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ORIGINAL ARTICLES

Determination of Predictors for Peak Expiratory Flow Rate (PEFR) in Normal Adults (18-45 Years)

Md. Zakir Hossain Sarker[1], Md. Ali Hossain[2], Md Rashidul Hassan[3], Muhammad Murtaza Khair[4], Mirza Mohammad Hiron[3], Asif Mustaba Mahmud[6], Md. Mostafizur Rahman[7], Md. Mohiuddin Ahmed[8], Md. Khorshed Alam[9], Samir Majumder[10], Abdur Rouf[11], Abdullah Al Mamun[12]

Abstract

A cross sectional prospective study was conducted to find out the correlation of various anthropometric parameters with PEFR in different age and sex of Bangladeshi adult population. Peak expiratory flow rate (PEFR) is a lung function test which is easily measurable and reproducible but base line value of PEFR has not been studied in large scale among Bangladeshi adults.

A total of 3401(2515 men and 886 women, M F= 1: 0. 35) normal adults (18 to 45 years), were selected randomly to obtain peak expiratory flow rate (PEFR) from seven different areas of Dhaka city. The mini-Wright peak flow meter was used to measure peak flow rate in a standard way. The highest of three reading was taken as the correct value. Anthropometric parameters including body weight and height were recorded appropriately and body surface area was calculated. Data were analyzed by SPSS program.

Significant correlation was found between PEFR with height, weight, surface area and age. The regression equations for PEFR were determined for adult male and female considering height, weight, surface area and age separately as independent variables. Correlation of height with PEFR was the highest in comparison to other anthropometric parameters (weight, surface area and age). Men had significantly higher values of PEFR than women at any height. PEFR of Bangladeshi adult were nearly similar to Indian but lower than that of American and African adult.

[Chest & Heart Journal 2005; 29(2) : 79-89]

Introducdtion:

Pulmonary function testing has come to assume a central place in the practice of pulmonary medicine. Pulmonary function tests of various types are used clinically and epidemiologically to measure functional status in order to assess the disease[1]. Breathing tests have several important uses like to screen for the presence of respiratory impairment, to measure its magnitude, to look for diagnostic patterns of abnormality as part of the investigation of respiratory symptoms, as well as in epidemiological research. Good quality control and appropriate reference values are essential.

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The peak expiratory flow rate is the fastest flow rate that can be sustained for 10 milliseconds at the start of a maximal expiration after full inspiration. The measurement is simple and reproducible and reliable way of judging the degree of airway obstruction in various obstructive pulmonary diseases, specially asthma. Peak expiratory flow rate is easily measured by using a mini-Wrights peak flow meter (mWPFM)[2], which is easy to use, reliable and can be recorded even by the patients at home[3-6]. This instrument is cheap, portable, understandable and useful for physicians in managing patients with respiratory diseases.

PEFR measurement can reveal the diurnal variability of airflow obstruction of patient has been suffering from reactive airway diseases[7,8] that provides early clue to the diagnosis and management. Fall of peak expiratory flow rate in a child with asthma is an impending sign of acute asthma. The response to treatment can be monitored by using serial PEFR measurement[9]. Peak expiratory flow rate measurement gives the idea of status of airway caliber of respiratory system and regulatory function of respiration which some times are affected by certain progressive neurological diseases. As no physician can understand the status of progress and treatment of diabetes mellitus without doing simple blood sugar test, no clinician could manage a patient with potential renal failure without an estimation of blood urea level; PEFR can be used as pulmonary function test in the same way[10,11]. The occurrence of diurnal variation of symptoms and airway resistance in asthmatics are well perceived, thereby early intervention of treatment pattern and efficacy of drug can be documented by measurement of peak flow rate[12,14]. PEFR can be used not only to evaluate the airway obstruction, can also be used to classify the severity of airway obstruction and its management and as a guide line of admission and discharge of asthma patients[14]. It can also be used to categorize and asses the treatment response of the COPD patients.

Normal lung function values serve as important references for diagnosis of respiratory disorders. These values vary with race and human settlements and are affected by environmental, nutritional, genetic and anthropometric factors. They are hardly the same for any two populations or communities. Hence, the need for each community or population to have its own reference values. There is no such reference values for Bangladesh. The absence of such data encourage reliance on foreign and most times western standards even when those may not correctly represent the normal local values and could lead to misreading of the degree of respiratory dysfunction in indigenous patients. It is also important to know which one of the anthropometric factor has got maximum representation of peak expiratory flow so that a simple reference table can be prepared.

This study examined the PEFR values in normal healthy adults of Dhaka city with a view to highlighting the need for a comprehensive national study to develop a reference table which will help in diagnosis and proper monitoring of obstructive airway diseases specially bronchial asthma, to find out the correlation of PEFR with various anthropometric parameters in different age and sex.

Material and Methods:

The study was carried out from 1st July, 2000 to 30th June, 2002, for a period of twenty four months. Seven different areas in Dhaka city were included in this study namely Lalbag, Khilgaon, Gopibag, National Medical College, Shaheenbag, Arjat Para, and Nakhal Para. It was a prospective cross sectional study. Total 3401 volunteers of both sexes from all socioeconomic status were targeted and collected accordingly.

From 90 (ninety) wards of Dhaka city 7 (seven) were selected by lottery and from each ward 7 (seven) areas (mohalla) were included in the same way. From every selected area main road was chosen purposively. House holds situated on the right side of the main road were targeted purposively. Every alternate house of the selected side was included into the study. Informed consent was taken prior to the procedure. A detail history was taken from adults (both male & female) between the ages of 18-45 yrs before inclusion into the study. Adults who fulfilled the inclusion criteria were examined properly and questionnaire were filled up appropriately. Height was measured and weight was recorded on bare foot and wearing light clothes. Seven high range

(60-800 1/min) models well functioning mini Wright Peak flow Meter (mWPFM) were used to record PEFR (1,/min). Initially all the family members or group of adults were demonstrated how to use mWPFM correctly. For each determination the person was instructed to make a maximal inspiratory effort and then to make the maximum and most rapid expiratory effort possible in standing position. After initial demonstration of the procedure, majority of the subjects were asked to repeat the procedure 2-4 times on trial basis, to make the person familiar with the technique. Then serial 3 blows for PEFR were registered in individual sheet after the person had become familiar with the technique. Average 34 volunteers could participate in each day of total 100 days visits. Sample collection was started from the first week of July 2000 and completed by last week of June 2002. Before going to the locality a pilot programme was conducted to teach the investigators about the use of mWPFM, to demonstrate the reproducibility of PEFR. Piloting was done at Asthma Centre, NIDCH among the normal adults accompanying asthma patients who are not suffering from Respiratory diseases.

Normal healthy adults of both sexes aged 18 to 45 years were included in this study.

Adults that have been suffering from asthma or any obstructive airway diseases or history of ARI within two weeks or history of atopic conditions like eczema and hay fever were excluded.

Statistical Analysis

Statistical analysis was done using the statistical package for the social science (SPSS) program in computer. Linear and multiple regression analysis were performed by using age, weight, height and surface area as the independent variables and PEFR as the dependent variable. Independent sample test and group test statistics were also done.

Results:

The study population included 3401 normal healthy adults of both sexes from seven different areas of Dhaka city namely Lalbag, Khilgaon, Gopibag, National Medical College, Shantibag, Agat Para and Kalachadpur (Tble-1). Among them 2515 and 886 were male and female respectively. Male and female ratio was 1: 0.35 (Table-II). Distribution of study population according to age interval was analyzed and maximum male population was in 20-24 years of age and female was in less than 20 years of age (Table-III). Mean age, height, weight and surface area was calculated (Table-IV).

Table-I

Study population accord in to locality (n=3401)

Locality	Male	Female
Lalbag	591 (23.5%)	211 (23.8%)
Khilgaon	458 (18.2%)	146(16.5%)
Gopibag	197(7.8%) -	78(8.8%)
National M College	253 (10.5%)	92(10.5%)
Shantibag	289(11.5%)	147(16.5%)
Ariat Para	318(12.5%)	105 (11.8%)
Kalachadpur	409(16.0%)	107(12.1%)

Table-II

Sex distribution (n=3401)

Sex	Number	Percentage	M:F ratio
Male	2515	73.9	
Female	886	26.1	1:0.35
Total	3401	100	

Table-III

Distribution of 3401 subjects according to age

Age (years)	Male	Female
<20	287(11.4%)	214(24.2%)
20-24	564(22.4%)	212 (23.9%)
25-29	481 (19.1%)	127 (14.3%)
30-34	496(19.7%)	172(19.4%)
35-39	377(15.0%)	94(10.6%)
>39	311 (12.4%)	67(7.6%)

Table-IV

Anthropometric measurements (n=3401)

Sex	Male	Female
Mean age(±SD)	29.28±9.36	26.57±7.77
Mean height(±SD)	159.8±5.9	153.5±4.6
Mean weight(±SD)	61.6±6.8	57.1±7.0
Mean surface area(±SD)	1.67±0.11	1.59±0.12

PEFR values were higher in the age group of 25-29 years in both the sexes in this study (Table-V). During analysis of height interval it was seen that PEFR (L/min) was maximum between 171-190 cm group (Table-10).

The regression equation (Table-VIII) was derived from the regression analysis and ANOVA test where PEFR of individual person was considered as dependent variable and other anthropometric parameters as independent variables. These regression equations enabled us to construct the nomogram.

Table-V

PEFR (</min) of Bangladeshi adult in relation to age interval (n=3401)

Age (years)	Male PEFR ± S.D. (L/min)	Female PEFR ± S.D. (L/min)
<20	472 ±65	370 ±38
20-24	508 ± 64	383 ±52
25-29	510 ± 49	_ 391 ± 54
30-34	508 ± 49	387 ± 44
35-39	501 ± 51	384 ± 44
>39	485 ± 50	348 ± 64

Table-VI

PEFR (1/min) of Bangladeshi adult in relation to height interval (n=3401)

	Male		Female	
Height interval (cm)	N	PEFR (+/- SD)	n	PEFR (+/- SD)
130-140	3	373.3(63.5)	3	353.3(50.3)
141-150	90	427.7(73.8)	227	353.9(41.3)
151-160	1365	489.9(51.1)	596	385.1(42.7)
161-170	945	518.8(48.71)	58	420.6(81.2)
171-190	112	524.65(78.2)	2	460.0(14.1)

PEFR values (both mean and range) were calculated. The values were found

Table-VIII

PEFR I/min according to sex (n=3401)

Sex	PEFR (I/min) Best of 3		
	Mean	Standard Dev.	Range
Male	500	57	250-800
Female	380	49	200-610

significantly lower in women than that of men (Table-VII).

Table-VIII

Regression equation for prediction of PEFR (L/ min) from different independent variables

Model Variable	Sex PEFR (1/min)	Independent	Regression equation	SEE*
Male	Ht (cm)	3.51x Ht- 60.3	52.77	
1	Female	Ht (em)	3.75x Ht-195.4	45.91
	Male	Wt (kg)	2.89,\ Wt +321.9	53.18
2	Female	Wt (kg)	1.59x Wt +288.5	47.18
	Male	S.Area(m.sq)	188.8,\ SA+185.3	53.00
3	Female	S.Area(m.sq)	100.2x SA+219.7	47.60
	Male	Ht & Wt	2.63,\ Ht + 2.Ox Wt -44.8	51.20
4	Female	Ht & Wt	3.39x Ht +1.13x Wt- 204	45.30
	Male	Ht,Wt &	2.97x lit- 15.60xWt+1123.76x	50.30
5	S.Area	SA -887		
	Female	Ht,Wt & S.Area	3.42x Ht- 11.72x Wt +768.90x SA -702	44.60

*Standard error of the estimate

To explain and predict the changes in the magnitude of a given variable (PEFR) in terms of one or more variables (Height, Weight, Surface area, age, Smoking Habit etc) regression analysis can be conducted. The most well known method of estimation procedure is the ordinary least square (OLS) method. In regression analysis we express the relationship between the dependent variables and the independent variables by means of an equation. The OLS regression model for estimating PEFR in terms of explanatory variables may be expressed in the following form.

$PEFR = \beta_0 + \beta_1 H + \beta_2 W + \beta_3 SA + \beta_4 A + \Sigma$

In this máodel the dependent variable is PEFR and independent variables are Height (H), Weight (W), Surface Area (SA), and Age (A). $\hat{a}_0$ $\hat{a}_1$, $\hat{a}_2$, $\hat{a}_3$, $\hat{a}_4$, $\hat{a}_1$ are regression coefficient and å is error term in the model. An OLS regression will be run to find out the principal determinant of PEFR. By statistical significance test we shall determine whether the explanatory variables have any significant relationship or not.

Now we can discuss the estimate of the parameters for the PEFR OLS regression model. The level of significance for all the tests is considered to be 0.05. The estimated values for the PEFR model along with their t-values, model F values, coefficient of determination, adjusted coefficient of determination and p-values are given in the table-13.

OLS PEFR regression model revealed that the intercept was significant for the PEFR model. Height, Weight, and Surface area were highly significant and age was also significant at .5% level of significance. Model F was highly significant. Multiple and adjusted multiple coefficient of determination were .601 and .524, showing explanatory power of the model was good.

