THE ASSOCIATION BETWEEN ASTHMA AND NUTRITIONAL PATTERN

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ABSTRACT

Asthma and obesity remain two of the fastest growing and most pervasive public health problems in developing countries. Obesity appear to increase the risk of asthma.both disorder may share common genetic ,enviromental causes. We studied the asthmatic patients and calculate BMI of patient and study the relation between BMI and eosinophilia,pulmonary function tests varities. Eosinophilia is present in 31 cases (72.09%) but it absent in 12 cases (27.9%). p-value =0.290 whish is insignificant. Restrictive response of the patients which are 21 patients the number of mild restrictive pattern 5 patients (23.8%), number of moderate restrictive are 13 patients (61.9%), number of severe restrictive are 3 (14.3%) p-value = 0.146 which insignificant. Ostructive response are present in (11)patient 6 of them are .

mild obstructive (54.5%) and 4 patient are moderate obstructive (36.4%) and 1 patient is severe obstructive in (9.1%) p- value is 0.420 which is insiginificant. Asthma and obesity, control of one of both give better chance to the other, for example weight reduction improve the severity of asthma

Key Words: Asthma, obesity

INTRODUCTION

Asthma is defined by episodic airflow obstruction ,increase airway responsiveness and airway inflammation characterized by infiltration with eosinophils and T lymphcytes particularly in CD4 T. lymphocyte that express

T helper cell type 2 cytokines such as (IL-4), (IL-5), (IL-13) *(Weiss and Shore, 2004).*

Asthma and obesity are both chronic conditions affecting millions worldwide. over the last 20 years there has been a rapid increase in the prevalance of both of these conditions (Tantisira and Weiss, 2001).

The histopathologic appearance of the airways includes denudation of the airway epithelium , thickening of the basement membrane, mucous production and smooth muscle hypertrophy (Weiss and Shore, 2004).

Traditionally, adult overweight has been defined as a body mass index (BMI) of 25-29,9 Kg/m2 .Adult obesity is represented by a body mass index of ≥ 30Kg/m2 . The rise in the obesity has been especially notable in women where the prevelance has increased from 15.1% to 24.9% (Tantisira and Weiss, 2001) .

There has also been a disperate increase in the prevalence in asthma in women with an increase of obesity in women with an increase of 80% in women between 1982 and 1992 but by only 29% in women *(Tantisira and Weiss, 2001).*

Obesity per se is associated with systemic inflammation including elevation in blood leucocytes with increase in serum C- reactive protein(CRP) and pro-inflammatory cytokines (TNF-α) and IL-6 (Rajala and Scherer, 2003).

Given the parallel increases in asthma and obesity it is not surprising that the prevalence and incidence of asthma and its related symptoms and phenotypes have been increasingly associated with body mass index and obesity *(*Tantisira and Weiss, 2001).

Additionally, adipocytes are known to produce angiotensin-II and leptin has direct sympathetic effects on the renal outflow tract, both of which may lead to the sodium retention seen in obesity. In asthma, excess sodium has been associated with increased airways reactivity in a number of studies, although other studies did not note this finding. Following sodium restriction in three double blind clinical trials, improvements were noted in airways responsiveness, FEV_1, and asthma symptoms (Tantisira and Weiss, 2001).

So, while the association between dietary polyunsaturated fats and asthma in children is interestingly, the overall evidence argues against a causal relationship (Woods *et al.*, 2004).

We have not found any evidence to suggest that n-3 fatty acids are associated with a reduced risk of asthma or atopy among young adults. Indeed, our results suggest that is the n-6 polyunsaturted fat DHGLA (dihomo-γ-linolenic acid) that has the strongest association with asthma. Given that was a cross-sectional study, we were unable to establish a cause and effect relationship for the fatty acids / asthma associations found. Nevertheless, these results raise intriguing new possibilities about the role of dietary fats in asthma and warrant further research*(Woods et al., 2004)*

AIM OF THE STUDY

1) To investigate the association between season and asthma and its possible mechanisms.
2) To determine the modifying effects of diet, sex and allergy history on the association of body mass index and asthma prevalence.
3) To calculate the prevalence of asthma according to body mass index, age and history of allergy in men and women separetly.

