Effects of Nasal Septum Deviation and Septoplasty on Cardiac Arrhythmia Risk

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Abstract

Objective. Upper airway obstruction (UAO) can result in cardiac complications, including arrhythmias and sudden cardiac death. Nasal septum deviation (NSD) is a common cause of UAO. The aim of this study was to assess the risk of cardiac arrhythmias in patients with NSD. To assess this risk, we measured noninvasive indicators of atrial arrhythmia (P-wave dispersion [Pd]) and ventricular arrhythmia (corrected QT dispersion [QTcd]) and compared these values between NSD patients and healthy subjects.

Study Design. Prospective study.

Settings. Tertiary referral center.

Subjects and Methods. This study included 53 consecutive patients who had underwent septoplasty due to marked NSD. Electrocardiographic records were used to determine Pd and QTcd values preoperatively and 6 months postoperatively. Fifty-three consecutive age- and sex-matched subjects without any UAO were also examined as a control group.

Results. Preoperative Pd and QTcd values were significantly higher in NSD patients than in the control group (Pd: 57.40 ± 14.21 vs 34.11 ± 7.12 milliseconds, \(P < .001\); QTcd: 81.77 ± 16.39 vs 50.25 ± 11.51 milliseconds, \(P < .001\), respectively). In addition, Pd and QTcd values were significantly greater in preoperative NSD patients when compared with the same patients postoperatively (Pd: 57.40 ± 14.21 vs 36.32 ± 8.9 milliseconds, \(P = .013\); QTcd: 81.77 ± 16.39 vs 55.76 ± 11.4 milliseconds, \(P = .012\), respectively).

Conclusion. In conclusion, NSD patients are at risk for both atrial and ventricular cardiac arrhythmias; however, septoplasty in these patients can relieve UAO and reduce the risk of arrhythmias.

Keywords

airway obstruction, atrial arrhythmia, cardiac arrhythmia, electrocardiography, nasal septum, ventricular arrhythmia

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Cardiac complications due to upper airway obstruction (UAO) have been previously investigated. Studies revealed a strong association between UAO and heart rhythm disorders.\(^1,2\) Factors thought to contribute to cardiovascular morbidity in individuals with UAO are enhanced oxidative stress, sympathetic nervous system activation, and exaggerated negative intrathoracic pressure swings.\(^2,3\) One of the most common causes of UAO is nasal septum deviation (NSD).\(^4,5\) The prolongation of P-wave dispersion (Pd) has been observed in adult patients with adenotonsillar hypertrophy and obstructive sleep apnea (OSA).\(^6,7\) However, the risk of arrhythmia in NSD patients has not been studied in detail.

QT and Pd dispersion parameters can indicate abnormalities in the autonomic nervous system and cardiac function. Pd is defined as the difference between the longest and shortest P wave durations recorded from multiple electrocardiogram (ECG) leads. Pd reflects the inhomogeneous propagation of sinus impulses. Thus, Pd has been performed in the assessment of the risk for atrial fibrillation, which is characterized by inhomogeneous and discontinuous atrial conduction.\(^8,9\) QT dispersion is defined as the difference between the longest and shortest QT intervals on the 12-lead ECG. Heart rate–corrected QT dispersion (QTcd) is an indirect measure of the heterogeneity of ventricular depolarization, which may contribute to ventricular arrhythmias.\(^10\) Increased QTcd has been shown to be correlated with the risk of arrhythmic death in a variety of cardiac or noncardiac disorders.\(^11\) Therefore, the use of QT dispersion to evaluate an individual’s susceptibility to ventricular arrhythmias has become a standard for many noncardiac conditions.\(^12-15\)

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The aim of this study was to quantify the risk of atrial and ventricular arrhythmias in NSD patients and to examine the effect of septoplasty on arrhythmia risk in these patients by measuring Pd and QTcd values during the pre- and postoperative periods.

Materials and Methods

This study adhered to the guidelines of the Helsinki Declaration of the World Medical Association and was approved by the research ethics committee at Van Training and Research Hospital (no. 2015/7). Written informed consent was obtained from all participants prior to the study.

The prospective study involved 53 consecutive patients (18 women and 35 men) who underwent septoplasty due to marked C- or S-shaped NSD from November 2014 to March 2015. The control group was composed of 53 consecutive healthy age- and sex-matched subjects (20 female, 33 male) without NSD or other UAO reasons.

All participants were evaluated with the same systematic protocol. For the diagnosis of presence or absence of NSD or other UAO, a detailed otorhinolaryngologic examination was made—including Mallampati classification, Friedman tongue position, anterior rhinoscopy, and endoscopic nasal, nasopharyngeal and hypopharyngeal examinations—and obstructive symptoms were questioned with the Epworth Sleepiness Scale (ESS).