Simple regression model of PEFR for different independent variables can be considered. In that model Height was considered as an independent variable.

$PEFR = \beta_0 + \beta_1 H + \beta$

The intercept was significant for the PEFR model (Table-X). Height was highly significant. Multiple and adjusted multiple coefficient of determination were .371 and .363.

When weight was considered as an independent variable and set up a simple regression model of PEFR as follows.

$PEFR = \beta O + \beta 2W + \Sigma$

Table-IX

Estimation Results of OLS PEFR regression model

Variable	Parameter	Estimate value	t_{cal}	P-value	$F_{mode\ el}$	R^2	R^2
Intercept	β_0	-	15.899	.000			
			1351.495				
Height	β_1	5.698	30.562	.000			
Weight	β_2	-15.518	-8.636	.000	474.478	.601	.524
Surface area	β_3	1134.225	10.182	.000			
Age	β_4	-.428	-2.783	.005			

Table-IX

Estimation Results of Simple OLS PEFR regression model (Height)

Variable	Parameter	Estimate	t_{cal}	P-value	$F_{mode\ el}$	R^2	R^2
Intercept	β_0	-571.793	-20.644	.000	810.2	.371	.363
Height	β_1	6.579	37.591	.000			

Table-XI

Estimation Results of Simple OLS PEFR regression model (Weight)

Variable	Parameter	Estimate	t_{cal}	P-value	$F_{mode\ el}$	R^2	R^2
Intercept	β_0	204.175	20.215	.000	670.4	.251	.233
Height	β_1	4.379	26.361	.000			

Table-XII

Estimation Results of Simple OLS PEFR regression model (Surface area)

Variable	Parameter	Estimate	t_{cal}	P-value	$F_{mode\ el}$	R^2	R^2
Intercept	$\hat{a}_0$	11.084	0.646	.000	715.01	.261	.257
Height	$\hat{a}_1$	277.739	26.747	.000			

Table-XIII

Estimation Results of Simple OLS PEFR regression model (age)

Variable	Parameter	Estimate	t_{cal}	P-value	$F_{mode\ el}$	R^2	R^2
Intercept	$\hat{a}_0$	439.492	87.114	.000	35.579	.101	.101
Height	$\hat{a}_1$	1.020	5.965	.000			

Table-XIV

Estimation Results of Multiple (Height & Weight) OLS PEFR regression model

Variable	Parameter	Estimate	t_{cal}	P-value	$F_{mode\ el}$	R^2	R^2
Intercept	β_0	-537.695	-19.953	.000	861.495	0.336	0.336
Height	β_1	5.442	29.219	.000			
Weight	β_3	2.414	14.806	.000			

Intercept was found significant for the PEFR model (Table-XV). Weight was highly significant. Multiple and adjusted multiple coefficient of determination were about .251 and .233.

When surface area was considered as an independent variable, the intercept was significant for the PEFR model. S.Area was highly significant. Multiple and adjusted multiple coefficient of determination were about .261 and .257 (Table-XVI).

In case of Age the intercept was significant for the PEFR model. Age was highly significant. Multiple and adjusted multiple coefficient of determination were about .101.

From above findings it was obvious that the Multiple and adjusted multiple coefficient of determination of height is more (.371 and .363) than other independent variables like weight, surface area and age.

When Height and Weight were considered as independent variables and set up a multiple regression model of PEFR as follows.

$$PEFR = \beta_0 + \beta_2 H + \beta_3 H + \Sigma$$

There we saw the intercept, height and weight were highly significant for PEFR model (Table-XIV). Similar findings were seen in case of height, age and height, surface area.

Table-XV

Estimation Results of Multiple (Height and Age) OLS PEFR regression model

Variable	Parameter	Estimate	t_{cal}	P-value	$F_{mode\ el}$	R^2	R^2
Intercept	β_0	575.316	-20.778	.000	712.905	0.296	0.295
Height	β_1	6.522	36.092	.000			
Age	β_3	0.443	3.050	.002			

Table-XVI

Estimation Results of Multiple (Height and S. Area) OLS PEFR regression model

Variable	Parameter	Estimate	t_{cal}	P-value	$F_{mode\ el}$	R^2	R^2
Intercept	β_0	-650.871	-23.917	.000	881.475	0.342	0.342
Height	β_1	5.423	29.421	.000			
S. Area	β_3	159.052	15.730	.000			

But the value of R' was highest (0.342) in multiple (height and s. area) OLS PEFR regression model.

Nomogram of normal PEFR (L/min) were constructed from regression equation to show relationship with height weight and surface area in both the adult male and female.

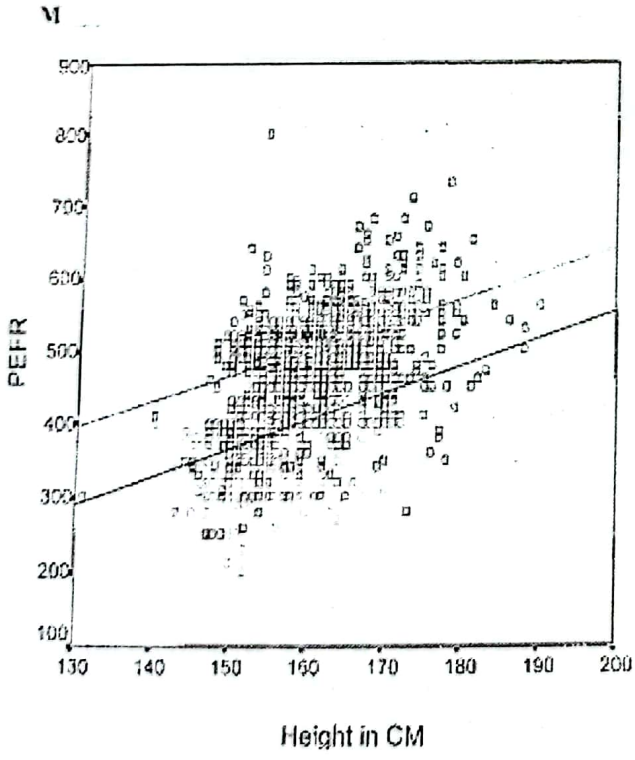

Fig.-1 : *Peak expiratory flow (L/min) in relation to height (male & female).*

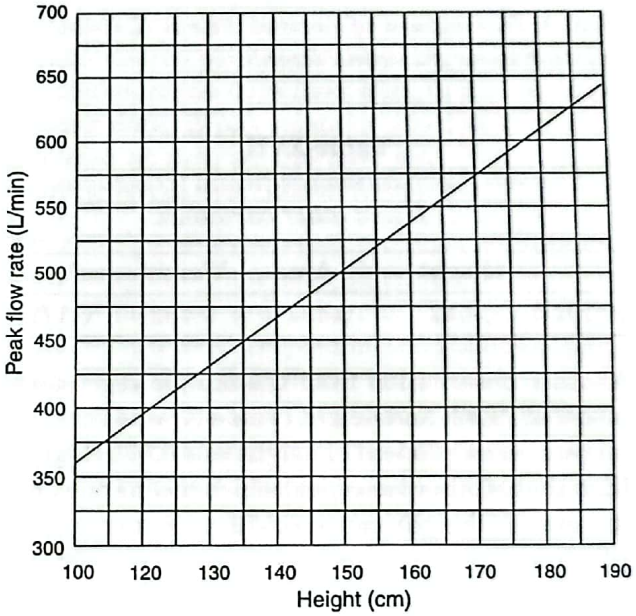

Fig.-2: *Nomogram of normal PEER (L/min) in relation to height (Bangladeshi male).*

It was observed that the Pearsons correlation co-efficient between PEFR and height was 0.642 (highest) which means strong positive relationship height and PEFR. Again surface area and weight had also positive relation with PEFR but age had very weak positive relationship with PEFR (Table-XVII).

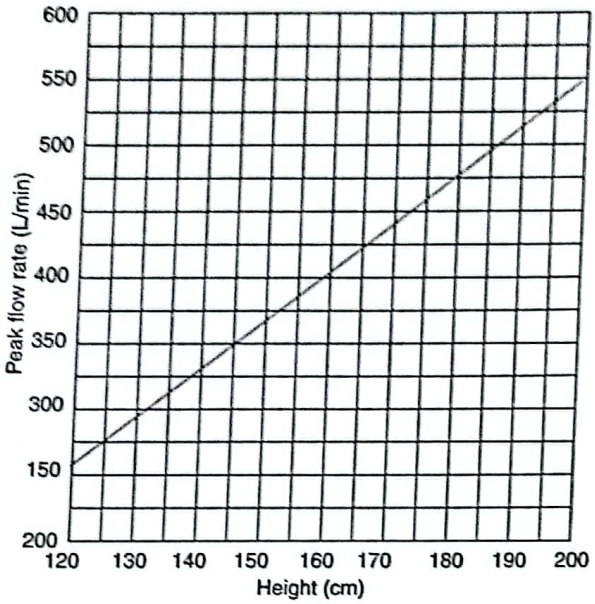

Fig.-3: *Nomogram of normal PEFR (L/min) in relation to height (adult female).*

Table-XVII

Pearson's correlation coefficient (r) between PEFR and other variables:

	Height	S. Area	Weight	Age
PEFR	0.642	0.487	0.482	0.172

Another observation from the simple regression model of PEFR and height (Table-X) was that the regression co-efficient of height was 6.579 (higher than that of other variables) which means one unit increase in height cause 6.579 unit increase in PEFR and the findings was concordant with the correlation co-efficient.

Discussion

Peak expiratory flow rate (PEFR) of 3401 normal adult (18 to 45 years) from seven different areas in Dhaka city were measured to determine relation among different factors and between male and female populations and to understand the normal value (Table-IV). Male female ratio was 1:0.35 (Table-II). In this study significant difference of values of PEFR (litre/minute) in relation to height, weight, age, and body surface area was found specially in respect to height. PEFR values of women (in relation to height) were always lower than that of the men which was statistically significant. The difference of PEFR in male and female were observed by other investigators[15]. Same difference was also observed in different studies between boys and girls[16]. The factors that determine PEFR are predominantly expiratory muscle effort, lung elastic recoil pressure and air way size[17]. The muscle effort intern depends on the physical strength and physical activity. It is possible that these lower values in women were due to physiological reason.

The positive correlation of PEFR with height, age, weight and body surface area was observed in both the male and female which means that the value of PEFR increased with increase in those anthropometric parameters. The most significant correlation was observed between PEFR and height (Fig-3) similar to other studies[16] Thus the height had been found to provide a good basis for prediction of normal values of PEFR. Other investigators[18] also found the superiority of height as an independent parameter which correlated well with PEFR and other ventilatory functions. Pulmonary measurements such as forced vital capacity (FVC), forced expiratory volume in one second (FEV1 1) and peak expiratory flow rate (PEFR) are best correlated with height[17]. One study[19] observed that for clinical evaluation of child's lung function, height was the best independent parameter in comparison to age and weight. Several recent studies had also shown the highest correlation coefficient between PEFR with height. There was no disagreement regarding positive correlation of PEFR with height as an independent body parameter. Standing height is the best predictor of PEFR in children and height should have the first preference for prediction of PEFR because of more accuracy, easily measurable at any place and its highly significant relationship with PEFR.

Significant association was observed between PEFR with body surface area and with body weight. But the correlation of PEFR with body surface area was more significant than that of weight. On the other hand, the level of significance of correlation of PEFR with body surface area and with weight was less than that of height. Such variation may be due to wide variation in weight and height within same age group.

Age is the variable which has also positive correlation with peak flow rate (PEFR) in this study

(Table-XVII). But correlation coefficient values are less than that of the height, surface area and weight (Table-XVII). Another study[16] concluded that it is lower than height but higher than weight and surface area. It could be because his study population was children aged 5 to 15 years, where with increase age muscular development also increases causing better performance.

OLS PEFR regression model revealed that the intercept was significant for the PEFR model. Height, Weight, and Surface area were highly significant and age was also significant at .5% level of significance. Model F was highly significant. Multiple and adjusted multiple coefficient of determination were .601 and .524, showing explanatory power of the model was good.

When height was considered as an independent variable, the intercept was significant. for the PEFR model (Table-X). Height was highly significant. Multiple and adjusted multiple coefficient of determination were .371 and .363. When weight was considered as an independent variable, the intercept was round significant for the PEFR model (Table-XI). Weight was highly significant. Multiple and adjusted multiple coefficient of determination were about .251 and .233.

Similarly for surface area the intercept was significant for the PEFR model. S.Area was highly significant. Multiple and adjusted multiple coefficient of determination were about. 261and .257 (Table-XII).

In case of Age the intercept was significant for the PEFR model. Age was highly significant. Multiple and adjusted multiple coefficient of determination were about .101.

So it was obvious that the Multiple and adjusted multiple coefficient of determination of height is more (.371 and .363) than other independent variables like weight, surface area and age.

When Height and Weight were considered as independent variables it was seen that the intercept, height and weight were highly significant for PEFR model (Table-XIV). Similar findings were seen in case of height, age and height, surface area.

But the value of R^2 was highest (0.342) in multiple (height and s. area) OLS PEFR regression model.

