



Allergic Rhino-sinusitis and Bronchial Asthma Co-existence: Is it A Functional or Anatomical Correlation?

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ABSTRACT

Background: Few studies were conducted to explore the correlation between bronchial asthma and allergic rhino-sinusitis.

Objectives: To clarify the nature of co-morbidity between bronchial asthma and allergic rhino-sinusitis.

Materials and methods: A cross sectional hospital-based study was conducted during the year 2014. A total of 83 subjects were selected and classified into four groups: 23 normal subjects as control in group (1); 20 asthmatic patients (group 2); 20 patients with rhino-sinusitis (group 3); and 20 patients with co-morbid asthma and rhino-sinusitis (group 4). CT scan (coronal sections) of paranasal air sinuses and pulmonary function tests were done for all participants under the study.

Results: Anatomical abnormalities were identified in the maxillary, frontal, and ethmoid sinuses in the form of mucosal thickening, bony wall erosion, complete opacification and polyps in all groups under the study. However, compared to the control group (normal subjects), the mucosal thickening and polyps were more evident in the maxillary sinus in group 2 (asthma only, $p=0.013$), group 3 (rhino-sinusitis only, $p=0.001$) and group 4 (co-morbid asthma plus rhino-sinusitis, $p=0.013$). Pulmonary function tests were found to be significantly impaired in asthma only group, $p=0.026$ for FEV1 and co-morbid group ($p=0.046$ for FEV1). Compared to control group, the mean FEV1 (L) was significantly reduced in patients with maxillary sinus abnormalities among asthmatic and co-existence groups ($p < 0.001$).

Conclusion: Maxillary sinus abnormalities could partially explain asthma and rhino-sinusitis co-existence as it predisposes to allergic reaction and generation of systemic inflammatory mediators that lead to functional impairment in both upper and lower respiratory tracts.

KEYWORDS : asthma, rhino sinusitis, allergic rhinitis.

Introduction:

Paranasal sinuses are groups of air-filled spaces developed as expansions of the nasal cavities eroding the adjacent bone structures¹. Anatomical variations, in association with their inherent conditions, were found to be risk factors for many respiratory tract pathological conditions. Therefore, identifying these variations has recently been critical for clinical practice^{2,3}.

Paranasal sinus anatomy and variations have gained interest with the introduction of functional endoscopic sinus surgery and the knowledge of anatomical variations is most important in the surgical management and specifically in the prevention of complications⁴. The acquisition of an excellent definition of the sinus anatomy for a pre-operative endoscopic evaluation can be done by means of computed tomography that is the gold standard in the study of such structures, for providing accurate information on soft tissues, bone structures and air, thus characterizing a highly sensitive imaging method^{4,5}.

The association between allergic rhino-sinusitis and bronchial asthma was established by many studies 5-8. Many studies including very recent ones were conducted to explain the association between upper and lower airway diseases especially allergic rhino-sinusitis and bronchial asthma. Four theories have been raised by these studies. The first theory by Turgut S, et al (2005) and Kantarci M, et al (2004) has explained the association by the presence of systemic inflammatory response which involve blood circulation and the bone marrow as well as allergic hypersensitivity reaction mediated by high level of circulating IgE^{3,5}. The second one by Seybt MW, et al (2007) hypothesized the one airway one disease theory (the concept of united airway). Seybt,s study found that 80% of asthmatics suffer from sinus diseases and 40% of patients with sinus diseases suffer from asthma⁶. The third theory described by Stelmach R, et al (2010) considered that

the paranasal abnormalities are causes of co-existence between asthma and rhino-sinusitis⁷. The fourth theory suggested by Bachert C, et al (2010) argued that the superior and inferior airway diseases were incorporated in the triad syndrome of asthma, sinusitis and aspirin intolerance (ASA), in which patients on long term aspirin therapy initially develop rhinitis which afterwards complicated by nasal polyps and severe asthma⁸.

The present study aimed at demonstrating the main anatomical variations that may be detected in paranasal air sinuses in Sudanese resident in Khartoum state using Computed tomography (CT) scan; and to find out the relation between anatomical variations of paranasal sinuses and co-existence between upper and lower respiratory tract diseases mainly rhino-sinusitis and asthma.

Materials and methods:

A cross sectional hospital-based study was conducted at Alamal National Hospital during the year 2014. A total of 83 subjects were included and classified into four groups: 23 subjects as control in group (1); 20 asthmatic patients (group 2); 20 patients with rhino-sinusitis (group 3); and 20 patients with co-morbid asthma and rhino-sinusitis (group 4). Asthmatic and rhino-sinusitis patients were randomly selected from chest and ENT clinics and diagnosis of the patients was confirmed by chest physician and ENT consultant respectively. A questionnaire including personal data, medical history, drugs history, predisposing factors of airway diseases, and history of allergy or nasal or paranasal air sinuses surgery was filled by all subjects under the study.

