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What is the Relationship Between Chronic Sinus Disease and Isolated Nasal Septal Deviation?

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OBJECTIVE: The aim of this retrospective study is to examine the role of isolated nasal septal deviation (NSD) in the pathogenesis of chronic rhinosinusitis (CRS).

STUDY DESIGN AND SETTING: The interaction between isolated NSD and chronic sinus disease were retrospectively evaluated in 1452 patients. Out of 1452 patients, 152 patients were included in the study. Patients with anatomical variants other than NSD were excluded from the study. Patients with NSD were enrolled in the study group and patients without NSD were enrolled in the control group.

RESULTS: There was no statistically significant difference between NSD group and non-NSD group with respect to the CRS.

CONCLUSIONS: The mild to moderate degree of NSD was not a risk factor for chronic sinus disease. Only gross deviation of the nasal septum itself is a risk factor for the development of CRS.

SIGNIFICANCE: Excluding the subjects with ostiomeatal anatomic variations has differentiated this study from the previously reported researches (isolated NSD).

Chronic rhinosinusitis (CRS) is a group of disorders characterized by inflammation of the mucosa of the nose and paranasal sinuses of at least 12 consecutive weeks' duration.[1] Although the exact pathogenesis of CRS remains unclear, most authors agree that it is an inflammatory condition of the nose and paranasal sinuses.[2] Various etiologies have been implicated including bacterial infection, viral infection, sinus outflow obstruction, allergy, Helicobacter pylori, decreased levels of trace element, and functional disorders of leukocytes.[2-5] Nasal septal deviation (NSD) has been implicated in the pathogenesis of CRS by means of sinus outflow obstruction or interference of the mucociliary activity. In this respect several studies have attempted to elucidate the relationship between NSD and CRS. But the results are contradictory.[6,7] Although the role of NSD on the development of CRS (regardless of other anatomical variants) has been examined, the exact role of the NSD (isolated effect), within the context of the structural factors that predispose to CRS, has as yet remained elusive.

The main intention of this study was to evaluate the exact relationship between NSD and chronic sinus disease.

METHODS

This study was conducted from 1996 to 2003 in Suleyman Demirel University by taking all findings from charts of patients' records of in- and outpatient clinics. A total of 1452 adult patients who underwent paranasal sinus CT imaging for evaluation of either rhinologic or orbital complaints were divided into 2 groups; those with NSD were enrolled in the study group and those without NSD were enrolled in the control group. The main basis for selection of patients to study or control group was the presence or absence of NSD. The study group was also divided into 3 subgroups (mild, 0°-10°; moderate, 11°-20°; severe, >21°) regarding the severity of NSD angle. All the clinical and laboratory findings were also recorded from the chart review of patients.

Consecutive coronal CT scans were evaluated regarding NSD, chronic sinus disease, and paranasal anatomical

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Table 1
Characteristics of study and control groups

Groups	Age range (y)	Mean age (y)	SS			% of sinusitis	Gender	
			I	C	B		M	F
Study (N: 152)	18-74	37.61 ± 13.30	27	25	17	69/152 (45.39%)	79	73
Control (N: 57)	18-67	33.52 ± 14.25	7	8	4	19/57 (33.33%)	27	30

SS, The side of involved sinus as number of patients; I, Ipsilateral; C, Contralateral; B, Bilateral; M, Male; F, Female; y, Years.