PATIENTS AND METHODS

Type of the study: Cross sectional study.

Study site: The study will take place in the Imbaba National Institute for Asthma and chest diseases.

The investigator will contact the asthma patients at the asthma clinic.

Selection criteria:

1) Clear history of asthmatic attacks.
2) Reversible pulmonary airway obstruction of 15%after 20 min from the use of a bronchodilator
3) Target population of the survey is adult between 20 – 60 years of age which classified into two groups asthmatic patients and controls. With different nutritional pattern

exclusion criteria

1- smokers.

2 -X-Rays shadows.

3 - Past history of any kind of chest diseases that may affect the lung perminantly like old TB, lung fibrosis, bronchiectasis, chronic lung

abscess, to exclud anything that may cause a wheeze on oscultation and can be confused with the asthma wheeze .

Study design: The population groups will be subjected to the following:

History taking

Personal history involving:

1. nutritional history

2. Residence: certain places and condtions affect the subjects of the study

3-*Occupation*: like persons contact with sweet and drinks.

4-*Smoking*: smoking history no. of cigarette smoked per no of years affect on the asthma, nutritional pattern and apetite

5- *History of illness*: frequency of the attacks, weather it happen monthly, or weekly or daily even more than once daily and the no.of night attacks . no, of hospital admission per year

6 - *Medication history* :Either the use of inhaled steroids alone or with the long acting beta agonist.some medication may increase the apetite.

7- *History of other kinds of allergy* in skin, eye, nasal sinuses

8- *Family history* for asthma or any kind of allergy.

Clinical examination :

including general and special emphasis on chest examination. Patient should be examined during and between attacks to exclude any emphysema

Chest x ray :

All subjects will be filmed in the erect position with postro-anterior view projection. X-ray are done to exclude any other chest problem but asthma .

Blood picture: with special emphasis on the eosinophilic count.

Pulmonary function :

Zhan body box pulmonary function instrument will be used to do the following :

1- Vital capacity .

2-Forced vital capacity

3- FEV1/FVC: detect the difference between the restrictive and the obstructive pattern of the lung function

4- Forced Expiratory Volume in the first second detect obstruction of large airways

5-Forced expiratory flow 25-75: to detect obstruction in small airways

6-flow volume loop for every case

7-FEV1 reversibility pre and 20 min following 2 puffs of inhaled salbutamol improvement of more than 15% is considered a reversible obstruction .

Reversibility in pulmonary function is diagnostic to asthma condtion

Serum IgE : This is an IgE enzyme immunoassay.

Patients with atopic allergic diseases such as atopic asthma , atopic dermatitis , and hay fever have been shown to exhibit increased total immunoglobulin E levels in blood IgE quantitative test is based on solid phase enzyme-linked immunosorbent assay.

The assay system utilizes one mono clonal anti-IgE antibody.

Blood sample will be collected from the study subjects is added to the IgE antibody coated microtiter wells and incubated with zero buffer at room temperature for 30 minutes.

If humen IgE is present in the specimen , it will combine with the antibody on the well .

Skin test: to exclude type of food causing atopy or asthma like fish and egg protein.

Blood picture: with special emphasis on the eosinophilic count

weight and height measurement: by BMI (Body Mass Index), skin fold test, triceps test

Table(1) : Demographic distribution among BMI groups

BMI groups	underweight		Normal		Over weight		Obesity - I		Obesity - II		Obesity - III		Total		P- Value x^2
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	
Sex															
Male	1	50	4	57.1	4	57.1	2	14.3	1	12.5	0	0	12	27.9	P. Value = 0.60 x^2=10.603
Female	1	50	2	42.9	3	42.9	12	85.7	7	87.8	5	100	31	72.1	
Age	2	24	7	17.64	7	30	14	17.75	8	27	5	25	43		P. Value = 0.226 x^2=6.931

