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Bronchial asthma: a risk factor for hypertension?

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Summary

Several attempts have been made to improve primary prevention of essential hypertension and many of these have been directed at avoiding the well known risk factors. Both asthma and hypertension are spastic disorders of smooth muscle, also asthmatics and hypertensives have been found to be salt sensitive. There is a suspicion that the similarities between these two diseases may predispose the individuals with one disease to the other, as pulmonary hypertension has been described during exercise- induced bronchoconstriction. We therefore, studied the blood pressure pattern during and after acute severe asthma (ASA) along with the frequency of hypertension in stable asthmatic patients. Two groups of patients were studied. Group 1 consisted of 12 patients with ASA (2 males, 10 females) with a mean age of 30 ± 9,9.years. The mean blood pressure during attack of ASA (147±16.9/ 100±8.2 mmHg) was higher than the mean BP (132±8.3/82±7 mmHg) 2 weeks after discharge from hospital without treatment in all patients ($P < 0.05$). Group 2 included 134 asthmatic subjects in stable state (54 males, 80 females) with a mean age of 45 ± 15 years and a range of 15-90 years. The overall frequency of hypertension was 37% with a proportion of 39% in males and 35% in females. Hypertension was defined as systolic blood pressure of ≥ 140mmHg and or diastolic blood pressure of ≥ 90mmHg. There was no difference between the frequency of attack of ASA in hypertensives (5.7 ± 5.6 per year) and nonhypertensives (5.5 ± 3.8 per year), $P < 0.05$. We concluded that transient elevation of blood pressure may occur during ASA. The frequency of hypertension among asthmatics is quite high and concurrent family history of hypertension and frequency of attack of ASA did not seem to determine the status of blood pressure. Patients with asthma should have regular blood pressure check during follow-up visits.

Keywords: *Bronchial, asthma, hypertension, risk factor*

Résumé

Phinews tentatives ont ele effectives afin rdavveliore la prvention poemaire de l'hypertension essentielle, et plurieurs de les tenttives ont toujous ele donge vers l'evitage des derordres pratiqus des muscles de la bouche. Deplus les asthmatiques et les hypertensifs sont sensibles au sel. Il ya une suspission selon laquelle les similarites entre ces 2 maladies pourraient predispose le individuo affectes a une maladie ou une autre. l'une de ces maladies pourrait etre l'hypertension pulmonaire decrite pendant la bronchoconstriction induite a l'excercice. Nous avous par consequent etudies le mode de la pression arterielle pendant et apress les crises d'esthmas severes (ASA) ensemble avec la frequence de l'hypertension chez les patients asthmatiques stables.

Deux groupes de patients avaient ete etudies. Le groupe 2 avait consiste de 12 patients avec de l'ASA (2 males et 10 females), ayant une moyenne d'age de 30 ± 9. 9 ans. La moyenne de la pression arterielle pendent l'attaque de l'asa (147 ± 16.9/100 ± 8.2 mmHg) et plus forts que la moyenne de la pression arterielle, BP (132 ± 8.3 / 82 ± 7 mmHg) 2 famaines apress la sortive de l'hopitale, sans traitement chez tans les patients $P < 0.05$). Le groups 2 comprenait, 134 sujets asthmatiques dans un etat stable (54 males et 80 femelles) ayant une moyenne d'age de 45 ± 15 ans et groupe entre 15 et 90 ans. La frequence totale de l'hypertension etait de 37% avec une proportion de 39% chez les hommes et 35% chez les femmes. L'hypertension avait ete definit commune la Pression arterielle systolique 40mHg, ou une pression arterielle diastolique 90mmHg. Iln'y avait pas de difference entre frequence d'attaque de l'ASA chez les hypertensifs (5.7 ± 5,6/ans) at les non hypertensifs (5.5 ± 3.8/ans), $P < 0.05$. Nous avons conclut que la pression sanquine pourrait survenir during l'ASa la frequence des asthmatiques estassez fortes et unistorique des cas d'hypertension dans la famile concourenent avec la frequence des attacques de l'ASA avait semble ne pas determine le status de la pression arterielle. Les patients ayant l'asthme devrait avoir une pression sanguine requilierement verifie pendant les visites de suovies.

Intoduction

Both asthma and hypertension are two of the most important non-communicable diseases worldwide. Hypertension is however a more sinister disease because of the array of cardiovascular morbid events that often follow it, especially in the developing world. Prevention and treatment of hypertension have thus been the primary objectives of reducing the associated morbidity and mortality.