variations. The patients with previous nasal or sinus surgery, S-shaped nasal septal deviation, congenital facial deformity, systemic immune deficits (AIDS, leukemia, patients receiving immunosuppressive medications), malignant disease involving the head and neck, anatomic ostiomeatal complex variation, allergic rhinitis, or nasal polyp disease were excluded. CT evaluation of the paranasal sinuses was performed in the coronal projection without the administration of intravenous contrast material. We have performed the CT examinations with the patient prone on the scanner bed with the head in hyperextension. Thin-section CT scanning (Philips, Tomoscan AVPS, Netherlands) was obtained with the following parameters: field of view, 160 mm; section thickness, 5 mm; index, 5 mm; 120 mA; 175 kV; small focal spot; mean total acquisition time, 5 minutes. Examinations were evaluated using both bone and soft tissue windows. One radiologist and one ENT specialist evaluated all of the paranasal sinus CTs. The mean value of 3 consecutive coronal CT images at the level of the ostiomeatal complex (OMC) was used for the calculation of the direction and degree of NSD. The superior insertion of the nasal septum at the level of the crista galli, its inferior insertion at the anterior nasal spine, and the apex of the NSD were all identified and clearly marked on the respective CT film. The resultant angle was calculated using a standard protractor with the direction of the NSD noted.

The χ^2 test (with Spearman's correlation) was used to analyze the differences in the frequency of sinus disease between patients with and without NSD.

RESULTS

A total of 1452 adult patients (744 male and 708 female) who underwent paranasal sinus CT imaging for evaluation of either rhinologic or orbital complaints were divided into 2 groups (the criteria of this separation was the presence or absence of NSD); 152 patients with NSD were enrolled in the study group, 79 male and 73 female. Age ranged between 18 and 74 years (mean age was 37.61 ± 13.30 years). Fifty-seven patients without NSD were enrolled in the control group, 27 males and 30 females. Age ranged between 18 and 67 years (mean age was 33.52 ± 14.25 years) (Table 1). The remaining 1243 patients were excluded from the study according to the exclusion criteria mentioned previously. The study group consisted of 152 patients with mean NSD angle of 15.90° ± 5.63° (range 5°-32°). Sixty-nine out of 152 patients (45.39%) has had CRS. The study group was divided into 3 subgroups regarding the severity of NSD angle (Table 2). Subgroup I consisted of 23 patients with mean NSD angle of 7.34° ± 1.69° (range 5°-10°). Eight out of 23 patients (34.78%) have had CRS. Subgroup II consisted of 96 patients with mean NSD angle of 15.36° ± 2.86° (range 11°-20°). Forty-three out of 96 patients (45.36%) have had CRS. Subgroup III consisted of 32 patients with mean NSD angle of 23.71° ± 2.95° (more than 21°). Eighteen out of 32 patients (56.25%) have had CRS. Nineteen out of 57 control subjects (33.33%) have had CRS. The only statistically significant difference was between subgroup III (severe NSD subgroup) and control group ($P = 0.035$). There was no statistically significant difference among other subgroups and control group (Table 3). Max-

Table 2
Degree of NSD and sinus involvement in subgroups I, II, and III of study group

	Range of NSD angle (°)	Mean angle of NSD (°)	No. of patient	Patient with sinusitis	% CRS patient	SS		
						I	C	B
Subgroup I	5-10	7.34 ± 1.69	23	8	34.78	4	2	2
Subgroup II	11-20	15.36 ± 2.86	96	43	45.36	19	15	9
Subgroup III	>21	23.71 ± 2.95	32	18	56.25	11	5	2
Total		15.90 ± 5.63	152	69	45.39	34	22	13

SS, The side of involved sinus as number patients; I, Ipsilateral; C, Contralateral; B, Bilateral.

Table 3
Statistical analysis of control and study groups (subgroups I, II, and III) regarding the presence of sinusitis

	S - C groups	SgI - C	SgII - C	SgIII - C
P values	0.116	0.901	0.165	0.035*

S, study group (patients with NSD); C, Control group; SgI, SgII, SgIII, Subgroups I, II, and III of study group respectively.
*Statistically significant.

illary and anterior ethmoid sinuses (sometimes frontal sinus) were the involved sinuses in all sinusitis patients. There was neither posterior ethmoid nor sphenoid sinus involvement in patients with sinusitis. The rates of CRS ipsilateral to and contralateral to the side of the NSD were not statistically different among CRS groups