Table(2) : Hypersenstivity among BMI groups

BMI groups	underweight		Normal		Over weight		Obesity - I		Obesity - II		Obesity - III		Total		P- Value X2
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	
Seasonal Variation Negative	2	100	3	42.9	2	28.6	5	35.7	2	25	2	40	16	37.2	P. Value = 0.516 X^2=4.235
Positive	0	0	4	57.1	5	71.4	9	64.3	6	75	3	60	27	62.8	
Eosinophilia Negative	1	50	7	87.8	6	100	9	75	5	45.4	3	75	31	72.09	P. Value = 0.290 X^2=8.849
Positive	1	50	1	12.5	0	0	3	25	6	54.5	1	25	12	27.9	

Table(3): Pulmonary Functions Test results among BMI groups

BMI groups	underweight		Normal		Over weight		Obesity - I		Obesity - II		Obesity - III		Total		P- Value X2
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	
Obstructive Mild	1	100	0	0	1	50	1	50	1	33.3	2	100	6	54.5	P. Value = 0.420 X^2=10.236
Moderate	0	0	1	100	0	0	1	50	2	66.7	0	0	4	36.4	
Severe	0	0	0	0	1	50	0	0	0	0	0	0	1	9.1	
Restrictive															
Mild	1	100	0	0	0	0	3	50	1	25	0	0	5	23.8	P. Value = 0.146 X^2=14.646
Moderate	0	0	5	100	2	50	3	50	2	50	1	100	13	61.9	
Severe	0	0	0	0	2	50	0	0	1	25	0	0	3	14.3	

RESULTS

The results of this study are shown in tables(1-3)

Table. (1) show that there was insignificant difference as regard age in studied cases

43patient : male count 12 (27.9 %) female count 31 (72.1 %)

female male patients are divided in 6 groups underweight , overweight , obesity I,II,III normal.

In Age the mean rank is underweight = 24

Normal = 17.64

Over weight = 30

Obesity I = 17.75

Obesity Π = 27

Obesity III = 20

p.value of the Age =0.226 which insignificant , but be significant value due to small no of groups

Table (2) show seasonal variation in different BMI group

43 patient :-ve variation 16 (37.2%)

+ve variation 27 (62.8%)

p- value = 0.516 which is insignificant

And eosinophilia is present or not in different body groups:in 31 cases(72.09%) No eosinophilia but is positive in 12 cases (27.9%).

p-value =0.290 whish is insignificant .

Table.(3) show restrictive response of the patients which are 21 patients the number of mild restrictive pattern 5 patients (23.8%), number of moderate restrictive are 13 patients (61.9%) , number of severe restrictive are 3 (14.3%)

p-value = 0.146 which insignificant

And obstructive response are present in (11)patient 6 of them are mild obstructive (54.5%) and 4 patient are moderate obstructive (36.4%) and 1 patient is severe obstructive in (9.1%)

p- value is 0.420 which is insiginificant

DISCUSSION

Many hypotheses have been proposed to explain the association between asthma ad obesity. One possibility is that obesity and asthma share common risk factors *(Chen et al., 2002).*

An increase in sedentary lifestyle may be responsible for the increase in obesity, whereas the coinciding increase in time spent indoors may be contributing to rise in asthma prevalence via increased exposure to household allergens *(Platts-Mills et al., 2000).*

A sedentary lifestyle may also contribute to asthma by eliminating the apparent protective effect of bronchial muscle stretching associated with sleep breathing during exercise *(Platts-Mills et al., 2000).*

Dietary factors could also confound the association between asthma and obesity *(Fogarty et al., 2000),* or it may be that the two conditions have a common genetic determinant *(Tantisira and Weiss, 2001).*

A second possibility is that obesity cause asthma. In support of this, Stenius-Aarniala et al. showed weight reduction in obese asthmatic patients improved lung function and alleviated symptoms *(Stenius-Aarniala et al., 2000).*

In addition, Castro-Rodriguez et al. found in a large single city (Tucson) with cohort that girls who became overweight between the ages of 6 and 11 years were at an increased risk of developing new asthma symptoms at ages 11 and 13 years compared with girls who did not became overweight *(Castro-Rodriguez et al., 2001).*

It may be that obesity affects asthma risk by mechanical effects such as airways latching, by cytokine modulation via adipose tissue, or by the effects of sex hormones on airway reactivity (Tantisira and Weiss, 2001).