In the last decade, several attempts were made to improve primary prevention of hypertension and many of hypertension have been found to be associated with high these were directed at avoiding the well known risk factors.[1] One of the most studied is salt intake. Asthma and levels of salt intake[2, 3]. Salt-sensitive hypertension is common in blacks, while dietary salt restriction in asthmatic patients results in improvement of symptomatology with lower consumption of bronchodilators [4]. Both diseases are spastic disorders of smooth muscle[4]. Also, pulmogenic hypertension has been documented in asthmatics during exerciseinduced bronchoconstriction[5]. There is a suspicion that the similarities between these two diseases may predispose the individual with one disease to the other. Thus, we studied the blood pressure pattern during and after acute severe asthma (ASA), along with the frequency of hypertension in stable asthmatic patients in order to provide information on the relationship between asthma and the risk of hypertension in Nigerians.

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Methods

The study was done at the University College Hospital, Ibadan. Two groups of patients were studied. Group I included all patients with purely acute severe asthma who were admitted in to the medical wards between January and December, 1997. Group 2 consisted of all asthmatic patients in "stable state" seen at the medical outpatient clinics during the same period.

Acute severe asthma (ASA) was defined as asthmatic attack characterized by the use of accessory muscles of respiration, respiratory rate of ≥ 30/minutes, tachycardia of ≥110 beats/minutes and pulsus paradoxus.[6]. All the patients with ASA were placed on cotrimoxazole, steroid, salbutamol and /or aminophylline during attack. Most patients in stable state were on salbutamol or steroid inhaler and were free of attack in the preceding eight weeks. Hypertension was defined as systolic blood pressure (SBP) ≥ 140 mmHg and/or diastolic blood pressure (DBP) ≥ 90 mmHg or history of being on hypertensive treatment. The average blood pressure (BP) during attack in group I was calculated from daily BP measurements taken by the same medical registrar usually in the mornings. Patients were considered to be out of attack when patients felt better associated with clinically clear chest, lack of use of accessory muscles of respiration, wheezes and normal pulse rate. BP was measured again two weeks after the attack of ASA was over. The status of blood pressure in the patients at diagnosis of asthma including history of hypertension, were extracted from the patients by direct questioning.

Blood pressure was measured in the stable asthmatics after 5 minutes of rest in the sitting position and average of two measurements taken. A standard mercury sphygmomanometer was used and SBP and DBP were taken as Korotkoff sound phases I & V, respectively. Secondary causes of hypertension were excluded by history, clinical examination, renal ultrasonography, chest X-ray, urinalysis, full blood count, electrolyte and urea and urinary excretion of cathecolamines. Statistical analysis was by t-tests and significance was reached at $P < 0.05$.

Results

Group1

Twelve patients including (2 males) with acute severe asthma were studied during the period. Their ages ranged from 21 to 51 years with a mean of 30±9.9 years. Eight of them (7 females and 1 male) presented at UCH for the first time. The mean duration of attack was 8±1.5 days. Table I shows the clinical data of the 12 patients with acute severe asthma. All the patients developed systolic or diastolic hypertension or both. Average systolic blood pressure was normal in 3 while DBP was normal in 1 subject throughout attack. Only 3(25%) of the subjects had positive family history of hypertension.

Their mean blood pressure during attack of 147 ± 16.9/100 + 8.1 mmHg was significantly higher than the mean blood pressure 132±8.3/82±7 mmHg 2 weeks after discharge from the hospital without treatment of hypertension ($P<0.05$).

There were four patients with blood pressure in the hypertensive range 2 weeks after discharge, but they recorded normal blood pressure 4 weeks later. They are marked with asterisk in table one.

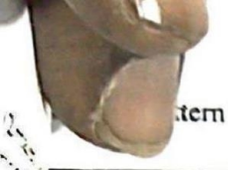
Group 2

There were 134 subjects seen during same period with stable bronchial asthma (54 males) with a mean age of 45 ± 15 years (range 15-90 years). Forty-nine (37%) of them were hypertensive with 21(39%) males and 28[34%] females. The mean age of the hypertensives (52±13.4 years) as compared to that of non-hypertensives (43±15 years) was significantly higher ($P < 0.05$). Table 2 shows the pattern of blood pressure in the 49 hypertensive but stable asthmatics.

Table 1: Clinical data of 12 patients with acute severe asthma

Serial no.	Age	Sex	Pulse/ during	rates resp. attack per/min	Status of BP before attack mmHg	Average BP during attack	Average BP(mmHg) after attack 2 weeks	4 weeks	Family history of hypertension
1	21	F	116	32	Normal	130/90	120/64	-	+
2	36	F	124	40	"	130/100	130/80	-	-
3	51	F	120	36	"	150/100	140/90*	130/80	Don't know
4	25	F	132	30	"	150/80	150/80*	120/82	+
5	25	F	112	32	"	150/110	130/90*	130/80	-
6	19	F	120	30	"	150/105	140/80	-	+
7	21	F	120	36	"	130/100	140/80	-	-
8	23	F	132	44	"	160/100	130/80	-	-
9	40	F	140	40	"	150/110	130/80	-	Don't know
10	25	F	124	36	"	146/13	130/85	-	-
11	37	M	128	30	"	132/98	120/80	-	Don't know
12	37	M	136	32	"	190/100	130/90*	120/70	-
Mean ± SD	30 ± 9		125±8.4	34±4.6		147±16.9 99.8±8.2	132+8.7 82±7.0		