Nomogram of normal PEFR (L/min) were constructed from regression equation to show relationship with height (Fig-2,3) in both the adult male and female.

Scatter diagrams were prepared which revealed maximum relation of height with PEFR than other variables (Fig-1).

Sample size of the study was reasonably large enough (n=3401). Four physicians working in the field of respiratory medicine were involved in the data collection procedure in every step. Seven peak flow meters of good quality were selected for the procedure. Each and everyday prior to data collection, the validity of the devices was checked. Intensive quality control measure was taken at every stage of the study. The investigators were provided extensive supervision. They were directly involved in data collection at field level. Filled up questionnaires were rechecked every night after completion of the days field work to find out any missing data. If any mistake was found, it was corrected at field on the next day. This type of study is for the first time in Bangladesh.

However, the study suffers from some limitations. Like, sample was not selected randomly, because it is not possible in Dhaka city. Though cluster sampling is a well accepted method in developing countries. During data collection female participants were less than that of male, because of conservativeness. Even then required sample size was fulfilled. The required number of each age group could not be strictly followed because of limited time and fund. With all these the study can be regarded as representative.

Gregg, 1. & Nunn, I.J[20] carried out a study on peak expiratory flow on normal subject in South West London among 202 men and 199 women to show the relationship between peak expiratory flow, age and height. They found the relationship between PEFR & age as curvilinear. In this study it was also observed that PEFR values increase gradually up to certain age limit then do fall again. All the variables were initially included then eliminated in turn if their additional contribution to the regression sum of squares was not statistically significant. Their findings suggested that in both sexes PEF does not begin to decline until about the age of 35 years. They also found that small departure from the correct technique

of performing the test may cause spuriously low value of PEF. And in general it is more difficult to persuade women to make a maximal effort than men.

In present study it was observed that PEFR values rises gradually with the increase of age and reaches maximum in the age group of 25-29 years in both the sexes. Similarly, it is difficult to persuade women to make a maximal effort than men and a small departure from the correct technique of performing the test causes spuriously low value of PEF. They also found relationship of PEFR with age and height which is consistent with present study.

Natarajan, S. and Radha, K. (1978) studied peak expiratory flow rate in normal south Indians (1850 men and 210 women). The highest readings for men were obtained in the age group of 21-25 years and for women in the age group of 17-20 years. In this study highest readings for both the sexes were in the age group of 25-29 years. In women the values were consistently lower than those in men of the same age and height. Similar lower values for women were observed in this study. The highest individual value recorded in their study was 720 L/min for a man aged 23 years weighing 63 kg and of a height of 175 cm. In present study the highest value is 800 L/min in a man aged 27 years weighing 65 kg with a height of 176 cm. Their another observation was that if height is eliminated for analysis of the data and all the value of a particular age group are pooled there is no appreciable difference in the PEFR of young women belonging to the age group 17-35 years, which shows height is one of the most important factor which determines the PEFR in an individual.

From the simple OLS PEFR regression model it is seen that the intercept was significant for the PEFR model. Height is highly significant, multiple and adjusted multiple co-efficient of determinations are .371 and .363, showing explanatory power of the model is not good. Explanatory power of height is more (.371) than that of any other variable like weight, surface area and age. Another observation is that combination of height and surface area represents more than combination of height and weight or height and age.

Nomogram for predicting normal values of PEF, derived from the data of this study, have been prepared.

In this study it is evident that mean expiratory flow for men is 500+/- 57 L/min and 380+/-49 L/min for women. A study conducted in India (Shah and Mehta, 1961) showed PEFR 500 L/min for adult male and 400L/min for female. So, it is almost similar to this study. It may be explained by same environment and identical build of the population. Though other study (SK Mallik et al. 1975) showed it 483+/-25 in India.

In Kenya Orie-NN (1999) in his study on Comparison of normal respiratory function values in young Kenyans with those of other Africans and Caucasians included 88 apparently healthy young Kenyans. The study revealed mean values of (male versus female) PEFR 586.30 ± 8.54 versus 438.30+/-8.55. The values for females were 25-26% lower than those for males. The FEV1, FVC and PEFR for both males and females correlated positively with heights. In present study the PEFR value is much lower (for male 550 + 57 and for female is 380 + 49). It could be due to increased height, better build and decreased environmental pollution. Similarly PEFR values were found always lower in female than male subjects and PEFR for both males and females correlated positively with heights.

Leiner et al, (1963) at New Jersey found the mean value of 600+/-70 for adult male; this higher value can be explained by same way.

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Role of Intravenous Magnesium Sulphate in Severe Acute Asthma

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Abstract

Magnesium is required after wide variety of cellular activities and biological process. It causes relaxation of bronchial smooth muscle. It is also known to reduce the amount of neurotransmitter released at motor neuron terminals diminish depolarisation action of acetvlcholine at the neurotransmitter end plate and depress excitability of smooth muscle membrane. Magnesium is necessary, for steps involving the interaction of beta-agonist receptor complex, G-protein and GTP leading to activities of adenylylcyclase. It reduces superoxide production in neutrophil. Therefore magnesium has an anti inflammation for the sustain improvement in pulmonary, function test even several hours after magnesium was administered.

This is a randomised controlled clinical trial. This study enrolled 60 patients with severe acuic asthma with FEV1 less than 30% of predicted of individual and age between 18-55 Years not needing assisted ventilation. After measurement of FEV_1 and PEFR all patients received 200 mg hydrocortisone intravenous, nebulised with 2.5 mgs ulbutamol and oxygen. Al the .same time study group patient (n=30) received intravenous 2gm magnesium sulphate in 50 ml and the control group patient (n=30) received intravenous 50 ml of normal saline. Magnesium .sulphate or normal saline was infused over 20 minutes. Afer 30 minutes at the end of the infusion, FE V_1 and PEFR were measured.

Initially, before infusion of $MgSO_4$ or saline PEFR L/min, PFER % predicted and FEV_1, L, FEV_1% predicted were similar in two groups. After 30 mins at the of infusion the PEFR L/min and PEFR % predicted were higher in studv group and it was 188.5 L/min vs 171.4 L/min and 42.5% vs 38.1 % respectively and which was statistically significant ($p<0.001$). Similarly FEV_1, L and FEV_1, % predicted were greater in study group and it was 0.87 L vs 0.81 L and 27.7% vs 25.6% respectively which was also statistically significant ($p<0.001$). This prospective controlled study concluded that Intravenous magnesium sulphate has better and rapid bronchodilating effect, patients treated with intravenous magnesium sulphate get quick relief from distress and no unwanted side effect is present.

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Introduction

Asthma is the most common respiratory crisis encountered in clinical practice. It is estimated that in the USA 1.8 million patient each year seek care for acute episode in emergency department with cost in excess of $430 million. Yet, despite this clinical and financial burden general agreement has yet to be reached about the best way to treat the acutely presenting patient and fundamental issues such as the choice of drugs and duration of treatment have not been resolved[1].

The morbidity and mortality rates for asthma continue to increase during a period when medical interventions have produced a decline in the morbidity of many other childhood diseases[2]. Globally it affects about 100 million people[3]. Asthma affects 3%-5% (approximately 14-15 million) of the US population including 6-9% (4.8 million) under 18 years of the age making it frequently encountered clinical problem both in pediatric and adult population and major cause of morbidity in the USA and around the world[4]. In our country, according to the First National Asthma Prevalence Study, 1999[5] about 7 million people (5.2% of the total population) are suffering from asthma. At least three episodes of asthma attack in the last 12 months. More than half of these patients are innocent children that are 7.4% of the total pediatric population (I-15 years age group).

Recent epidemiological data for acute asthma suggest there are about 5,00,000 hospitalization per year in the USA of which 65% occur in patient over 18 years of age. Acute asthma represent 4% of all emergency department visit involving about 2 million people. About 15% and 25% of the emergency department visit for acute asthma result in hospitalization. About 20-30 of the patient initially managed and discharged from emergency department have relapse[4].

Globally an increase in the asthma mortality has been noticed over the past 15 years[6]. From 1984 to 1994 the national hospitalization in USA rate for asthmatic children increased by 17%. The national death rate for asthma in children and adult are more than doubled from 1975 to 1995[7]. In UK, death from asthma rose steadily during 1980s, peaked in 1988 and have been falling during 1990s. However admission to hospital for acute severe asthma has not decreases and studies of asthma deaths, near fatal asthma and asthma admission reveal potentially preventable or avoidable factor in more than 75% of patient[8].

Despite their effectiveness some percentage of the patient with acute asthma fail to respond to beta-2 agonist and as many as 30% of the patient presenting emergency department fails to responds adequately to beta-2 agonist and require hospitalization[9]. Currently the cornerstone of the therapy for acute asthma is the rapid reversal of the patients airways obstruction. The main stay therapy for acute exacerbation is nebulised beta-2 agonist therapy repeatedly every 20 minute for one hour (serial 3 nebulisation) as initial therapy.

Early in the course of treatment systemic corticosteroid should be administered to patient with moderate to severe exacerbation or to patient who fail to respond promptly and completely to inhaled beta agonist[10]. . Regarding steroid it required hours to demonstrate significant benefit. Incase of hydrocortisone which is given intravenous, the peak effect on airway mechanics may be achieved more rapidly the time between administration and onset of benefit in asthma being thought to be about 5 hours compared with about 8 hour for prednisolone.

Addition of high dose of inhaled ipratropium bromide 0.5 mg in adult to an aerosolized solution of selective beta agonist has shown additional benefit in severe asthma exacerbation than either drug alone but again such improvement are usually small so that some workers reports no benefit whereas other repot trends that fail reach statistical significance[11].

Given the slowness of the onset of action anticholinergic and 60-90 minutes lag time before achieving a peak effect and their relatively limited bronchodilator activity, anticholinergic agent such as ipratropium bromide are not 1st line therapy for acute asthma. They do not add any therapeutic benefit to the effect of albuterol given in divided doses over I hour nor do they facilitate recovery in patient whose immediate response to sympathomimetics is impaired[12].

In the emergency department theophylline is not recommended because it appear to provide no additional benefit to optimum inhaled beta agonist

therapy and steroid and increase the adverse effect[13]. It has narrow therapeutic index and frequently associated with adverse effect even in therapeutic dose. Patient vary in dose requirement to keep plasma level therapeutic[11]. Intravenous theophyllin is less effective than nebulised beta agonist and should therefore be reserved for the few patients who fail to respond to beta agonist

Patient like severe acute asthma is hypoxaemic and struggling to breath as a result of severe bronchoconstriction. After administration of intravenous aminophylline an adverse effect like a grand mal seizure may deliver the coup de grace and should be avoided especially rapid administration[11]. Urgent level should be requested if suspicion about the patient taken theophyllin orally. Despite the refinement in therapeutic strategy for acute asthma emergency department visit and hospitalization continue to account for predominant proportion of health care costs for asthma.

These facts stress the need for the innovative emergency department based intervention. An efficient asthma adjunct is needed to help bridge the time to onset of corticosteroid therapy effects in subpopulation of patient with acute asthma who are resistant to standard bronchodilator treatment. This ideal drug should be fast acting safe and effective.

This study was carried out to search for adjunct to standard therapy in patient with severe acute asthma rapid recovery from severe acute asthma, to determine that there is better response when intravenous magnesium sulphate is used in the treatment of severe acute asthma and to elucidate any adverse effect of magnesium sulphate.

Materials and Methods

It was a prospective randomized case control study carried out at Asthma Out Patient Department, National Institute of Disease of the Chest & Hospital (NIDCH), Mohakhali. Dhaka from January 2003 to December 2001. Population of the study was known case of asthma patient or newly diagnosed asthma patient who presented to the emergency department with an acute asthma exacerbation . Inclusion Criteria was known cases or newly diagnosed bronchial asthma patient of either sex,age 18-55 years. FEV1 below 30% of predicted and non-smoker. Febrile,.any evidence of lower respiratory tract infection i.e. productive sputum., any history or evidence of cardiac, renal or hepatic dysfunction smoker (former or current smoker),pregnant women, breast-feeding mother, poor level of patients co-operation cyanosed patients were excluded.

60 patients were included in this study. Group-1 (n=30) patients were treated with 2gm MgS04+steroid+sulbutamol. Group-11 (n=30) patients were treated with normal saline + steroid+sulbutamol. Both groups treated with oxygen through out the study period. Patients were allocated into two groups randomly. Every alternate patient attended in the emergency room who met the criteria included sequentially in each group.

In each case, patient's consent was taken for the control and study groups for enrollment in this study. A standard proforma and questionnaire was designed and filled to identify patient with acute asthma. The patients were identified as acute asthma patient according to predominant criteria following history, clinical examination and objective measurement of the airway obstruction.

As the patient presented to the emergency department of asthma center with acute respiratory distress, they were clinically assessed with brief history of attack duration medication used etc. with a view to establish the diagnosis of asthma as well as severity assessment with the help of severity assessment chart. Physical examination of the patients were done with regards to the vital signs of acute attack as well as the chest findings. The exclusion criteria were ruled out. Questionnaire were filled. Objective measurement of airway obstruction were recorded with spirometer & peak flow meter . The procedure of using the spirometer & peak flow meter were demonstrated to the patient.