A third possibility is that asthma causes obesity. However, there is very little evidence to support this, particularly in light of recent prospective studies *(*Chen *et al.,* 2002).

The consistency of the relationship, the temporal association the dose response curve, and the association with intermediate phenotypes has caused at least one author that there could be a causal relationship between obesity and the onset of asthma (Shaheen, 1999).

In order to implicate a causal hypothesis, however, one or more plausible biological mechanisms must be established. Although few studies have specifically addressed this question, specific mechanisms relevant to this association can be easily elicited. Obesity may directly affect the asthma phenotype by direct mechanical effects, by enhancing the immune response, through related genetic mechanisms, and by sex hormones. Alternatively, obesity may be closely linked to other environmental factors such as physical activity, diet, and birth weight. These environmental influences, in combination with genetic susceptibility, may then lead to enhanced susceptibility to asthma (Tantisira and Weiss, 2001).

Mechanisms relating obesity to asthma include mechanical factors as Alterations in tidal stretch leading to latch,gastro-oesophageal reflux. and Immune modification (TNF-α,IL-1β,IL-6,Leptin) and genetic effects:

Common candidate genes (TNF-α, β2 adrenergic receptor),Candidate regions (5q, 6P, 11q, 12q),Obesity candidate genes related physiologically to asthma,Sex.,airway size differences,Inflammatory mediators enhanced in women.,Oestrogen,gene X environment interactions. (Tantisira and Weiss, 2001).

Obesity has been associated with increases in the incidence and prevalence of asthma in a number of epidemiological studies of adults and children. (Tantisira and Weiss, 2001).

Weight loss in obese asthmatic subjects results in an improvement in overall pulmonary function and asthma symptoms, as well as decreases in asthma medication usage. (Tantisira and Weiss, 2001).

Obesity may directly affect the asthma phenotype by mechanical effects including airways latching, by cytokine modulation via adipose tissue, through common genes or genetic regions or by sex specific effects including the hormone oestrogen. (Tantisira and Weiss, 2001).

Obesity may also be related to asthma by genetic interactions with environmental exposures, including physical activity and diet.

The Barker hypothesis may underscore the developmental relationship of obesity with asthma (Tantisira and Weiss, 2001).

The association between BMI and asthma and wheeze persisted on controlling for potential confounding factors. However, it might be explained by factors which were not measured. Firstly, a low level of physical activity is clearly associated with higher BMI and has also been proposed as a risk factor for asthma (Shaheen *et al.,* 1999).

Platts-Mills and Colleagues have suggested that reductions in deep breathing associated with a sedentary lifestyle may, by reducing the extent to which bronchial muscle is stretched, lead to airway narrowing (Platts-Mills *et al.*, 1997).

Secondly, the association between BMI and asthma and wheeze might be explained by dietary factors, for example, a low intake of anti-oxidants, which has been proposed as a risk factor for asthma, may be more common in obese than in non-obese subjects (Shaheen *et al.,* 1999).

The association between adult BMI and asthma was cross-sectional. One possible interpretation is that a higher BMI is a consequence of asthma; weight gain could occur as a side effect of oral corticosteroid therapy or because asthmatic individuals avoid vigorous physical activity in order to prevent exercise induced bronchospasm. However, only a very small proportion of adults with asthma take regular oral steroids and, in a study of young adults in south London, we have found that the frequency of participation in regular vigorous sporting activity was similar in those with and without asthma (Shaheen *et al.*, 1999).

The graded association between BMI and asthma in women is consistent with a causal effect. We propose that fatness may increase the severity of asthma in those with established disease and may increase prevalence by contributing to symptoms in individuals who would otherwise have subclinical disease. If it is concurrent fatness which increase risk of asthma, then it is perhaps not surprising that BMI at 10 year was not associated with adult asthma, since the majority of obese adults would not have been obese as

children. It is possible that there is a direct mechanism linking fatness to asthma in adults? The stronger findings in women may be relevant (Shaheen *et al.*, 1999).

However, it should be stated that the assessment of the temporal sequence between obesity and asthma is complex, as asthma can be associated to respiratory symptoms other than shortness of breath with wheezing, and it is very difficult to distinguish with any certainty incident from recrudescent asthma cases in an adult population (Guerra *et al.*, 2002).