We excluded patients with mild NSD, isolated unilateral NSD, NSD with turbinate hypertrophy, UAO-related conditions (ie, airway masses, vocal cord paralysis, adenotonsillar hypertrophy, or OSA), a history of cardiac disease (ie, arrhythmia, heart valve disorders, previous cardiac surgery, myocardial infarction, congestive heart failure, bundle branch block, or cardiomyopathy), medical conditions that may have disrupted the cardiac conduction systems (ie, hypertension, diabetes mellitus, hyperlipidemia, or thyroid disease), long-term drug usage for chronic disease, and tobacco use.

To identify patients with OSA for the purpose of exclusion, we examined patients with portable overnight polysomnography (Itamar Watch-Pat200; Itamar Medical, Caesarea, Israel), and we evaluated patient scores from the ESS, the Mallampati classification, and Friedman tongue position. Polysomnography examination was performed only in the study group. Patients with an apnea-hypopnea index >5, an ESS score >10, a Mallampati score >1, and a Friedman tongue position of 2, 3, or 4 were excluded. The ESS is a simple and validated questionnaire for assessing subjective daytime sleepiness in the context of sleep disorders. The ESS is a self-administered questionnaire containing 8 items that patients are asked to score on a scale of 0 to 3 (0, no chance of napping; 1, small chance of napping; 2, moderate chance of napping; and 3, strong chance of napping). Scores were totaled, with a range of 0 to 10 defined as normal.16, 17

Operations were performed under general anesthesia. Killian’s incision was preferred. The incision was usually situated on the concave side of the septum. The cartilaginous and bony nasal septum was exposed from both sides by the elevation of mucoperichondrial and mucoperiosteal flaps. Deviated structures were removed with cutting forceps or Ballenger’s knife. Sufficient cartilage and bone were preserved for structural support. Internal nasal splints were applied following transseptal suturing. Only the patients in the study group underwent septoplasty; participants in the control group did not undergo any surgery.

Electrocardiographic records were used to determine Pd and QTcd values in NSD patients and the control group preoperatively and 6 months postoperatively. A standard 12-lead ECG 50-mmV recording was performed following a 10-minute rest in the supine position. All recordings were performed during spontaneous breathing. All measurements were repeated 3 times and taken in the same quiet room. Prior to analysis, a single blinded investigator calculated the mean of 3 consecutive interval measurements. Pd was measured from the first sign of upward departure from the baseline to the point of return to the baseline. The difference between the longest and shortest P-waves in any of the 12 leads was defined as Pd (milliseconds). The QT interval was measured starting from the onset of the QRS complex until the end of the T-wave. QTcd dispersion was measured as the difference between the maximum and minimum QT intervals (milliseconds) with heart rate correction, according to Bazett’s formula.18

Measurements were statistically compared through SPSS 20.0 for Windows (SPSS Inc, Chicago, Illinois). Continuous variables are presented as mean ± SD. Qualitative values were compared via the chi-square test. Significant differences between the NSD and control groups were detected through a 2-tailed t test; significant differences among the control group and the pre- and postoperative NSD groups were detected through analysis of variance. A P value <.05 was taken to indicate statistical significance.

Results

The study group included 53 patients with marked NSD (18 women, 35 men) and a mean age of 38.03 ± 12.52 years (range, 18-54 years). The control group included 53 healthy subjects (20 women, 33 men) with a mean age of 41 ± 15.76 years (range, 20-58 years). The age and sex distributions of the patients did not differ significantly between groups (Table 1). Both Pd and QTcd values were significantly higher in the preoperative NSD group as compared with the control group (Pd, P < .001; QTcd, P < .001). Among NSD patients, preoperative Pd and QTcd values were significantly higher than postoperative values (Pd, P = .013; QTcd, P = .012). Furthermore, postoperative Pd and QTcd values in NSD patients did not differ significantly than values observed in the control group (Pd, P = .31; QTcd, P = .28). Table 2 presents the Pd and QTcd values.

