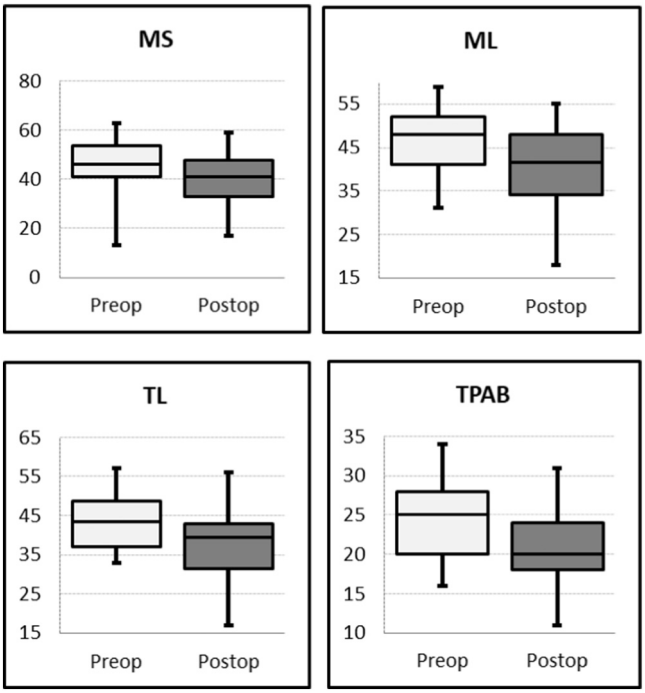

Fig. 1. Atrial electromechanical – condition delay measurement. AEMD was defined as the interval from the onset of atrial electrical activity (P-wave on surface ECG) to the beginning of the mechanical atrial contraction (A-wave).

Table 1
The patients' baseline characteristics.

	n	%
Female	9/32	%28,1
Male	23/32	%71,9
Hypertension	8	25
Diabetes	4	12,5
Cigarettes	14	43,8
Hyperlipidemia	4	12,5
Coronary disease	2	6125

	Median	Mean ± SD
Age	38,5	37,8 ± 14,0
Length	171,0	170,8 ± 8,6
Weight	77,0	76,8 ± 13,9
BMI	25,8	26,3 ± 4,4

3. Results

The patients' baseline characteristics are listed in Table 1. A total of 32 patients (mean age 37.8 ± 14.0 years; 28.1% female) with NSD who underwent nasal septoplasty surgery were included. The patients' pre- and postoperative echocardiographic results are shown in Table 2. The patients' pre- and postoperative heart rates were similar according to ECG analysis. The patients showed no significant change in basic echocardiographic measurements but a lower postoperative mPAP compared to preoperative mPAP (20.75 ± 4.83 vs. 24.68 ± 5.26; $P < 0.001$). The patients' postoperative AEMD-ML, AEMD-MS, and AEMD-TL were significantly lower compared to their preoperative measurements (AEMD-ML, 46.3 ± 7.4 vs. 40.6 ± 9.3; $P < 0.001$; AEMD-MS, 46.20 ± 8.5 vs. 40.5 ± 9.9; $P < 0.001$; AEMD-TL, 43.8 ± 7.0 vs. 38.1 ± 9.1; $P < 0.001$). We also compared the pre- and postoperative measurements of inter- and intra-atrial conduction delays, but there was no significant difference between measurements (Fig. 2).

Table 2
The patients' pre- and postoperative echocardiographic results.

	Mean ± SD	P
Heart rate		
Preop	70,3 ± 9,3	0,878
Postop	70,5 ± 9,2	
MS		
Preop	46,2 ± 8,5	0,000
Postop	40,5 ± 9,9	
ML		
Preop	46,3 ± 7,4	0,000
Postop	40,6 ± 9,3	
TL		
Preop	43,8 ± 7,0	0,000
Postop	38,1 ± 9,1	
TPAP		
Preop	24,4 ± 5,2	0,000
Postop	20,8 ± 4,8	
İnter ACD		
Preop	1781 ± 6913	0,873
Postop	1969 ± 4604	
İntra ACD		
Preop	2344 ± 5889	0,945
Postop	2438 ± 5447	
LVEF		
Preop	63,6 ± 4,5	0,36
Postop	64,8 ± 5,1	
RADD		
Preop	31,1 ± 2,3	0,39
Postop	30,7 ± 2,7	
RVDD		
Preop	25,6 ± 4,2	0,31
Postop	23,9 ± 3,9	

4. Discussion

In the current study, we evaluated pre- and postoperative atrial conduction features in 32 patients who underwent a nasal septoplasty procedure due to NSD. We demonstrated that AMED-ML, AMED-MS, and AMED-TL were significantly reduced after nasal septoplasty surgery. To our knowledge, this is the first study to investigate atrial condition features in adult patients with NSD that lead to partial upper respiratory tract obstruction.

Chronic UAO may cause hypoxia, hypercarbia, and/or respiratory acidosis. Respiratory acidosis and hypoxia may cause reversible or irreversible changes by inducing vasoconstriction in the pulmonary vascular bed. Increased pulmonary vascular resistance leads to pulmonary hypertension, RV dysfunction, and eventually cor pulmonale. Previous studies have reported that UAO, such as adenotonsillar hypertrophy and sleep apnea, result in pulmonary hypertension and cor pulmonale [13,16,17]. NSD is another frequent cause of obstruction of the upper airways.

Hassan pour et al. noted that mPAP was higher in patients with a markedly deviated septum who were undergoing septorhinoplasty [15]. Fidan et al. found that mPAP was increased in patients with a markedly deviated septum but decreased after septoplasty [14]. Thus, our findings are consistent with the results of previous studies. However, the superiority of the present study is the assessment of ACT.