Each patient received one unit of 0.5 ml salbutamol (2.5mg) diluted in 3ml normal saline via wet nebuliser, iv hydrocortisone 200 mg , and either iv magnesium sulphate 2gm in 50 ml or iv 50ml normal saline over 15-20min. After end of infusion again the patient were nebulised with 0.5ml of salbutamol. Spirometry was performed at 30 min interval (after completion of infusion). Peak flow rates were measured at the same time.

Oxygen was given to the patient by nasal cannula at the rate of 6 liter/min through out the procedure.

Statistical Analysis

Statistical analysis was done by using the statistical package for the social science (SPSS) program in computer. Chi square test and unpaired "t" test are used to compare between two groups.

A 'P' value less than 0.05 is consider as significant.

Results and Observations

This prospective study conducted in National Institute of Chest Disease and Hospital, Dhaka for a period of one year starting from January 2003 to December 2003. The main objective of the study was to examine the therapeutic response of magnesium sulphate in severe bronchial asthma. A total of 60 patients were evaluated. The mean age of the patients were 27.2±5.7 years ranging from 18 to 40 years. Among the studied patients, 21(35.0%) were male and the rest were female 39(65.0%). The mean age of the male patients was 26.1±4.8 years and the female patients was 27.7±6.2 years. Highest percentage of studied patients 21(35.0%) were in the age group 30 years and above, followed by 20(33.3%) in the age range below 25 years and 19(31.7%) in the age group of 25-29 years. Analysis revealed that no statistically significant mean age difference was found between male and female patients (p>0.05), although the mean age was higher among female patients compared to male patients. (Table 1)

Table-I

Age and sex distribution of the study subjects

Age in years	Sex		Total
	Male	Female	
<25	8(38.1)	12(30.8)	20(33.3)
25-29	9(42.9)	10(25.6)	19(31.7)
>30	4(19.0)	17(43.6)	21(35.0)
Total	21(35.0)	39(65.0)	60(100.0)
Mean±SD	26.1+4.g	27.7±6.2 NS	27.2±4.7

N.B
Figure in parenthesis indicate percentage
P value reached from unpaired student's t test (p>0.05)

In this study, to compare the therapeutic efficacy of magnesium sulphate, the studied patients were divided into two groups. Thirty patients were treated by magnesium sulphate with steroid and salbutamol and designated as group I patients and the rest were treated with normal saline with steroid and salbutamol and designated as group II patients. The mean age of the group I patients was 28.4±6.1 years ranging from 18 to 40 years and the mean age of the group II patients was 25.9±5.2 years ranging from 18-35 years. Analysis revealed that no statistically significant mean age difference was found between two groups of patients (p>0.05). (Table II)

Table-II

Age distribution of the study subjects

Age in years	Study subjects		Total
	Group I	Group II	
<25	7(23.3)	13(43.3)	20(33.3)
25-29	11(36.7)	8(26.7)	19(31.7)
>30	12(40.0)	9(30.0)	21(35.0)
Total	30(50.0)	30(50.0)	60(100.0) ..
Mean±SD	28.4±6.1	25.9±5.2^{NS}	27.2±5.7

N.B
Figure in parenthesis indicate percentage
Group I = Magnesium sulphate with steroid and salbutamol
Group II = Normal saline with steroid and salbutamol
p value reached from unpaired student's t test (p>0.05)

Table III shows the sex distribution of the studied subjects. Among group I patients, 10(33.3%) were male and 20(66.7%) were female. Similar pattern of sex distribution was found among group II patients with 11(36.7%) were male and 19(63.3%) were female. No statistically significant sex difference was found between to groups of patients (p> 0. 05).

Table-III

Sex distribution of the study subjects

Sex	Stud y subjects		Total
	Group I	Group II	
Male	10(33.3)	I I (36.7) NS	21 (35.0)
Female	20(66.7)	19(63.3)N^{S}	39(65.0)
Total	30(50.0)	30(50.0)	60(100.0)

N.B
Figure in parenthesis indicate percentage
Group I = Magnesium sulphate with steroid and salbutamol
Group II= Normal saline with steroid and salbutamol
p value reached from chi square test (p>0.05)

It was observed that cent per cent of the attended patients had presented with breathlessness, wheeze and straining in accessory muscle followed by chest tightness (90.0%), cough (85.0%), sweating (1.7%). But no patients presented with cyanosis. No statistically significant difference was found between two groups of patients (p=%0.05) regarding clinical presentation (Table IV). Table V shows the comparative assessment of two treatment modalities. Analysis revealed that in both the groups the significant changes of PEFR and percent predicted PEFR was found (p<0.001), but the percentage of improvement was statistically significant in group I patients (p<0.001).

Table-IV

Clinical presentation of the study subjects

Symptoms	Study subjects		
	Group I	Group II	Total
Breathlessness	30(100.0)	30(100.0)	60(100.0)
Chest tightness	27(90.0)	27(90.0)	54(90.0)
Cough	24(80.0)	27(90.0)	51(85.0)
Wheeze	30(100.0)	30(100.0)	60(100.0)
Accessory muscle use	30(100.0)	30(100.0)	60(100.0)
Sweating	1(3.3)	0(0.0)	1(1.7)
Cyanosis	0(0.0	0(0.0)	0(0.0)

N.B

Figure in parenthesis indicate percentage

Group I = Magnesium sulphate with steroid and salbutamol

Group II = Normal saline with steroid and salbutamol

p value reached from chi-square test (p>0.05)

Table-V

PEFR, L/min and PEFR, % predicted before and after treatment

PEFR, % predicted

	Group I (n=30) Mean±SD	Group II (n=30) Mean±SD	Group I (n=30) Mean±SD	Group II (n=30) Mean±SD
Before	147.1±21.8NS	148.7±22.1	32.7±4.3NS	32.8±4.5
After	188.3±22.7***	171.4±21.6	42.5±4.3***	38.1±3.2
% improvement	28.9+9.8***	15.9±8.7	30.2+5.3***	17.1+7.6

N.B

p value reached from unpaired student's t test

***p<0.001

Table-VI

FEV_1, L and FEV1% predicted before and after treatment

	FEV_1, % predicted			
	Group I (n=30) Mean±SD	Group II (n=30) Mean+SD	Group I (n=30) Mean±SD	Group II (n=30) Mean+SD
Before	0.71±0.12^{NS}	0.73±0.13	22.4±4.2^{NS}	23.0±4.7
After	0.87±0.11***	0.81±0.11	27.7±3.79***	25.6±3.8
% improvement	22.3+5.7***	10.9±4.9	24.9±7.0-***	12.2±6.8

N. B
p value reached from unpaired student's t test ***P<0.001

Table-VII

Percentage of improvement of selected parameters after two treatment modalities

Parameters	Group I	Group II	p value
PEFR, L/min	28.9±9.8	15.9±8.7	0.001
PEFR,% predicted	30.2±5.3	17.1±7.6	0.001
FEV1/L L	22.3±5.7	10.9±5.0	0.001
FEV1, % predicted	24.9±7.0	12.2±6.8	0.001
Pulse /min	3.3±0.3	3.3±0.6	0.710
Respiration	18.6±1.5	15.3±10.1	0.080
Systolic blood pressure (mmHg)	0.4+ 1.5	0.2±0.8	0.463
Diastolic blood pressure (mmHg)	0.0±0.0	0.1+0.7	0.321

* p value reached from unpaired student's t test

Table VI shows the comparative assessment of improvement in two treatment modalities in terms of FEV_1, L and FEV, % predicted. Analysis revealed that in both the groups the significant changes of FEV_1, and FFV_1% predicted was found (p0.001), but the percentage of improvement was statistically significant in group I patients (p<0.001).

Analysis of the above table revealed that the after treatment with magnesium sulphate the percentage of improvement was significantly high in terms of PEFR L/min, PEER predicted, FEV1 L and FEV1% L (p<0.001). But no statistically significant changes were observed in pulse rate, blood pressure and respiration (p>0.05) (Table VII and figure 1).

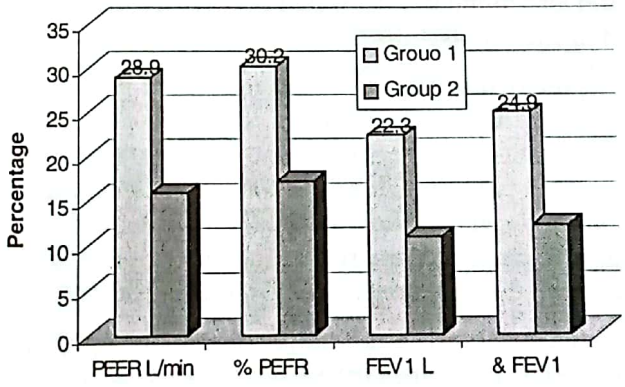

Fig.-1 : *Percentage of improvement of selected parameters after two treatment modalities*

Discussion:

The published literature on adult asthmatics has yielded conflicting results though in children demonstrate magnesium to be beneficial is severe

asthma. A number of case reports indicate magnesium is beneficial for treating acute severe asthma, while results of controlled clinical trials evaluating the effect of magnesium in acutely ill ED patient widely differ[14].

This prospective study conducted in National Institute of Chest Disease and Hospital, Dhaka for a period of one year starting from January 2003 to December 2003.

The studied patients were divided into two groups. Thirty patients were treated by magnesium sulphate with steroid and salbutamol and designated as group 1 patients and the rest were treated with normal steroid with steroid and designated and designated as group 11 patients. Among the studied patients, 21 (35.0%) were male and the rest were female 39 (65.0%). The mean age of the group I patients was 28.4+6.1 years ranging group 18 to 40 years and the mean age of the group II patients was 25.9±5.2 years ranging from 18-35 years. Analysis revealed that no statistically significant mean age difference was found between two groups of patients (p>0.05). It indicate that younger age group are suffering from severe acute asthma more than other age group. The probable causes might be they are more exposure to pollen, dust and other environmental cause.

The mean duration of suffering from asthma was 1 1.0±4.3 years ranging from I-18 years. It indicate that majority were suffered from early childhood and also found that they had positive family history of bronchial asthma.

The mean duration of symptoms before attendance to hospital was 5.2±.2 days. It was 5.4+2.6 days among the group I patients and 5.0±1.8 days among group 11 patients. This duration was little bit higher compare with other study. It could be due to ignorance of patient regarding the disease. They also tried to get treatment from local doctors.

It was observed that cent percent of the attended patients had presented with breathlessness, wheeze and straining in accessory muscle followed by chest tightness (90.0%), cough (85.0%) sweating (1.7%). But no patients presented with cyanosis. .

It was evident that although the cent percent of the patients were conscious, but 16(26.7%) were unable to talk and 44(73.3) were able to talks with words.

In the present study, the pulmonary functions were evaluated by measuring the PEFR L/min, PEFR %predicted, FEV1 L and FEV1 %predicted. Analysis revealed that the predicted PEFR increased significantly from baseline after administering the drugs in two groups of patients (p>0.001). The percent of improvement was significantly higher among the patients treated by conventional drugs adjunct with magnesium sulphate (30.2±5.3%) compared to group 11 patients treated by conventional drugs adjunct with normal saline (17.1±7.6%) (p<0.00 I).

The initial PEFR L/min was 147.1±21.8 L/min in group I patients and 148.7±221 L/min in group II patients after administration of medicine in increased to 188.3±22.7 I,/min in group I patients and 171.4+21.6 L/min in group II patients. Analysis also revealed that mean percent improvement was significantly high in patients treated with magnesium sulphate (28.9±9'%) compared to patients treated with normal saline (15.9±8.7 %) (p<0.001). It was also noted that within group, PEFR was significantly increased from baseline (p<0.001).

It was evident that the FEV_1: L was increased from baseline 0.71±0.12 L in group I and 0.73±0.13 L in group II to 0.87±0.02 L in group I and 0.81±0.10 L group group II patients and the difference was statistically significant (p<0.001) Analysis revealed that the mean percent of improvement was significant high among the group I patients (22.3±5.7%) compared to group II patients (10.4.9%) (p<0.001).

It was evident that no statistically significant mean difference was found between two groups of patients in terms of percentage predicted forced expiratory volume (p>0.05), although it was a bit higher in group II patients (23.0±4.7) than group I patients (22.4±4.2). After administration of drugs, it was increased significantly in both the groups of patients (p-10.001) and the mean difference was statistically significant between two groups of patients. Analysis also found that mean percentage of improvement was significantly high among the group I patients (24.9±7.0) compared to group II patients (12.2±6.8) (p-10.001). .

Ciarallo et al. (2000) [7] carried out a study on - Higher dose intravenous magnesium therapy for children with moderate to severe acute asthma".

They included 30 patients between the age of 6-17.9 years and found remarkable improvement in pulmonary function. The PEFR improved in all the analyzed patients at 20 min ($p < 0.001$) 50 min p- <0.001) and 110 min ($p<0.001$). Comparing the improvement of FEV 1 from the baseline between the 2 groups yielded similar pattern. At 50 mins between the two groups the absolute change in percent predicted PEFR was 2% vs 14% and FEV_1 was 2% vs 15 respectively .