Discussion

UAO is characterized by the partial or complete closure of the upper airway. NSD is a common cause of recurrent and chronic UAO and a possible risk factor for OSA.4, 19-21
Nasal airflow resistance constitutes a significant part of airway resistance; thus, small changes in the nasal patency affect total airway resistance.\textsuperscript{20,22} Mechanical UAO can lead to hypoxia, hypercapnia, and significant changes in intrathoracic pressure. All these factors may affect sympathetic and parasympathetic activation, as well as cardiac autonomic responses.\textsuperscript{23} Although the exact mechanisms underlying the link between UAO and cardiac arrhythmias are not well known, they could be similar to the mechanisms relating OSA to various cardiovascular diseases. Autonomic dysfunction, sympathetic nervous system activation, and hypoxia are believed to be the common pathophysiologic factors involved in arrhythmogenesis.\textsuperscript{24,25} Suppression of cardiac efferent vagal tone and an increase in sympathetic neural outflow may initiate arrhythmias directly by causing abnormal cardiac remodeling of the atrium and ventricle\textsuperscript{26,27} or indirectly by affecting heart rate, blood pressure, and coronary blood flow.\textsuperscript{28,29} Hypoxia and oxygen-derived free radicals can also damage cardiac myocytes and affect myocyte ion exchange, thereby increasing the likelihood of functional deterioration as well as facilitating arrhythmogenesis.\textsuperscript{3,25,30,31}

Several studies have recognized that UAO, specifically OSA, can contribute to cardiovascular morbidity. Although the role of nasal obstruction in the pathogenesis of OSA has been well studied,\textsuperscript{32,33} to our knowledge there has not been any published study evaluating the risk of arrhythmia in NSD patients by noninvasive markers. Nasal obstruction and mouth breathing could contribute to UAO through several mechanisms. First, nasal obstruction causes the mouth to open for the patient to breathe. This leads to a backward rotation of the jaw, displacing the base of tongue posteriorly and lowering the hyoid, which leads to increased pharyngeal collapse. Second, nasal obstruction and mouth breathing cause increased resistance upstream, which leads to increased downstream resistance through the loss of nasal ventilatory reflexes.\textsuperscript{23,24} It is confirmed that upper airway patency activates the nasal mechanosensitive receptors and leads to a direct positive effect on spontaneous ventilation, higher resting breathing frequency, and higher minute ventilation.\textsuperscript{35} Nasal obstruction and mouth breathing reduce the activation of these receptors, leading to (1) deactivation of the nasal ventilatory reflex; (2) inhibition of respiratory rate, minute ventilation, and muscle tone; and (3) an increase of bronchoconstriction, which can trigger respiratory events in susceptible individuals with subclinical OSA or exacerbate apnea episodes.\textsuperscript{20,36} It has been suggested that the treatment of NSD can be beneficial to overcome or prevent OSA.\textsuperscript{37}

Although the effects of chronic UAO disorders—such as adenotonsillar hypertrophy in childhood and OSA on the cardiopulmonary system—have been researched and the mechanisms have been demonstrated, the cardiopulmonary effect of pure NSD has not been investigated adequately. Therefore, it is difficult to explain the exact mechanisms underlying the relationship between NSD and arrhythmogenesis. Fidan and Aksakal noticed that pulmonary arterial pressure is higher in patients with marked NSD and decreases significantly after septoplasty.\textsuperscript{38} Literature has revealed that increased pulmonary artery pressure associated with hypoxia and pulmonary vasoconstriction may result in hypertrophy of the right cardiac ventricle, which can then lead to right cardiac failure.\textsuperscript{39} Derin et al carried out a prospective study to investigate any impact of NSD on cardiac arrhythmias by performing 24-hour rhythm Holter analysis.

### Table 1. Demographic Characteristics.

<table>
<thead>
<tr>
<th>Study Group (n = 53)</th>
<th>Control Group (n = 53)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y Mean ± SD</td>
<td></td>
<td>(.25^a)</td>
</tr>
<tr>
<td>Range</td>
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<td></td>
</tr>
<tr>
<td>Sex, n</td>
<td></td>
<td>(.38^b)</td>
</tr>
<tr>
<td>Women</td>
<td></td>
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<tr>
<td>Men</td>
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</table>

*Two-tailed t test.

\(a\)Chi-square test.

### Table 2. Pd and QTcd Values.

<table>
<thead>
<tr>
<th>Pd</th>
<th>Preoperative Values</th>
<th>Postoperative Values</th>
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<tbody>
<tr>
<td></td>
<td>NSD (n = 53)</td>
<td>Control (n = 53)</td>
</tr>
<tr>
<td>ms</td>
<td>57.40 ± 14.21</td>
<td>34.11 ± 7.12</td>
</tr>
<tr>
<td>Range</td>
<td>31-79</td>
<td>14-51</td>
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<tr>
<td>QTcd</td>
<td></td>
<td></td>
</tr>
<tr>
<td>ms</td>
<td>81.77 ± 16.39</td>
<td>50.25 ± 11.51</td>
</tr>
<tr>
<td>Range</td>
<td>57-112</td>
<td>30-73</td>
</tr>
</tbody>
</table>

*Abbreviations: NSD, nasal septum deviation; Pd, P-wave dispersion; QTcd, heart rate–corrected QT dispersion.

*Analysis between preoperative NSD and control groups.

\(^b\)Analysis between preoperative and postoperative NSD groups.