DISCUSSION

Paranasal CT is one of the best methods to evaluate the existence and extension of the sinus pathologies, to assess some important anatomical variations and dangerous areas of the paranasal sinuses.[8] Multiple conditions may play a direct or contributory role in the pathogenesis of CRS.[1] It is believed that the most critical factor in the development of sinusitis may be an intrinsic defect in the mucociliary clearance (epithelial metaplasia, ciliary defects, and increased mucus viscosity); anatomic narrowings or tortuous passageways may only compound the problem.[9] Many normal anatomic variants have been implicated in causing CRS by way of their obstructive effect on the OMC and interference with mucociliary clearance patterns.[7] The most important problem that arises when studying the correlation between NSD and CRS is due to the definition and methodologies of previous studies. Up until now, all the studies have investigated the relationship between NSD and CRS without excluding simultaneous sinonasal anatomical variation affecting ostiomeatal complex area. In our study we have excluded the subjects with simultaneous sinonasal anatomical variation such as uncinate process variations, middle turbinate pneumatization and hypertrophy, and ethmoidal variations. These variations are guilty for compromising the rhinosinusitis. The larger the concha bullosa, the more likely is compromise of the normal drainage of the middle meatus.[10] Some other variants of sinonasal anatomy related with obstruction of middle meatus are Haller cells, enlarged ethmoid bulla, hyperplastic uncinate process, uncinate bulla, and paradoxically oriented middle turbinates.[9] Maxillary sinus hypoplasia, which is an infrequently encountered abnormality, is also associated with sinusitis.[11,12] When two mucosal surfaces touch because of swelling or anatomic distortion of the underlying tissues, mucociliary transport is prevented in the area of contact.[10]

A higher incidence of ostiomeatal complex obstruction and increased incidence of CRS have been reported in subjects with NSD. It was suggested that contralateral ostiomeatal complex obstruction could be related to middle-turbinate and lateral nasal wall abnormalities, which appeared with increased frequency on the side opposite the NSD.[13] In most studies, prevalence of NSD looks the same when having been compared among patients with radiological rhinosinusitis and among the general population.[6]

An increased incidence and severity of sinus disease correlated to an increasing angle of septal deviation in the ostiomeatal complex area is reported in some studies. However, anatomic variants related with OMC were connivanced in these studies.[6,7,9,14-17] The most important aspect of our study is the exclusion of patients with important ostiomeatal complex variations leading to CRS.

There are 3 physiopathological hypotheses in the literature trying to explain how NSD could induce rhinosinusitis: 1) mechanical, 2) aerodynamic, and 3) alterations of sinusal ventilation and antral pressures.[6] The severity of septal deviation and their location, shape, and complexity all influence airflow dynamics in the nasal cavity.[13] There are some controversial reports regarding the interaction between NSD and mucociliary transport.[13,18] Passali et al[18] demonstrated that hypertrophy of the inferior turbinate and NSD do not interfere significantly with mucociliary transport that represents a defensive barrier against biological and physical insults in paranasal sinuses and the upper respiratory tract. Ingels et al[19] found no correlation between ciliary beat frequency and nasal patency. On the other hand, the antral pressure variations not only reflect the ostial function but, as well, the total aerodynamics of the upper respiratory tract.[20] The rates of CRS ipsilateral to and contralateral to severe NSD may lead to speculation that severe NSD itself causes CRS on both sides. Ipsilateral CRS may be due to mechanical obstruction, aerodynamics, and antral ventilatory changes. However, contralateral CRS may be due to 2 mechanisms other than mechanical effect. In light of these findings, the relationship between CRS and severe NSD corroborates the mechanical and aerodynamic effects of severe NSD.

CONCLUSION

This study showed that not all NSD, which may be regarded as a contributing factor for sinonasal disease, leads to formation of CRS. Only severe NSD seems to play a contributory role in the pathogenesis of CRS. The larger the septal deviation, the greater the incidence of sinusitis, until in the most severe group, group III, it reached statistical significance. This relationship may be by means of mechanical and aerodynamic mechanisms. That is, mild- to moderate-degree NSD may be associated with sinusitis, but do not play significant role in the pathogenesis of CRS.

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