In present study both the PEFR and FEV_1, response were also significant ($p < 0.001$). Absolute change in percent predicted PEFR is 17.1% vs 30.2% and FEV_1, is 12.2% vs 24.9% respectively. Here the change is relatively higher. It may be due to different type of patient selection.

Skobeloff et al. (1989)[15] conducted a study on "Intravenous magnesium sulphate for the treatment of acute asthma in the emergency department". They included 38 patients divided into two groups. They found magnesium infused group showed marked improvement than control group. "File PEFR improvement in all analyzed patients at 20, 30 and 45 mins were significantly different ($p <0.05$), Their result compatible to this study is less significant. Reason might be due to lower dose of magnesium sulphate as well as for the selection of those patients who did not responded to three times nebulisation. Besides this they introduced magnesium sulphate after 90 mins of nebulisation.

Similar type of study carried out by Ciarallo et al. (1996)[2] -"Intravenous magnesium therapy for moderate to severe pediatric asthma". They included 31 patient aged 6 to 18 years and found the magnesium group had significant improvement of FEV_1 % predicted from baseline (34%vs 1.1, $p < 0.05$) at 50 mins and this improvement was sustained even greater att 10 min (75% vs 5%, $p < 0.01$). Result was similar for PEFR at 80 through 110 mins (59 vs 20% at 110 min) In present study PEFR %predicted and FEV1 % predicted response were highly significant (P-< 0.001). Absolute change in PEFR, % predicted was 30.2% vs 17.1% and FEV_1, % predicted was 24.9% vs 12.2%. This difference might be they took different age group of patient and introduced magnesium sulphate after three times beta-2 agonist nebulisation.

Noppen et al. (1990)[16] carried out a study on "Bronchodilating effect of intravenous magnesium sulphate in acute severe bronchial asthma". They included 6 patients aged 45-60 years. They infused magnesium sulphate 0.615 mmol/min for 20 mins. They found marked improvement at 20 min $P<0.05$. But FEV1, fell after 30 mins though it did not reach the baseline value. Addition of beta-agonist after 30 mins causes significant improvement of FEV, in all patients $p<0.05$. Their result was less significant than recent study probably due to administration of two drugs separately at 30 mins interval. Results of their study and present study suggest that magnesium has bronchodilating effects in asthmatsic patient.

Skorodin et al. (1995)[17] conducted a study with "Magnesium sulphate in exacerbation of chronic obstructive pulmonary disease". They included 72 patients of aged 62-66 years and found remarkable improvement in pulmonary function. The PEFR improvement at all of the analyzed patient at 30 and 45 min were (162.3 vs 143, $p<0.03$) and (161.3 vs 143.3, $p<0.03$) respectively. They showed that magnesium sulphate have got not only significant bronchodilating effect, but also rapid improvement, which was similar to present study.

"Magnesium sulfate as a vehicle for nebulised sulbutamol in acute asthma" This work carried out by Nannini et al. (2000)[18] in Argentina. They enrolled 35 patients with acute asthma in randomized double-blind controlled trial. They found isotonic magnesium sulpahate plus sulbutamol increase the peak flow in comparison to sulbutamol plus normal saline. This result is consistent with current study in spite of different route of administration of drug.

Devi et al. (1997)[19] conducted a study - "Intravenous magnesium sulphate in acute severe asthma not responding to conventional therapy". They took 47 children of age between 1-12 years. They found significant improvement in PEFR at 30 mins and 1st, 2nd, 3rd, and 7th hours after stopping the infusion ($p<0.05$-0.01) They could have got more significant effect, if they had taken responder group. Moreover dose of magnesium sulphate was also less than recent study.

Okayama et al. (1987)[20] conducted a study on "Bronchodilating effect of intravenous magnesium

sulphate in acute bronchial asthma". They included 10 patient of 45-62 years and found significant improvement of FEV_1, at 20 min. Which declined and again improved by taking beta-2 agonist similar to initial one. If they had used both drugs simultaneously they could be availed better response. They also found in all patient that dyspnea was decreased after magnesium infusion. Which was similar observation with current study.

Silverman et al. (2002) have done similar type of study on Intravenous magnesium sulphate in the treatment of acute severe asthma. They took 254 patients from different hospitals of 18 to 60 years. They found remarkable improvement in pulmonary function. The FEV_1, improved on patients treated with magnesium sulphate 48.2% vs 43.5% at 240 mins. In current study FEV_1, in among the magnesium sulphate group improved 27.7% vs 25.6% at 50 mins. So, both the studies show improvement in pulmonary function. As their study revealed gradual improvement of all the parameter, so it can be concluded that present study is consistent with their study.

Small sample size is one of the limitations of this study.

In current study blood magnesium sulphate were not estimated before and after the infusion.

Measurement of PEFR L/ min, PEER % predicted, FEV1 L and FEV1 % predicted can be monitored for longer period.

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A Study of The Effects of Chemical Pleurodesis in the Recurrence of Spontaneous Pneumothorax Secondary to COPD

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Abstract

The high recurrence rate of spontaneous pneumothorax secondary to COPD highlights the need for the prevention of recurrence with cheap and costeffective method. Chemical pleurodesis with tetracycline hydrochlorides may be a good option for the prevention of recurrence of pneumothorax and thereby enables satisfactory patient outcome. The study was conducted prospectively in the National Institute of Diseases of the Chest and Hospital (NIDCH) from January 2003 to December 2003 on 60 patients with spontaneous pneumothorax, secondary to COPD. After randomization, 30 patients were treated with tube thoracostomy followed by pleurodesis with tetracycline hydrochloride and another 30 patients of control group were treated with tube thoracostomy alone. Patients were followed up upto 6 months and were looked for recurrence. Patients with spontaneous pneumothorax were of 4'r' to 6th decades of life and most of them were male. Duration of pneumothorax was 66-107 hours in most of the patients. Most patients presented with moderate size of pneumothorax and required 9I-110 hours for lung expansion after tube thoracostomy. Recurrence rate of spontaneous pneumothorax secondary to COPD in the tetracycline group was 3.3%, whereas in control group it was 30%. Intrapleural instillation of tetracycline hydrochloride significantly reduces the recurrence of spontaneous pneumothorax due to COPD. (P=0.015). Morbidity related to tetracycline was negligible. Moreover, tetracycline is cheap, easily available, non-toxic, well tolerated. It is concluded that recurrence rate of spontaneous pneumothorax secondary to COPD can be reduced effectively by chemical pleureidesis with tetracycline hydrochloride without any significant morbidity related to tetracycline hydrochloride and it is also very cost effective.

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Introduction

Pneumothorax is defined as an accumulation of air in the pleura space with secondary lung collapse. This accumulation may come from different sources, but rupture of the visceral pleura with secondary air leak from the lung is the single most common cause[1].

Pneumothoraces can be classified according to their cause and clinical presentation. They can either be spontaneous, traumatic, or iatrogenic, the first category includes both primary and secondary verities. Spontaneous pneumothorax develops annually in 17,000 individuals in the united states[2]. A primary spontaneous pneumothorax happens in individuals with no known pulmonary disease[1]. Secondary Spontaneous pneumothorax may arise from a variety of pulmonary and non-pulmonary disorders.

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Most patients with secondary spontaneous pneumothoraces are male and have a documented or clinically apparent pulmonary disease. Pneumothorax complicating chronic obstructive pulmonary disease is the most common variety of secondary pneumothorax. It occurs in patients older than 50 years of age and is the result of rupture of a bully into the pleural space[r]. Patients with spontaneous pneumothorax secondary to chronic obstructive pulmonary disease present with chest pain and acute sudden respiratory distress with hypoxia, hypercarbia and acidosis.

The diagnosis can be difficult, because the physical findings are those of chronic obstructive pulmonary disease. In most cases diagnosis is made by chest radiographs. Tube thoracostomy is a good option of treatment of spontaneous pneumothorax. Recurrence rate in the secondary spontaneous pneumothorax are high. The ipsilateral recurrence rate for patients with primary spontaneous pneumothorax treated with tube thoracostomy has varied from 23% to 52%. Although less data are available with secondary spontaneous pneumothorax, the recurrence rates seem to be comparable[2].

Pleurodesis, either chemical or surgical, is an effective procedure for prevention of recurrent spontaneous pneumothorax. Pleurodesis should be performed for every COPD patient with pneumothorax in order to prevent recurrence[4]. Many agents have been injected into the pleural cavity through a chest tube to induce pleural adhesion between the parietal and visceral pleurae. Historically silver nitrate guaiacol, hypermnic glucose, iodoform, iodized oil, outlooks blood, cyanoacrylate tissue adhesive and talcum have been used. Now a days tetracycline hydrochloride has been advocated.[5] It is believed that the local irritating effect of the drug is related to it's low pH which causes sterile inflammation with subsequent adhesion of visceral pleura to parietal pleura and obliteration of pleural space[r] Tetracycline hydrochloride is chosen as the sclerosing agent because it seems to be the most effective sclerosing agent because it is widely available and because it is devoid of significant systemic toxic effects when used inUapleuraly[2].

Spontaneous pneumothorax is a major Public health problem in the world. Despite significant social impact of spontaneous pneumothorax few epidemiologic data are available for the study of spontaneous pneumothorax using intrapleural tetracycline hydrochloride in developing countries including Bangladesh. So, it is the demand of the time to perform study in this particular subject. The present study was carried out (i) To study the outcome of chemical pleurodesis with tetracycline hydrochloride in the recurrence of spontaneous pneumothorax secondary to COPD. (ii) To compare the recurrence rate of spontaneous pneumothorax treated with chemical Pleurodesis with tetracycline hydrochloride and tube thoracostomy alone. (iii) To make a standard protocol for management.

Materials and Methods

A prospective randomized case control study was carried out in the dept. of thoracic surgery of the National Institute of Diseases of the Chest and Hospital (NIDCH), Dhaka, Bangladesh during the period January 2003 to December 2003. A total number of 60 patients had been taken in this study. These patients were divided into 2 groups randomly. In group A, 30 patients were treated with tube thoracostomy followed by chemical pleurodesis after lung expansion and in group B, 30 patients were treated with tube thoracostomy only.

Patient selection criteria

Inclusion criteria

Patients having spontaneous pneumothorax secondary to COPD admitted in NIDCH. Mohakhali. Dhaka, during the study period. The diagnosis of spontaneous pneumothroax secondary to COPD was based upon history, clinical findings, chest x-ray and lung function test. Lung function test was done after tube thoracostomy and when the patients condition was improved and became stable.

Exclusion criteria :

- Spontaneous pneumothorax secondary to COPD with broncho-pleural fistula.
- Secondary spontaneous pneumothorax due to COPD requiring thoracotomy.
- Primary spontaneous pneumothorax.
- Secondary spontaneous pneumothorcix due to COPD with failure of lung expansion after tube thoracostomy.
- Hypersensitivity to tetracycline.

Patients with spontaneous pneumothorax, who were hospitalized and required tube thorcostomy and met no exclusion criteria were eligible for the study. After evaluation, tube thoracoscotmy was done under aseptic condition. A 32 French size tube was inserted through the 4-6th intercostal space along the anterior axillary line under local anaesthesia, into the pleural space depending upon the side of the pneumothorax and the tube was connected with underwater seal drainage system. After tube thoracostomy, evaluation of the patients was done both clinically and radiologically. First x-ray was done after 24 hours to see the re-expansion of the lung. Further evaluation of the patients was done clinically and by chest x-ray. When the lung was expanded, the patients were randomized to the tetracycline or the control group.

The following treatment protocol was followed for individuals assigned to the tetracycline group:

20 ml of 1% Inj. lignocaine hydrochloride was introduced through the chest tube into pleural space. tube was clamped for 15 minutes. 2 gm of Tetracycline hydrochloride introduced through chest tube, 20m1 of normal saline was introduced to flash the tube, the tube was then clamped for 6 hours. Patient was told to change posture hourly. After 6 hours tube was declamped and allowed drainage. Patients in both tetracycline and control groups were observed carefully for 24 hours period and any complication related to tetracycline were recorded. Tube was removed usually after 24 hours both in the tetracycline and control groups and when the drainage was less than 50mI in 24 hours.

The following outcomes were measured: Age and sex distribution of the patient, height and weight of the patient, duration of pneumothorax, side of pneumothorax, size of pneumothorax, time of lung expansion after tube thoracostomy, lung function test, duration of hospitalization, recurrence of pneumothorax, side effects after tetracycline instillation, mortality, patients were followed up upto G months after hospital discharge and were looked for- recurrence of pneumothorax

All the relevant collected data were compiled on a master chart. Data analysis were done by using computer software devised with statistical package for social sciences (SPSS). Baseline characteristics between group A and group B were compared by chi-square test. Treatment comparisons for recurrence were made using chi square test. Lung function tests were compared using unpaired t-test., The P-value of less than 0.05 was considered significant.

Results

Data were prospectively collected on 30 patients assigned to the tetracycline group and 30 patients assigned to the control group. The patient group comprised of 29 male (96.7%) and 1 female (3.3%) in each group with male female ratio was 29:1. Patients age ranged from 40 years to 70 years in each group with mean 54.8±7.8 years in group A and 56.2±9.2 years in group B, which were similar and did not differ significantly. Distribution and comparison of age of the patients are shown in table-I. All the patients in each groups were in fourth to sixth decades of life, no patients were below forth decade.