\(^c\)Analysis between postoperative NSD and control groups.
before and after septoplasty, and they reported that septoplasty decreased ventricular and supraventricular extrasystoles.\textsuperscript{40} In an experimental study, hypoxia was shown to induce extrasystoles, and the improvement of hypoxia has been shown to decrease extrasystole occurrence.\textsuperscript{25} These studies partially reveal and represent the possible effects of nasal obstruction on cardiopulmonary function.

Recently, in a large prospective study, the predictive value of QTcd was assessed in 1839 American Indians followed up for 3.7 ± 0.9 years. QTcd was reported as a significant and independent predictor, with a 34% increase of cardiovascular mortality for each 17-millisecond increase, and the authors emphasized that QTcd >58 milliseconds was associated with a 3.2-fold increased risk of cardiovascular mortality.\textsuperscript{41} Reported values of QT dispersion vary mostly between 30 and 60 milliseconds in normal subjects, with average values around 70 milliseconds also being reported.\textsuperscript{41} In a meta-analysis of 6827 healthy subjects, the average Pd value was reported as 33.46 ± 9.65 milliseconds (range, 7 ± 2.7 to 58.56 ± 16.24 milliseconds).\textsuperscript{42} Also, a dispersion value ≥40 milliseconds was found to be significantly specific and sensitive in patients with atrial fibrillation when compared with healthy controls.\textsuperscript{43} Although there is no validated limit values to assess arrhythmia risk by QTcd and Pd, the values considered by the large population-based studies\textsuperscript{41-43} above could be accepted as limit values. Mechanical UAO due to NSD can lead to inhibition of nasal ventilatory reflex, hypoxia, hypercapnia, autonomic dysfunction, sympathetic nervous system activation, disturbances in coronary blood flow, and generation of oxygen-derived free radicals that could contribute to facilitate arrhythmogenesis. Therefore, we evaluated Pd and QTcd—useful and noninvasive markers for the prediction of arrhythmias—to quantify the risk of atrial and ventricular in patients with NSD.

In this study, we observed substantial differences between NSD patients and healthy controls. Pd and QTcd values were higher in patients with NSD than in the healthy subjects. We detected a significant reduction and normalization in Pd and QTcd values after septoplasty in NSD patients. These results suggest that NSD, a common cause of UAO, facilitates arrhythmogenesis. Furthermore, septoplasty proved to be beneficial in NSD patients by eliminating UAO and reducing arrhythmia risk by withdrawing sympathetic activation and enhancing parasympathetic tone. It is important for physicians to accurately diagnose UAO pathologies, such as NSD, in patients with cardiac arrhythmia or other cardiac diseases. Treatment of NSD may be useful following assessment in these patients. However, it is important to recognize that NSD may predispose individuals to future cardiac problems.

The strength of our study is the assessment of the risk of atrial and ventricular arrhythmias in patients with NSD and the effect of septoplasty on arrhythmia risk by measuring noninvasive markers (Pd and QTcd) for the first time. There are several limitations to our study. First, this study included a relatively small number of patients. Additional larger population-based studies with long-term clinical follow-up data are needed to accurately identify the potential effects of NSD and septoplasty on cardiac arrhythmia risk. However, inclusion of patients with bilateral marked septal deviation limits the interpretation and generalizability to all patients with septal deviation in the present study. The main limitation is the lack of rhinomanometry examinations. This type of information could determine the nasal airway resistance more accurately.

Conclusions

This study is novel, as it assessed specific ECG markers indicative of atrial or ventricular arrhythmic risk. In the 2 outcomes measures—Pd and QTcd—preoperative NSD patients showed significantly higher values than the control group, and these values “normalized” to levels similar to those of the controls at 6 months postoperatively. These results indicate the significant association between nasal obstruction and arrhythmia risk. In conclusion, patients with marked NSD should be encouraged to do septoplasty sooner for prevention of future cardiac problems. Additional larger population-based studies are needed to verify our preliminary results.

Author Contributions

Sinan Uluyol, wrote the article, performed surgeries, revised article, final approval of the version to be published, integrity of any part of the work; Saffet Kiliçaslan, wrote article, designed study, analysis of data, final approval of the version to be published, integrity of any part of the work; Mehmet Haftç Gür, conception and design, drafting the work, final approval of the version to be published, integrity of any part of the work; Nermin Erdas Karakaya, conception and design, revising article, final approval of the version to be published, agreement to be accountable for all aspects; İpek Buber, designed work, analysis of data, drafting the article, final approval of the version to be published, agreement to be accountable for all aspects; Sedef Gulec Ural, analysis of data, revising article, final approval of the version to be published, agreement to be accountable for all aspects.

Disclosures

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References