Several mechanisms have been proposed for a potential causal relationship between obesity and asthma. Obesity can directly affect the airway caliber through the chest wall restriction. Narrowing of airways and reduction of lung volumes have been associated with bronchial hyperreactivity. Obesity also predisposes to gastro-esophageal reflux disease, a known risk factor for asthma (Guerra *et al.*, 2002).

According to these potential mechanisms, however, the effect of obesity on asthma would be expected to be similar in both sexes, whereas the association between obesity and asthma was significant only among

women. The effect modification by gender suggests that sex hormones could be involved in the causal pathway. Estrogen and progesterone levels are affected by obesity and are related to asthma as well *(Guerra et al., 2002).*

Obesity is associated with a large number of changes in physiology that may mediate the relation of obesity with asthma (Tantisira and Weiss, 2001).

Obese persons show systemic inflammation that appear to play a role in the etiology of nonatopic conditions, including cardiovascular disease,

diabetes, and potentially asthma. Adipose tissue is a source of pro-inflammatory cytokines and chemokines such as IL-6, leptin, IL-18, and TNF-α. An increase in circulating levels or local concentrations of pro-inflammatory cytokines has the potential to enhance pulmonary inflammation, which a key component of asthma pathophysiology (Gilliland *et al.,* 2003).

Because obesity is not clearly associated with allergy, obesity may enhance noneosinophilic inflammatory pathways that increase the risk of nonatopic asthma. The effects of obesity may also be mediated by changes in airway function, since obesity and weight change have been prospectively associated with increased bronchial hyperresponsiveness in asthmatic children as well as in non-asthmatic children. The combined effect of increased bronchial hyperresponsiveness and the pro-inflammatory milieu in obese subjects may set the stage for the onset of asthma (Litonjua *et al.*, 2002).

CONCLUSIONS

Clearly, both asthma and obesity are common conditions and both are major public health problems. Moreover, obesity appears to increase the risk of asthma Both disorders may share common genetic and environmental causes. There are mechanical, developmental, hormonal, signal transduction, and immunologic reasons for their effects. The NHLBI workshop developed a total of complex recommendations to further research in this area:

1- More information, including better phenotyping and more longitudinal cohort studies, is required to determine what is the attributable risk for developing asthma given obesity and in sorting out cause-effect relationships between asthma and obesity.

2- It is clear that the impact of obesity in asthma is more pronounced in females than in males. Understanding this sex difference may help elucidate the mechanistic basis for the relationship between these two syndromes.

3- Leptin is increased in obesity. Thus, it is important to understand the mechanistic basis for effects of leptin on the immune system, on lung growth, and on the SNS and to determine how these effects influence the development of asthma.

4- Besides leptin, there are other hormones produced from fat cells, including resistin, adiponectin, and TNF-α, as well as other hormones that are affected by obesity such as the renin-angiotensin system. It may be important to determine what effect, if any, these hormones have on the

lung or on inflammatory/immune responses and to determine if these are relevant to asthma.

5- Evidence suggests that obesity is an inflammatory state.
6- Little is known about how intrauterine and early life relate to development of obesity and asthma.
7- Genome-wide scans have identified regions linked to asthma and to obesity, some of which overlap.
8- Obesity provides an important load to the respiratory system and alters lung volumes and the pattern of breathing, both of which can affect airway smooth muscle shortening.
9- There appear to be important interactions not only between asthma and obesity but also between asthma and sleep and obesity and sleep.

RECOMMENDATION

obesity is multisystem affection one of those is the respiratory system,when control the nutrition pattern you control body weight and control the asthma attacks.