Table-I

Distribution of age of patients

Age in years	Group A (No. of patients)	Group B (No. of patients)
40-50	11(36.7)	12(40.0)
51-60	12(40.0)	10(33.3)
61-70	7(23.3)	8(26.7)
Grand total	30(100)	30(100)
Man±SD	54.8±7.8	56.2±9.2

Figures in parenthesis indicate percentage. Chi-square = 6.9, df =2.. P>6.05 (Not significant)

Patients heights ranges from 5 feet to 6 feet 5 inches in each group with mean 5.6±0.14 feet in group A and 5.6±0.17 feet in group B respectively. Most patients i.e. 70% in group A and 80% in group B however, were in the range of 5 feet G inches to G feet 5 inches. These two groups were similar and did not differ significantly. Distribution of heights and their comparison between two groups were shown in table-II.

Table-II
Distribution of heights of patients

Hight in feet/ /inches	Group A (No. of patients)	Group B (No. of patients)
5'- 5'.5"	9(30.0)	G(20.0)
5'.6" – 6'.5"	21(70.0)	24(80.0)
Grand total	30(100)	30(100)
Mean±SD	5.6±0.14	5.6 to .17

Figures in parenthesis indicate percentage.
Chi-square = 0.800, df=1, P> 0.05 (Not significant)

Patients weight ranges from 40 to GO kg in each group with mean 50.8±5.6kg in group A and 31.1±4.4 kg in group B. But most patients weights were in the range of 46 to 50 Kg, i.e. 46.7%. in group A anil 40% in group B. Distribution of weights and comparison between two groups are shown in table -III.

Table-III
Distribution of weight of patients

Weight in kg	Group A (No. of patients)	Group B (No. of patients)
40-45	4(13.3)	3(10.0)
40-50	14(4G.7)	12(40.0)
51-55	6(20.0)	7(23.3)
56-60	6(20.0)	8(26.7)
Grand total	30(100)	30(100)
Mean±SD	50.8±5.6	51.1±4.4

Figures in parenthesis indicate percentage.
Chi-squire = 0.659. dl = 3. P>0.05 (Not significant)

Duration of pneumothorax

Duration of pneumothorax ranged from 45 to 170 hours in each group with mean 91.5±44.'2 hours in group A and 97.3±33.4 hours in group B respectively. Duration of pneumothorax of most patients in each group were from 66 to 107 hours (Shown in table IV).

Table-IV
Duration of pneumothorax

Hours	Group A (No. of patients)	Group B (No. of patients)
45-65	6(20.0)	4(13.3)
66-86	10(33.3)	10(33.3)
87-107	7(23.3)	9(30.0)
108-128	1(3.3)	2(6.7)
129-149	2(6.7)	2(6.7)
150-170	4(13.3)	3(10.0)
Grand total	30(100)	30(100)
Mean±SD	95.8±44.2	97.3±3 3.4

Figures in parenthesis indicate percentage.
Chi-square = 1.12G, d(= 5, P>0.05 (Not significant)

Side of pneumothorax

60% of patients in group A had right sided pneumothorax and 40% had left sided pncumoihoiax, on the other hand half of the patients in group B had right sided and half had left sided pneumothorax shown in table -V.

Table-V
Side of pneumothorax

Side	Group A (No. of patients)	Group B (No. of patients)
Right	18(60.0)	15(50.0)
Left	12(40.0)	15(50.0)
Grand total	30(100)	30(100)

Figures in parenthesis indicate percentage, Chi-square = 0.606. df = t. P>0.05 (Not significant)

Percentage of lung collapse

Percentage of lung collapse ranged from IS to 45% in each group with mean (29.5 ±5.3) in group A and (29.0±4.5) in group B respectively. But most patients i.e. 93.3% in group A and 9G.7% in group B ranged from 21 to 40%. Shown in table -VI and in photograph-1

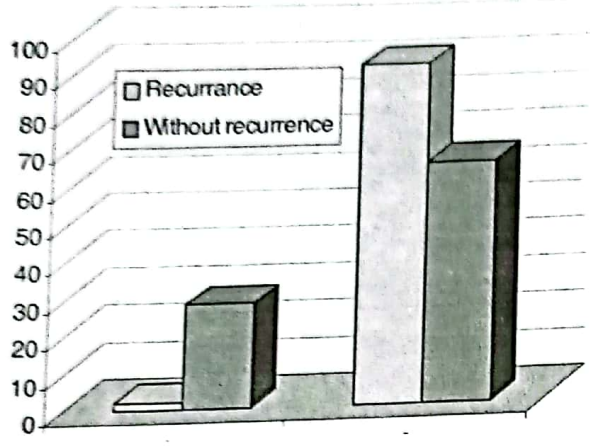

Fig-1: *Bar diagram showing comparison of recurrence rates of spontaneous pneumothorax between two groups*

Table-VI

Percentage of lung collapse

Percentage	Group A (No. of patients)	Group B (No. of patients)
<20	1(3.3)	1(3.3)
21-40	28(93.3)	29(96.7)
40+	1(3.3)	0(0.0)
Grand total .	30(100)	30(I00)
Mean±SD	29.i±5.3	29.0±4.5

Figures in parenthesis indicate percentage.
Chi-square = 1.018.
df= 2, 1'>0.05 (Not significant)

Time for lung expansion after tube thoracostomy

Time for lung expansion after tube thoracostomy ranges from 50 to 144 hours in group A and from and from 50 to 120 hours in group B with mean time for expansion 90.6 ± 17.3 hours for group A and 93.3±15.3 hours for group B. This time ranged from 01 to 110 hours for 50% patients of group A and 53.3% of patients of group B. Shown in table-VII.

Table-VII

Time for lung expansion after tube thoracostomy

Hours	Group A No. of patients	Group B No. of patients
50-70	1(3.3)	1(3.3)
71-90	12(40.0)	10(33.3)
91- 110	15(50.0)	16(53.3)
111-144	2(6.7)	3(10.0)
Grand total	30(100)	30(100)
Mean±SD	90.6±17.3	93.3±15.3

Figures in parenthesis indicate percentage.
Chi-squire = 0.411, dl = 3, F'>0.05 (Not significant)

Recurrence rates of spontaneous pneumothorax

The recurrence rate was 3.3% in the tetracycline group and 30% in the control group (P=0.015). The intrapleural administration of tetracycline resulted in a significant decrease in the ipsilateral recurrence rate of spontaneous pneumothorax. Shown in table -VIII and Fig.-1

Table -VIII

Recurrence rates of spontaneous pneumothorax

Group of Patients	Number of patients having recurrence	% Recurrence	Number of patients without recurrence	% without recurrence
Tetracycline group	1	13	29	96.7**
Control group	9	30	21	70

Chi-square = 5.88. df= 1. ** P<0.01 (Significant)

Recurrence rates of spontaneous pneumothorax at different intervals after randomization

A11 recurrences (3.3%) in the tetracycline group were in the first month after randomization, and in the control group 16.6% recurrence were in the first month. In the control group 10% recurrence was between 2-3 months. and 3.3% recurrence between 4-6 months respectively. Shown in table-IX and fig 2.

Table-IX

Recurrence rates of spontaneous pneumothorax at different intervals after randomization

Time in months	Group A	Group B
	No. of patients	No. of patients
<1	1(3.3)	5(16.G)
2-3	0	3(10)
4-6	0	1(3.3)

Figures in parenthesis indicate percentage

Time in months

Fig-2: Recurrence rates of spontaneous pneumothorax at different intervals after randomization

Discussion

There have been few publications in recent decades relating to the role of chemical pleurodesis in the management of spontaneous pneumothorax secondary to COPD. This study, which was conducted in the National Institute of the Diseases of the Chest and Hospital, the only referral hospital in Bangladesh, shows that chemical pleurodesis using tetracycline hydrochloride plays a substantial role in the prevention of recurrence of spontaneous pneumothorax secondary to COPD. This study is particularly designed to study the outcome of chemical pleurodesis with tetracycline hydrochloride in the recurrence of spontaneous pneumothorax, to compare the recurrence rate of spontaneous pneumothorax treated with chemical pleurodesis by tetracycline hydrochloride and tube thoracostomy alone.

In this study patients with spontaneous pneumothorax secondary to COPD are of 4th to 6th decade, of life with mean age 54.8 ± 7.8 years for group A and 56.2 ± 9.2 years for group B (p=0.892). Age incidence of secondary spontaneous pneumothorax due to COPD is consistent with the study of Light and his colleagues[2]. This shows that secondary spontaneous pneumothorax due to COPD occurs commonly after 4th decades. Most of the patients in this series were male (9G.7%) and all were smoker showing strong likelihood of association of smoking with COPD and pneumothorax. Sex distribution and smoking history is also consistent with previous study of DeVries et al. (1980) and Limthongkul et al. (1992) and smoking history is almost similar with the study of Light et al. 19902·4·5

Patients with spontaneous pneumothorax having COPD are usually tall (mean height 5.6 ± 0.14 feet for group A and 5.6 ± 0.17 feet for group B (P=0.371) and underweight (mean weight 50.8 ± 5.6 kg for group A and 51.1 ± 4.4 for group B (p=0.883) causing increasing height to weight ratio predisposing pneumothorax.

In this study mean duration of pneumothorax was 95.8±44.2 hours in group A and 97.3 ± 33.4 hours in group B (p=0.952). This duration of pneumothorax in this study seems to be prolonged and may be due to the fact that, NIDCH is the only referral chest disease hospital in Bangladesh. So patients from remote areas loose their time from being referred from one hospital to another before reaching to NIDCH.

Right sided pneumothorax in the study group was 60% and left sided 40% and in control group 50% each side (p=0.936). In the previous study by Light et al. (1990) left sided pneumothorax was 52.2% in study group and 53.5% in control group[2]. But no significant differences were noted between two groups. In previous study of Getz et al. (1953) a slight predilection for right side was noted (54%) compared with left side. It is consistent with present study[6].

Most patients had moderate size of the pneumothorax with mean (29.5±5.3) % for group A and (29.0±4.5)% for group B(p=0.601). This is consistent with the previous study of Limpthongkul and associates which was 29.3±23.3 percent of hemithorax[5], but differs from previous study by Light and his colleagues. Their result were 54.0 ± 25.1 percent for group A and 57.6 ± 27.6 percent for group B respectively i.e. most of their patients had large size pneumothorax[2]. This is because they

included patients with primary spontaneous pneumothorax and secondary spontaneous pneumothorax due to pneumonia and asthma along with COPD.

Time required for lung expansion between two groups are identical (Group A- m±SD=90.6±17.3. group B- m±SD=93.3±15.3 hours, p=0.937). Most of the patients required 91-1 10 hours for lung expansion alter tube thoracostomy. Which is almost similar to previous study (median time 120 hours) of Limthongukul and associates[4].

The main adverse effect associated with tetracycline instillation was chest pain only in 2 patients in spite of intrapleural administration of lignocaine; and slight rise of temperature in 4 patients that subsided in the next day. This is much less compared with previous study of Light et al., (1990). The pain and rise (it temperature did not alter the general welbeing of the patients.

In this study intrapleural instillation of tetracycline hydrochloride resulted in a significant (P=0.01) decrease in the rate of recurrencé of spontaneous pneumothorax secondary to COPD which is 3.3% for group A but 30% for croup B. Most of the recurrences happened within 6 months of initial episode.

Present study shows that the recurrence rate following instillation of tetracycline is 3.3%, whereas it was 25% in previous study of Light et al. (1990). On the otherhand recurrence rate in tube only group in this study is 30%, whereas it was 41% in previous study of Light and his associates[2]. Both in this study and in the previous study tetracycline hydrochloride significantly reduced the recurrence rate of spontaneous pneumothorax, but the recurrence rates of the previous study are higher than the present study possibly because of their long follow up period and use of tetracycline at lower dose[2].

In a previous study it was shown that recurrence rate was 13% after tetracycline instillation compared with 36% in patients with chest tube drainage alone[1]. Differences in these recurrence rates were not statistically significant because they used tetracycline at lower dose (500mg). Present study showed much lower recurrence rate (3.3%) of spontaneous pneumothorax after tetracycline instillation. Tetracycline hydrochloride at a dose of 35ma/kg was more effective than at a dose of 7 or 20 mg/kg.

As intrapleural tetracycline decreased the recurrence rate in the present study, it should be considered the agent of choice in attempts to create a chemical pleurodesis. Previous reports suggests that aerosolized talc, dissolved talc, quinacrine or silver nitrate are effective in preventing recurrence of pneumothorax. Until further study documents that one of the other agents is superior to tetracycline, we believe that it should he considered the agent of choice for the following reasons- aerosolized talc is more difficult to administer, intrapleural instillation of dissolved talc has been associated with the development of acute respiratory distress syndrome, quinacrine is no longer available, administration of silver nitrate is even more painful than tetracycline, and lastly, tetracycline is cost effective, easily available in our country and relatively nontoxic[1,2].