**Patients whose blood pressure normalized 4 weeks after ASA.*



tern of BP in 49 hypertensive asthmatics

atus of BP mmHg	Male n = 21	Female n = 28	M + F n = 49
BP>140 alone	1(5%)	1(4%)	2(4%)
BP ≥90 alone	7(33%)	9(32%)	16(33%)
ystolic + diastolic ypertension	11(52%)	16(57%)	27(55%)
n treatment for ypertension	2 (10%)	2(7%)	4(8%)
otal	2 (100%)	28(100%)	49(100%)

Two (4%) of them had systolic hypertension alone, 16 (33%) had diastolic hypertension alone while 27 (55%) had both systolic and diastolic hypertension. Only 4(8%) of them were on treatment, though 13 of them had hypertension when asthma was diagnosed. Seven of the hypertensives had family history of hypertension whereas, 13 of the non-hypertensive subjects had same. The overall frequency of hypertension amongst the asthmatics studied was therefore 37% with a proportion of 39% in males and 34% in females. Twenty out of 134 subjects (14.9%) had family history of hypertension. Of this number, 7 had both asthma and hypertension. There was no significant difference between the mean frequency of attack in hypertensives (5.7 ± 5.6 per year) as against 5.5 ± 3.8 per year non-hypertensives (P >0.85).

Discussion

Smooth muscle contraction is perhaps the basis of the physiology and reactivity of several systems including the respiratory system. Asthma being a spastic disorder of smooth muscle has been shown to be associated with arterial hypertension syndrome (another spastic disorder of smooth muscle) during exercise-induced bronchoconstriction. This study also confirmed this phenomenon in ASA and further showed that the elevated blood pressure during ASA is transient in nature and may require no treatment, although asthmatics who develop severe hypertension during attack should be treated. Several factors may be responsible for this hypertension and its transient nature. Perhaps the most important is stress which causes production of cathecolamines and increased sympathetic discharge both of which are normal physiologic responses to stress. Drugs used during treatment i.e., infusion of normal saline and salbutamol may also be incriminated in the etiology of the transient hypertension.

The chronic use of some sympatomimetic agents and steroids in some of the patients may also play a part. Oral and nasal spray/inhalation of steroids have been reported to cause elevated intra-ocular pressure in some patients with prolonged use such that one of the patients required medication to control the intra-ocular pressure[7,8]. Moreover the lungs play a significant role in the metabolism of steroid in bronchial asthma, however, in asthmatics there is impaired feed back mechanism of the hypothalamo–hypophysial gonadal system which may be responsible for the stabilization of the transient hypertension later in life [9]. This perhaps may be the origin of hypertension in asthmatics.

It is therefore not surprising that the elevated blood pressure noted becomes normal 2-4 weeks after the attack of ASA even without treatment in all the subjects. However, recurrent bronchoconstriction, increased vascular resistance and hypertrophy which may be due to mediators of asthmatic response like kinins and platelet activating factors, especially in the pulmonary vasculature may lead to subsequent elevation of arterial blood pressure[10]. The recurrent attacks therefore may be expected to predispose the individual asthmatic patient to the development of arterial hypertension. From the group 2 data, the proportion of hypertension amongst asthmatics was 37% which is quite high, though not directly comparable, it is much higher than the overall prevalence of hypertension (11.2%) in the Nigerian population.[11]. This suggests that asthmatics may be at a higher risk of developing hypertension than the normal population. The hypertensive subjects were also noted to be much older than their non-hypertensive counterparts, reflecting the trend of blood pressure increasing with age and the fact that frequency of hypertension generally increases with age.

The recurrent, transient elevation of blood pressure during attack may thus be a factor, but the frequency of attack in hypertensive asthmatics was the same with non-hypertensive asthmatics, suggesting that the frequency of attack is of no consequence. Although, a marked correlation has been found between increased pulmogenic hypertension and some indices of bronchoconstriction like peak expiratory flow rate in some studies[3].

It is therefore, concluded that transient elevation of blood pressure may occur during attack of acute severe asthma. The frequency of hypertension seems to be high amongst asthmatics but the frequency of attack and family history of hypertension did not seem to determine status of blood pressure in asthmatics. It is also recommended that patients with bronchial asthma should have regular blood pressure checks during follow-up visits.

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