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دراسة العلاقة بين الربو الشعبي والنمط الغذائى

[٧]

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المستخلص

تعتبر حساسية الشعب الهوائية والربو عبارة عن التهاب مناعى للشعب الهوائية. وقد أظهرت الدراسات المختلفة ان الملوثات الهوائية تعزز حساسية الشعب الهوائية. الربو الشعبي من الأمراض المزمنة التي أصابت في الفترة الأخيرة العديد من ملايين البشر. فعلى مدار العشرين سنة الأخيرة لوحظ زيادة سريعة في معدل انتشار الربو الشعبي. من المتعارف عليه أن الزيادة في الوزن يتم تعريفها علي أنها الشخص الذي يكون معدل كتلة الجسم بالنسبة له (ما بين ٢٥_٥و٢٩ كجم/م^2). أما السمنة فتكون معدل كتلة الجسم أكثر من ٣٠ كجم/م^2. وقد لوحظ أن الزيادة في انتشار السمنة يكون في السيدات بنسبة ما بين (١و١٥%_ ٩و٢٤%). زياده الوزن علي تعريفها المعتاد تكون مصحوبة بالتهاب منتشر في جميع أجزاء الجسم مصحوباً بارتفاع في عدد الخلايا من كرات الدم البيضاء مع زيادة مصحوبة في نسبة معامل البروتين _س، العديد من السيتوكين مثل انترلوكين رقم ٦ و(TNF_α).

الهدف من الدراسة

١) بحث مضاعف لتحديد كيفية العلاقة بين الربو الشعبي والنمط الغذائي والتفسيرات المقترحة لتلك العلاقة

٢) تحديد مدى استفادة مرضى الربو الشعبي من نقص الوزن

٣) تقدير انتشار الربو الشعبي طبقاً للعمر والتاريخ المرضي للحساسية ومعدل كتلة الجسم في الرجال و النساء كلٌ على حدة.

٤) تحديد تأثير نوع الغذاء و التاريخ المرضي للحساسية والنوع على العلاقة بين بين الربو الشعبي والنمط الغذائي

نوع الدراسة: دراسة مقطعية

طريقة البحث:

موقع الدراسة: الدراسة ستجرى في المركز القومى للحساسية والأمراض الصدرية بإمبابة. المحقق سوف يقوم بالاتصال بمرضى الحساسية في عيادة الربو بالمركز.

تصميم الدراسة: الأشخاص محل الدراسة ما بين سن ٢٠عام حتى ٦٠عام يتم تقسيمهم إلى مجموعتين: مجموعة مرضى الربو الشعبي ومرضى كمرجعية للدراسة وذلك من مختلف الانماط الغذائية

يتم دراسة الأشخاص السابقين كمحل للدراسة من حيث :

أ- أشعة سينية على الصدر

ب- تاريخ مرضي كامل

ج-الفحص السريري العام

د- حساب الوزن عن طريق معدل كتلة الجسم.

هـ-وظائف الرئة:

و- صوره دم كاملة:

ز-اختبارات الحساسية: نسبةIgE و اختبار الجلد

ملخص النتائج: نتائج هذه الدراسة تظهران ٤٣ مريض: عدد الذكور ١٢ (٢٧,٩٪) عدد الإناث ٣١ (٧٢,١٪)

وينقسم المرضى الذكور والمرضى الإناث في ٦ مجموعات من نقص الوزن، زيادة الوزن، السمنة I ، السمنة II ، السمنة III، والوزن الطبيعي.

ويظهر التباين الموسمي في المجموعات المختلفة كالتالى:

لم يحدث اختلاف بنسبة (٣٧,٢٪) ونسبة (٦٢,٨) حدث تباين موسمى قيمة P-= 0.516 غير ذات أهمية

وفرط الحمضيات: في ٣١ حالة بنسبة (٧٢,٠٩٪) ولا يوجد فرط للحمضيات في ١٢ حالة بنسبة (٢٧,٩٪). قيمة P-= ٠,٢٩٠ غير ذات أهمية.

وتظهر استجابة وظائف التنفس فى ٢١ مريض كالتالى:

نمط تقييدا بنسبة (٢٣,٨٪) خفيف، وعدد من المعتدلين تقييدا بنسبة (٦١,٩٪)، وعدد من المرضى شديدة التقييد بنسبة (١٤,٣٪) قيمة ٠,١٤٦ P-= غير ذات أهمية.

والانسداد الرئوى موجودة في (١١) مريض كالتالى:

انسداد خفيف (٥٤,٥٪) وانسداد معتدل (٣٦,٤٪) وانسداد شديد (٩,١٪) قيمة P-هو ٠,٤٢٠ وهي غير ذات أهمية.