Conclusion

The recurrence rate of spontaneous pneumothorax secondary to COPD highlights the need for prevention of recurrence with cheap and cost-effective method. Chemical pleurodesis with tetracycline hydrochloride is a good option for the prevention of recurrence of pneumothorax. This prospective study was conducted in the NIDCH from January 2003 to December 2003 on GO patients with spontaneous pneumothorax secondary to COPD. After randomization 60 patients were treated with tube thoracostomy followed by pleeurodesis with tetracycline hydrochloride and another 30 patients of control group were treated with tube thoracostomy alone. Patients were followed up upto 6 months for recurrence. Recurrence rate of spontaneous pneumothorax secondary to COPD in the tetracycline group was 3.3, whereas in control group it was 30%. Chemical pleurodesis with tetracycline hydrocloride significantly reduces the recurrence of spontaneous pneumothorax due to COPD (P=0.015). Morbidity related to tetracycline was negligible, moreover tetracycline is cheap, easily available, non-toxic, well tolerated. From this study we conclude and recommend that in Bangladesh including other developing countries, where treatment facilities are minimum, recurrence rate of spontaneous pneumothorax

secondary to COPD can be reduced effectively by chemical pleurodesis with tetracycline hydrochloride without any significant morbidity and it is also very cost effective.

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Clinical Presentation and Risk Factors of Acute Myocardial Infarction

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Abstract:

This prospective study was carried out in the coronary care unit, department of cardiology in National Institute of Cardiovascular Diseases, Dhaka during the period of June 2003 to November 2003. 200 patients of anterior myocardial infarction were enrolled with some inclusion and exclusion criteria to evaluate the clinical presentation and risk factors of myocardial infraction. The age range of the study patients was 29 to 68 years with mean age of 49.2 ± 11.3 years. The mean age of the male patients was 47.9 ± 10.8 years and it was 57.3 ± 11.3 years for female patients. The highest number of patients (66%) was in the age group from 40 to 59 years. The male and female ratio was 6.69:1. It was evident that commonest risk factor was smoking 64 % followed by hypertensive 40%, diabetic 24%, dyslipidaemic 15 % and family history of IHD present in 11% patients. Commonest presenting symptoms were chest pain 97.0%, followed by shortness of breath 29. 0%, sweating 20.0% and vomiting 14.0%. In this study 27 percent patients had history of previous angina. The mean pulse rate per minutes, systolic blood pressure and diastolic blood pressure in mmHg were 84.92 ± 16.60 per min, 128.75 ± 19.80 mmHg and 86.10 ± 14.87 mmHg respectively. On admission > Killip class-11 heart failure was found in 15.0% patients. Majority patients, 76 % comes within 360 minutes and majority patient 93 % received thrombolytic therapy with streptokinase.

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Introduction:

Ischemic heart disease is the commonest form of heart disease and the single most important cause of premature death in the developed world'. Approximately 800,000 people in the United States experience AMI annually, of these about 213,000 die. Of those who die, approximately one-half do so within 1 h of the onset of symptoms, before reaching a hospital[2,3]. The major cause of myocardial infarction is atherosclerotic disease of the epicardial coromary arteries. Ischemic heart disease becomes an important health hazard in the third world countries including Bangladesh. In 1996, the prevalence of ischemic heart disease in urban population was found about 10 percent[4]. In 1998, one study showed prevalence of ischemic heart disease was 8.14 percent [5].

Anterior myocardial infarction was highest location in the left ventricular infarction. Brown et al. and Zaher et al[7]. found acute anterior MI in 52% and 53.12% cases respectively. Anterior wall infarction is usually larger than inferior and lateral wall infarction and has a substantially worse prognosis[8,9]. Hospital mortality after AMI is also predicted by early evidence of haemodynamic instability[7].

Approximately one third of patients with AMI do not present with classic chest pain. Non-diagnostic ECGs are recorded in approximately half of patients presenting to emergency departments with chest pain suggestive of MI who ultimately are shown to have an AMI. Among patients admitted to the hospital with a chest pain syndrome, fewer than 20 percent are subsequently

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diagnosed as having had an AMI. Therefore, in the majority of patients, clinicians must obtain serum cardiac marker measurements at periodic intervals to either establish or exclude the diagnosis of AMI, such measurements may also be useful for a rough quantitation of the size of infarction. The use of more sensitive biomarkers of AMI has necessitated establishing the diagnosis of AMI. European society of cardiology and American College of Cardiology adapted a new definition of MI[11]. According to the new definition of myocardial infarction, either one of the following criteria needs for the diagnosis of an acute, evolving or recent MI.

1. Typical rise and gradual fall (troponin) or more rapid rise and fall (CK-MB) of biochemical markers of myocardial necrosis with at least one of the following:
   a. Ischemic symptoms
   b. Development of pathological Q-wave on the ECG
   c. ECG changes indicative of ischemia (ST-segment elevation or depression) or
   d. Coronary artery intervention (e.g. coronary angioplasty) 2.Pathological findings of an acute MI[11].

Materials and methods:

This prospective study was done in the coronary care unit, department of Cardiology, National Institute of Cardiovascular Disease (NICVD), Dhaka, Bangladesh during the period of June 2003 to November 2003. During this period 200 patients of acute anterior myocardial infarction between the aged of 29 to 68 years of both sexes were selected for this study.

In this study, ST-segment elevated myocardial infarction defined as new or presumed new ST segment elevation of the j-point in two or more contiguous leads with the cutoff points > 0.2mV in leads V_1, V_2 or V_3 and $\geq$ 0.1 mV in other leads in appropriate clinical setting". Anterior myocardial infarction defined as characteristic ECG change > 2 adjacent leads from V, to V6 with or without I and aVL[2,13].

Results:

The mean age of the study patients was 49.2 ± 11.3 years ranging from 29 to 68 yeas. The highest number of patients (66%) was in the age group from 40 to 59 years. The mean age of the male patients was 47.9±10.8 years ranging from 29 to 65 years and it was 57.3±11.3 years for female patients with ranging from 38 to 68 years. Out of 200 patients, 174(87.0%) were male and 26(13.0%) were female and the male and female ratio was 6.69:1.

Table-I shows the percentage distribution of major risk factors associated with cardiovascular diseases of the study subjects. Out of 200 patients, 128(64%) were smoker followed by hypertension 80(40.0%), diabetes mellitus 48(24.0)%, dyslipidaemia 30 (15.0%) and family history of ischemic heart disease 22(11.0%). The mean ±SD of composite risk factors were 1.56± 0.9.

Table-I

Risk factors of the patients

Risk factors	No of patients N=200	Percent
Smoking	128	64.0
Hypertension	80	40.0
Diabetes mellitus	48	24.0
Dyslipidaemia	30	15.0
Family history of IHD	22	11.0
Composite risk factors (Mean±SD)	1.56±0.9	

Table-II shows the percentage distribution of presenting symptoms of the study subjects. Out of 200 patients, 194 (97.0%) presented with chest pain followed by shortness of breath 58 (29.0%), sweating 40(20.0%) and vomiting 28(14.0%).

Table-II

Presenting symptoms of the study subjects

Presenting Symptoms	No of patients (N=200)	Percent
Chest Pain	194	97.0
Shortness of breath	58	29.0
Sweating	40	20.0
Vomiting	28	14.0
Palpitation	0	0.0

It was found that out of 200 patients, 54(27.0%) had history of previous angina and 146(73.0%) did not. The mean time of onset of symptoms to admission in hospital was 287.1 with standard deviation of 152.9 minutes. CK-MB was estimated at admission and at 6 hours after admission. The mean±SD CK-MB at admission and at 6 hours were 58.47±24.0 U/L and 73.10±22.6 U/L respectively. 93% of patients of this study were received thrombolytic therapy with streptokinase. The mean pulse rate per minutes, systolic blood pressure and diastolic blood pressure in mmHg of the study patients were 84.92 ± 16.60 per min, 128.75 ± 19.80 mmHg and 86.10 ± 14.87 mmHg respectively. These parameters show in table-III.

Table –III

Heamodynamics of the study subjects on admission

Parameters	Mean ±SD
Pulse/minute	84.92± 16.60
Systolic BP (mmHg)	128.75± 19.80
Diastolic BP (mmHg)	86.10±14.87

Table-IV shows the heart failure in Killip Class among the study subjects. On admission > Killip class-II heart failure was found in 30 (15.0%) patients.

Table –IV

Heart failure of the patients on admission

Heart Failure (Killip class)	No of patients (N=200)	Percent
Class- I	170	85.0
Class- II	28	14.0
Class- III	2	1.0
Class- IV	0	0.0
Total	200	100.0

Table-V shows the arrhythmia of the study subjects. Among the patients, 78(39.0%) developed arrhythmia. The most frequent arrhythmia was PVC 50(25.0%) followed by VT 14(7.0%), VF 10(5.0%), SVT 2(1.0%) and AF 2(1.0%).

Table- V

Arrhythmia of the study patients on admission

Arrhythmia	No of patients (N=200)	Percent
None	122	61.0
PVC	50	25.0
VT	14	7.0
VF	10	5.0
SVT	2	1.0
AF	2	1.0

Table-VI shows the heart block of the study subjects. 25(12.5%) cases developed heart block. The most frequent heart block was RBBB 10(5.0%) followed by 3rd degree AV block 8(4.0%), 2nd degree AV block 4(2.0%) and 1st degree AV block 3(1.5%).

Table- VI

Heart block of the study patients on admission

Heart block	No of patients (N=200)	Percent
Absent	175	87.5
1st degree AV block	3	1.5
2nd degree AV block	4	2.0
3rd degree AV block	8	4.0
RBBB	10	5.0
Total	200	100.0

Discussion:

A total of 200 patients with acute anterior myocardial infarction admitted in Coronary Care Unit, National Institute of Cardiovascular Diseases, Dhaka were included in this prospective study by some inclusion and exclusion criteria.

Increasing age is associated with higher incidence of atherosclerotic coronary artery disease. The age range of the study patients was 29 to 68 years with mean age of 49.2 ± 11.3 years. Similar patters of age group were reported by several studies[14, 15,16]. Age is a strong and independent risk factor for CHD, increases markedly with age up to an age of about 65[17].

It was observed that 87 percent of the study patients were male. Male and female ratio was 6.69:1. In

the previous studies: Amanullah M et al[18]. found 89 percent, Begum F[19] 90 percent and Rahman MM et al[15]. reported 92 percent were male, these are also consistent with the present study. Estrogen may be the most obvious factor responsible for the protection against CHD in cases of female[20]. Coronary lesions appeared to progress faster in males than in females, evident by the presence of much more advanced plaque in 30 to 34 year old men compared with age-matched women[21].

The percent study attempted to assess the risk factors associated with ischemic heart disease. It was evident that 64 percent patients were smoker followed by hypertension 40 percent, diabetes mellitus 24 percent, dyslipidaemia 15 percent and family history of IHD 11 percent. Similar patterns of risk factors observed by Sayemi A 22. Studies done by Rahman MM et al[15], Haque MT[14] also reported comparable data.

The classic symptoms of AMI is chest discomfort that is commonly retrosternal or precordial in location and is described as constricting, crushing, oppressing, compressing, sensation of a heavy weight, squeezing in the chest, choking, viselike or heavy pain, it may also be characterized as a stabbing knifelike, boring or burning discomfort[2]. Other common symptoms of AMI are dyspnea, diaphoresis, nausea and vomiting. Nausea and vomiting occur in more than 50 percent of patients with transmural MI. These symptoms occur more commonly in patients with inferior MI than in those with anterior MI [23]. In this study 97 percent patients presented with chest pain followed by shortness of breath 29 percent, sweating 20 percent, and vomiting 14 percent.

It is not uncommon for patients to experience brief episodes of ischemia before an acute myocardial infarction. This preinfarction angina preconditions the heart; it may confer protection at the time of infarction[24]. Patients with the history of angina preceding myocardial infarction are more likely to have multivessel coronary artery disease[25]. In this study 27 percent patients had history of previous angina.

The mean systolic blood pressure and diastolic blood pressure in mmHg of the study patients were 128.75 ± 19.80 mmHg and 86.10 ± 14.87 mmHg respectively. In most cases with transmural infarction systolic pressure decrease approximately 10 to 15 mmHg from the pre-infarction state[26]. Heart failure was found in 30 (15.0%) patients, consistent with study done by Haque (17%)[20]. 39.0% patients developed arrhythmia. The most frequent arrhythmia was PVC followed by VT, VF. The most frequent heart block was RBBB followed by 3rd degree AV block, 2nd degree AV block and 1st degree AV block.

Conclusion:

This study attempted to evaluate the clinical presentation and risk factor of patient with acute anterior myocardial infarction. In this study acute anterior myocardial infarction commonly encountered in male of forth and fifth decade. Commonest risk factors were smoking, hypertension and diabetes. Dyslipidaemia and family history of IHD were less common risk factors. Commonest presenting symptoms were chest pain, followed by shortness of breath, sweating and vomiting. The most frequent arrhythmia was PVC and heart block was RBBB.