RV function is considered a major determinant of the prognosis and symptoms in patients with pulmonary hypertension [18]. Previous studies have revealed that diseases that cause UAO, such as OSAS, adenotonsillar hypertrophy, and NSD, increase mPAP and disturb RV functions [1,9,15,19,20].

El-Moneim et al. reported that the correction of UAO is associated with improvements in RV filling pressure and a decrease in PAP [1].

However, our results are consistent with previous studies that evaluated changes in mPAP in patients with adenotonsillar hypertrophy who underwent surgery. However, most of these studies were performed in pediatric patients [10,11]. Furthermore, our patients were adults and we examined changes in AEMD that have not been previously evaluated in these patients.

Few studies have investigated the effects of adenotonsillar hypertrophy and OSAS on the myocardial performance index (MPI) in children and adults. Duman et al. found that RV MPI was significantly higher in the patient group than in the control group ($P < 0.001$) and was strongly correlated with mPAP and OSAS scores in children [14].

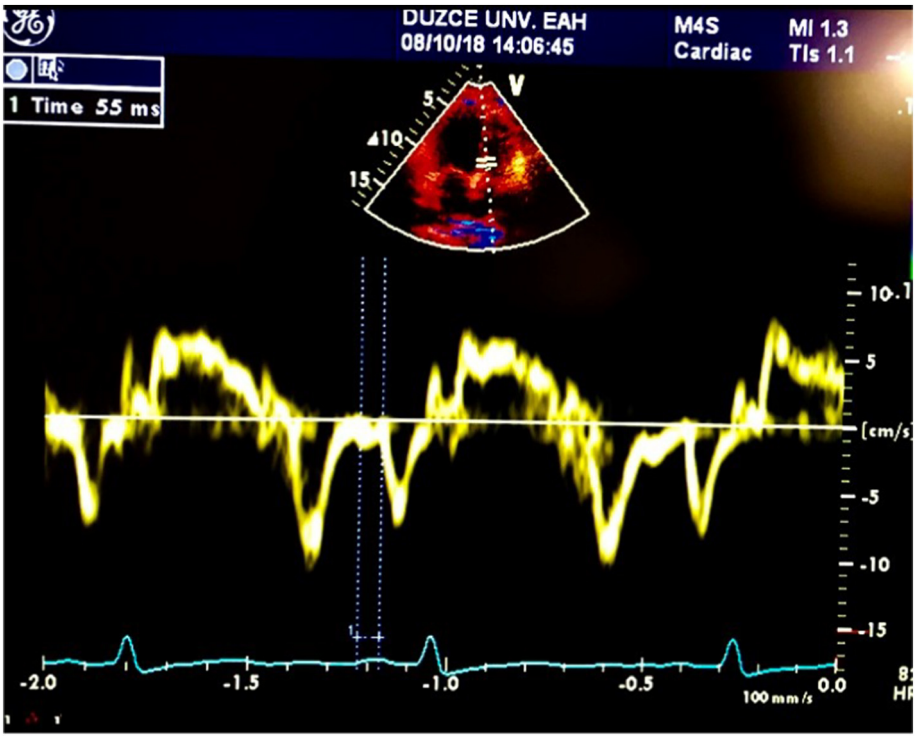

Fig. 2. Changes in atrial electromechanical times and mPAP.

Similarly, Koc et al. showed a significant decrease in MPI and mPAP values 3 months after adenotonsillectomy in children with adenotonsillar hypertrophy [16]. While most researchers have focused on RV function in patients with adenotonsillar hypertrophy, biventricular echocardiographic evaluation has been performed in only a limited number of studies. Attia et al. investigated the impact of OSAS and adenotonsillectomy on global myocardial performance in children with adenotonsillar hypertrophy. They demonstrated that patients with OSAS had higher systolic pressure and mPAP, RV end-diastolic diameter, pulmonary vascular resistance, and left ventricular mass index [2].

The surface ECGs of the patients examined in our study did not reveal any significant arrhythmia. However, we did not perform 24-h rhythm monitoring to assess the rhythm disturbances during sleep; an increase in AEMD delay is reported to be an important risk factor in the pathophysiology of AF. AEMD duration prolongation causes AF by affecting the homogeneous spread of sinusoidal waves [8,12].

In this study, it was similarly observed that AEMD shortened significantly after the operation, similar to the study of Yagmur et al. [12]. They showed that AEMD, specifically intra-ACT, extends significantly in adult patients with moderate to advanced OSAS. İn this study, there was no significant change in ACT, which may have been due to the higher PAP and UAO in Yagmur's study.

den Uijl et al. showed that an increased total atrial conduction time assessed by TDI echocardiography is an independent predictor of AF recurrence after radiofrequency catheter ablation for paroxysmal AF [7]. Similar results were obtained in the present study.

In conclusion, the findings from the present study showed that the mean AEMD times and PAP decreased 6 months after septoplasty operation. NSD may lead to UAO, which may cause an increase in pulmonary vascular resistance, PAP, and mPAP and may negatively affect RV functions. Increased AEMD may be predictive of AF. Therefore, patients with NSD should have a thorough cardiac evaluation and should be encouraged to undergo septoplasty.

Study limitations

First, a relatively small number of patients was examined, which may have affected the power of the study. Second, although the ECGs of the patients were normal and the patients had no history of palpitations or syncope, 24-h rhythm Holter monitoring was not performed to assess the rhythm during sleep.

Conflict of interest

All authors declare no conflict of interest.

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