After explanation of the steps of the procedures and the safety measures for the candidates and having a written consent for participation in the study, all subjects under the study (patients and control

group) had coronal sections CT scan using Toshiba Aquillion 64 slices at Almal National Hospital. Coronal sections CT scan were done by a certified expert technologist and result of slices were diagnosed and reported by a consultant radiologist. Pulmonary function tests (FVC, FEV₁, PEFr and FEV₁%) using Microplus spirometer (ME, 2Az England) were done for all asthmatic patients (asthmatic alone and patients with co-morbid asthma and rhino-sinusitis). Bronchial reversibility test was also performed for asthmatic patients. After explaining the procedure, FEV₁ and PEFr were recorded (before bronchodilator inhalation). The second record was taken 15 minutes after having inhaled a short term bronchodilator (B2 agonist). An increment of 12% in FEV₁ and 20% in PEFr was considered positive (significant) reversibility test.

Results:

A total number of 83 subjects were included and divided into 4 groups as shown in table 1.

Table 1: Study groups classification:

Study groups		N	Percent
Group 1	Normal subjects	23	27.7%
Group 2	Mild asthma	4	4.8%
	Moderate asthma	15	18.1%
	Severe asthma	1	1.2%
Group 3	Rhino sinusitis	20	24.1%
Group 4	Rhino sinusitis+ mild asthma	13	15.7%
	Rhino sinusitis+ moderate asthma	6	7.2%
	Rhino sinusitis+ severe asthma	1	1.2%
Total		83	100.0%

Table 2: Mean values of FEV1 in control, asthma and co-morbid groups:

Study group	FEV ₁ analysis reports		
	Number of subject	Mean	± SD
Control group	23	4.0413	0.12
Asthmatic group	20	2.2885	0.32
Co-existent group	20	2.1155	0.13

Compared to control group, the mean FEV₁ with maxillary sinus abnormalities among 0.001).

(L) was significantly reduced in patients asthmatic and co-existence groups (p <

Table 3: Percentages of nasal septum abnormalities:

Abnormalities	Control N=23	Asthmatic (A) N=20	Rhino-sinusitis (RS) N=20	Co-existent (A+ RS) N=20
Deviation (D.)	75%	75%	70%	70%
D. to right anterior	35%	15%	25%	40%
D. to right posterior	0%	0%	0%	5%
D. to left anterior	30%	25%	45%	50%
D. to left posterior	0%	10%	0%	0%
Mucosal thickening	20%	0%	10%	15%
P values by Chi-square		0.000	0.628	0.706
Risk by Odds ratio		1.5	2.8	4.5

Table 4 shows that mucosal thickening and polyps were more frequent abnormalities identified in the maxillary sinus among all groups.

Table 4: Percentages of maxillary sinus abnormalities:

Abnormalities	Control N=23	Asthmatic (A) N=20	Rhino-sinusitis (RS) N=20	Co-existent (A+ RS) N=20
Mucosal thickening	8.5%	25%	35%	55%
Polyps	17%	30%	30%	35%
Bony wall erosion	0%	5%	0%	5%
Complete opacification	0%	0%	15%	15%
P values by Chi-square		0.013	0.001	0.013
Risk by Odds ratio		3	3.3	4.7

As shown in table 5, frontal sinus abnormalities were more obvious in rhino-sinusitis group compared with other groups under the study.

Table 5: Frontal sinus abnormalities:

Abnormalities	Control N=23	Asthmatic (A) N=20	Rhino-sinusitis (RS) N=20	Co-existent (A+ RS) N=20
Mucosal thickening	0%	5%	45%	55%
Polyps	0%	0%	0%	5%
Bony wall erosion	0%	0%	0%	0%
Complete opacification	0%	0%	10%	0%
P values by Chi-square		0.119	0.005	0.465
Risk by Odds ratio		1.2	6.5	7.42

Table 6 shows that mucosal thickening of the ethmoid sinus was most frequent abnormality and the rhino-sinusitis group was most affected than other groups under the study.

Table 6: Ethmoid sinus abnormalities:

Abnormalities	Control N=23	Asthmatic (A) N=20	Rhino-sinusitis (RS) N=20	Co-existent (A+ RS) N=20
Mucosal thickening	13%	10%	45%	55%
Polyps	0%	0%	0%	0%
Bony wall erosion	0%	0%	0%	0%
Complete opacification	0%	0%	10%	0%
P values by Chi-square		0.210	0.048	0.066
Risk by Odds ratio		.975	3.5	4.1

Figure 1 Shows that mucosal thickening of the turbinates was identified in all groups under the study with insignificant inter-group difference (asthma only, p= 0.000), rhino-sinusitis only, p=0.936 and co-morbid asthma plus rhino-sinusitis, p= 0.432).

Figure 1: Mucosal thickening of the turbinates.

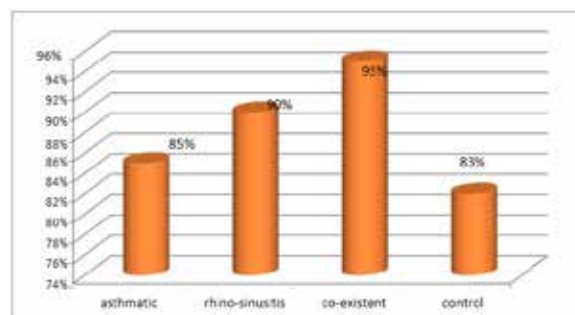
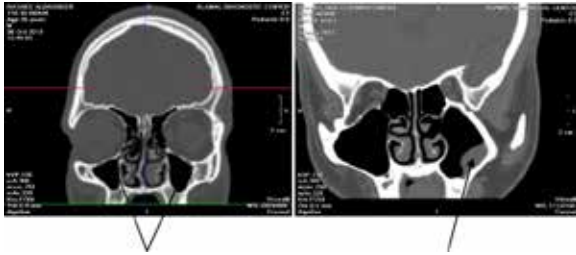


Figure 2: Left sided maxillary sinus polyps compared with normal.



Normal maxillary sinuses

Left sided maxillary sinus polyp

Figure 3: Right sided complete opacification of the maxillary sinus compared with normal.



Normal maxillary sinuses

Right sided maxillary sinus opacification

Discussion:

In the present study, nasal septum abnormalities (mucosal thickening and deviation) were nearly equal in all groups. More than 80% of turbinates showed mucosal thickening in one or both sides in all groups under the study which exclude or insignify their roles as a causative factors concerning co-existence between bronchial asthma and rhino-sinusitis. Probably these abnormalities may be due to allergic environmental agents in Sudan. Most of the frontal and ethmoid sinuses in all groups were normal which could be explained by their gravity-facilitated drainage. Mucosal thickening was shown in about half of patients with rhino-sinusitis alone and those with co-morbid rhino-sinusitis and asthma which could result from the associated inflammatory process in the mucosa of the sinuses. In our study, polyps were considered a weak cause of co-existence between bronchial asthma and rhino-sinusitis. Our finding were not consistent with the study done by Bachert C, et al (2010) in which the presence of nasal polyps was considered as one of ASA triad syndrome that was regarded as a cause of co-morbidity between upper and lower airway diseases (because the polyps was identified in normal control group in addition to other patients groups). Maxillary sinus abnormalities apart from polyps were considered as a cause of co-existence of upper and lower airway diseases because it was present in 40% (total number= 20) of asthmatic, 70% (total number= 20) of patients with rhino-sinusitis and in 45% (total number= 20) of patients with co-existent diseases while seen in low percent 8.5% (total number= 23) of the control group. These findings agree with a study done by Stelmach R, et al (2010) in which 91 patients were included and underwent paranasal radiograph and 8 coronal sections CT scan. The authors reported 40.5% and 56.7% abnormal maxillary sinus on X-ray and CT scan respectively. Maxillary sinus abnormalities (mucosal thickening, polyps, bony wall erosion and complete opacification) which were more evident in group 2 (asthmatic patients), group 3 (rhino-sinusitis patients) and group 4 (patients with co-morbid asthma and rhino-sinusitis) could be explained by difficult anti-gravity drainage that may lead to easy accumulation of mucosal secretions and foreign bodies and enhancement of inflammatory reactions responsible for development of the mucosal thickening, polyps, bony wall erosion and complete opacification. These in turn give rise to development of upper respiratory tract allergy in form of rhino-sinusitis or lower respiratory tract diseases in the form of asthma.

Conclusion and Recommendations:

Maxillary sinus abnormalities could partially explain asthma and rhino-sinusitis co-existence as it predisposes to allergic reaction and

generation of systemic inflammatory mediators that lead to functional impairment in both upper and lower respiratory tracts. Early diagnosis and management of allergic rhino-sinusitis particularly in children could help prevention of the development of bronchial asthma in adulthood. Patients with co-morbid asthma and rhino-sinusitis are strongly advised to undergo paranasal air sinuses CT scans for detection of air sinuses abnormalities. Management of such abnormalities will yield a better control of both bronchial asthma and rhino-sinusitis.

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