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Role of Tranexamic Acid Nebulization in the Management of Haemoptysis- Experience at NIDC&H

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Abstract:

This study was done to determine the effectiveness of Tranexamic Acid Nebulization in the management of haemoptysis. This prospective randomized case control study was carried out iron January 2002 to December 2002 in the Department of Respiratory Medicine, NIDCH, Dhaka. 50 patients (Male 48, Female 2) admitted to NIDCH with haemoptysis were enrolled in this study. Alternatively, 25 patients were enrolled in group A and in group B respectively. Group B was taken as control group. Group A (n = 25) were treated with Tranexamic Acid Nebulization along with other drugs (Anti TB / Antibiotics / Chemotherapy, Diazepam etc.) and Group B (n = 25) received normal saline Nebulization along with other drugs (Anti TB / Antibiotics / Chemotherapy, Diazepam etc.). In group A, haemoptysis was completely controlled in 3 (12%) case on the 2nd day, 13 (52%) cases on the 3rd day, 4 (16%) cases on the 4th day, 1 (4%) case on the 5th day of Nebulization of Tranexamic Acid. In 4 (16%) cases haemoptysis was not controlled rather deteriorated. In group B, haemoptysis was controlled in only 5 (20%) cases on the 10th days of treatment and in other cases haemoptysis was not stopped within 10 days of treatment.

So, Tranexamic Acid can be used safely in Nebulized form in the management of mild to moderate haemoptysis.

Key Words : *Tranexamic Acid. Nebulization, Haemoptysis*

[Chest & Heart Journal 2005; 29(2) : 113-116]

Introduction:

Haemoptysis is the expectoration of blood that originates below the vocal cord. It is a cause of great anguish to the patient and his/her family and a daunting clinical challenge for the physician. It is commonly classified as trivial or mild, moderate and grave or massive, the last defined as more than 200-600 ml in 24 hours. The dividing line is arbitrary since the amount of blood is rarely quantified with perception[1]. Coughing up blood irrespective of the amount is an alarming symptom and almost always brings the patient to the doctor. A clear history should be taken to establish whether it is true haemoptysis and not haematemesis or epistaxis (Nose bleed). Haemoptysis must always be assumed to have a serious cause until appropriate investigations have proved otherwise. The common causes of haemoptysis are pulmonary tuberculosis, Bronchiectasis, Lung Abscess, Lung Cancer , Acute

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pulmonary infarction Left ventricular failure (LVF), Mitral stenosis. Many episodes of haemoptysis are unexplained even after full investigation and are likely to be caused by simple bronchial infection. A history of repeated small haemoptysis or blood streaking of sputum is highly suggestive of bronchial carcinoma. Chronic fever and weight loss may suggest tuberculosis. Pnpneumonia is often the cause of rusty colored sputum but can cause frank haemoptysis as can all the pneumonic infections which lead to suppuration and lung abscess formation Bronchiectasis can cause catastrophic bronchial bleeding[2] Tranexamic acid is an anti fibrinolytic agent, which competitively inhibits the activation of plasminogen to plasmin. Tranexamic acid is usually used systematically but can be used topically for control of bleeding.

Materials and Methods

This was a prospective case control randomized study carried out from January 2002 to December 2002. Fifty patients of either sex presenting with haemoptysis admitted to NIDCH were included in this study. After proper diagnosis and ruling out the exclusion criteria, the patients were alternately divided into two groups .

Inclusion criteria: Patients having any grade of haemoptysis and those patients who gave consent were included in this study.

Exclusion criteria: Patients of extreme of ages and having life- threatening conditions or shock were excluded from the study. Patients having bleeding disorders and poor co-operation were also excluded from the study. Initially 60 patients were enrolled in this study. Later on 10 patients dropped out. Finally, 50 patients completed the study. there were 48 males and 2 females. The mean age was 35.7 (SD ± 24.9) years with age range of f8-65 years. The patients were divided alternatively into two groups 25 in group A (case) and :25 in group B (control). Group A (n=25) received Tranexamic Acid Nebulization (Tranexamic Acid 25cc. Plus Normal Saline 2cc) 8 hourly. On the other hand, group 13 (n=25) was nebulized with only Normal saline 2cc 8 hourly. Both groups received other definitive treatment and were observed daily.

Study Procedure:

- Informed Consent obtained
- Detailed history taken
- Thorough physical examination done
- Necessary investigations performed to rule out exclusion criteria.
- Questionnaire filled up.
- Group A (n=25) treated with Tranexamic acid Nebulization along with other drugs (Anti-TB/ Antibiotic / Chemotherapy, Diazepam etc.)
- Group B (n=25) received Normal Saline nebulization along with other drugs (Anti-TB/ Antibiotic / Chemotherapy, Diazepam etc.)

Results

All cases were evaluated for diagnosis. Out of 50 patients, 30 had pulmonary tuberculosis Bronchiectasis- 13, Lung Abscess- 4, Lung cancer - 2 and Mitral stenosis- 1 (Table 1). Majority of the patients belonged to 31-40 year age group. Out of 50 cases, 32 had mild haemoptysis. 13 had moderate haemoptysis and 5 massive haemoptysis (Table 2). Group A (n= 25) were treated with Tranexamic Acid Nebulization along with other drugs (Anti-TB/ Antibiotics/ Chemotherapy, Diazepam etc.) and group B received normal saline, Nebulization along with other drugs (Anti-TB/ Antibiotics / Chemotherapy, Diazepam etc.) In group A, (n=25) haemoptysis was controlled after 10 days of treatment in 21 (84%) cases and not controlled in 4 (16%) cases, on the other-hand in group B (n=25) haemoptysis was controlled in 5 (20%) cases and not controlled in 20 (80%) cases (Table 3). In group A, haemoptysis was completely controlled in 3 (12%) cases on 2"d day, 13(52%) on 3'd day, 4 (16%) cases on the 4t" day, 1 (4%) case on the 5th day of Nebulization of Tranexamic Acid. In 4 (16%) patients, haemoptysis was not controlled rather deteriorated. In group B. haemoptysis was controlled in only 5 (20%) cases on the 10'h day of treatment and in other 20 (80%) cases haemoptysis was not stopped within 10 days of treatment (Fig. 1). When group A was compared with group B and Chi-square test was done, the p-value was found to be <0.001 which was highly significant.

Table-I

Diagnosis of Patients n=50

Diagnosis (n=25)	Group A (%)	Percentage (%)	Group B (n=25)	Percentage (%)
Pulmonary Tuberculosis	16	64	14	56
Bronchiectasis	6	24	7	28
Lung Abscess	2	8	2	8
Lung Cancer	1	4	1	4
Mitral Stenosis	0	0	1	4

Table-II

Amount of Haemoptysis

Extent of Haemoptysis	Group A (n=25)	Percentage (%)	Group B (n=25)	Percentage (%)	P-values
Mild	15	60	17	68	NS P=>0.05 Z= 0.56
Moderate	7	28	6	24	NS P=>0.05 Z= 0.32
Massive	3	12	2	8	NS P=>0.05 Z= 0.47

Table-III

Control of Haemoptysis in different treatment groups

Treatment Group	Control of Haemoptysis after 10 days of treatment	
	Controlled	Not Controlled
Group A	21	4 (n=25)
Group B	5	20 (n=25)
x2 = 20.5,	df =1, p= < 0.001,	highly significant

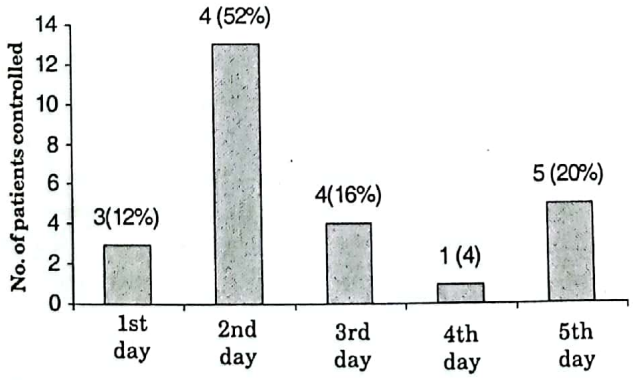

Fig. 1 : *Control of haemoptysis by days of treatment in diferrent treatment groups*

Discussion:

Tranexamic Acid is an anti fibrinolytic agent. it acts principally by inhibiting plasminogen activators as is used in the treatment of hemorrhage or threatened hemorrhage, assocaited with excessive fibrinolysis. It is being used for the control of bleeding of the gastrointestinal and gynecological origin for a long time. Tranexamic Acid is 7 to 10 times sure potent than aminocaproic acid[3]. Although, it can be used topically[4]. therefore, it has never been used in nebulised form in the management of haemoptysis.

We have given Tranexamic Acid in nebulized form in 25 patients in this study to evaluate its efficacy in the management of haemoptysis as a topical agent.

Table 1 shows that majority of patients had pulmonary tuberculosis (n=30, 60%). Thirty-two patients (64%) had mild hemoptysis i.e. less than 100 ml/day followed by moderate (100-200 ml/day) hemoptysis in 13 patients (26%) and severe (200-600 ml/day) hemoptysis in 5 patients (10%). Fig. 1 shows that in group A, majority of patients 13(52%) noticed control on 3rd day; whereas in group B, haemoptysis was controlled in only 5 (20%) cases on the 10th day of treatment. Surgical opinion was sought in patients with uncontrolled hemoptysis.

All patients remained hemodynamically stable during the course of treatment. No systemic on local side effects were observed during therapy and 2 weeks after therapy.

Conclusion:

Tranexamic Acid may be used safely in Nebulized form in mild to moderate haemoptysis. A multi-centre study is required to determine the efficacy of Tranexamic Acid nebulization in larger number of patients.

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Pattern of Dyslipidemia In Patients With Cardiovascular Dysmetabolic Syndrome Manifesting Ischemic Heart Disease

AA Jamil[1], KMHSS Haque[1], AA Mamun[2], MA Siddique[1], MN Islam[3]

Abstract:

Objective: To see the pattern of dyslipidemia in patients with Cardivascular Dysmetabolic Syndrome (CDS) manifesting ischemic heart disease.

Methods: A cross-sectional study was carried out in the Department of Cardiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh from June 1999 to May 2000. The patients clinically diagnosed or documented to have coronary artery disease (CAD) were included in the study, and were grouped into patients with CDS (CDS Group) and without CDS (non-CDS group). The CDS was diagnosed by using the diagnostic criteria defined by Western Working Group, Hawaii, 1997. Patients with hypertrophic and dilated cardiomyopathies, valvular and congenital heart diseases were excluded from the study. Fasting lipid profile was done in all the patients and the pattern of dyslipidemia was compared between the groups.

Results: Among 132 patients, 101 (76.5%) were in CDS group and 31 (23.5%) were in non- CDS group. The mean ages of the two groups were 50.21±8.08 and 44.03±11.89 years respectively. The patients presented with chronic stable angina, unstable angina, angina equivalent, atypical chest pain, AMI (Q and non-Q) and Old MI and there was no significant difference between the two groups (P>0.05). Total 108 patients had dyslipidemia and the difference was highly significant [99 (98.02%) vs 9 (29.03%); P<0.001]. There were highly significant differences regarding high triglyceride [89 (88.12%) vs 4 (12.9%); P<0.001] and low HDL-cholesterol [78 (77.23%) vs 9 (29.03%); P<0.001] types of dyslipidemias between CDS and non-CDS patients. High total cholesterol and high LDL-cholesterol were more in CDS patients but the differences were not statistically significant (P>0.05).

Conclusion: Analysis of lipid profile revealed that high triglycerides and low HDL cholesterol is the typical findings in CDS patients manifesting of ischemic heart disease.

***Key words**: Dyslipidemia, Cardiovascular Dysmetabolic Syndrome.*

[Chest & Heart Journal 2005; 29(2) : 117-123]

Introduction:

Coronary artery disease (CAD) is the leading cause of death and disability throughout the world. Dyslipidemia is one of the major risk factors for coronary artery disease. It is now well established that the risk of CAD is increased in individuals with dyslipidemia and the risk increases more if there is multiple major coronary risk factors including hypertension, smoking, dyslipidemia, diabetes mellitus and family history of CAD in a single individual. A clustering of metabolic abnormalities associated with cardiovascular disease was first described by Reaven in 1988[1]. This clustering has been given many unsatisfactory

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names including Syndrome X, the Deadly Quartet, and the Insulin Resistance Syndrome. The Western Working Group renamed this group of abnormalities as "Cardiovascular Dysmetabolic Syndrome (CDS)" in the consensus conference held on November 20-21, 1997 in Maui, Hawaii[2]. They proposed the mnemonic D-R-O-P, which stands for Dyslipidemia, insulin Resistance, Obesity, and high blood Pressure. A diagnosis of CDS requires the presence of criteria for at least 2 of the first 3 components (dyslipidemia, insulin resistance, and obesity). Dyslipidemia is more complex in the CDS than has been generally appreciated in the general population[2]. High-density lipoprotein (HDL) cholesterol levels are unequivocally inversely related to CAD and are protective cholesterol against CAD in both men and women[3]. The role of plasma triglycerides (TG) and TG-rich lipoproteins has been less clear. Elevated TG levels or very-low-density lipoprotein (vLDL) cholesterol levels appear to be an independent risk factor for CAD only when accompanied by lower levels of HDL cholesterol[4].

The aim of this study was to see the pattern of dyslipidemia in patients with Cardivascular Dysmetabolic Syndrome (CDS) manifesting ischemic heart disease (IHD).

Methods:

This cross-sectional study was carried out in the Department of Cardiology, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh during the period of January 1998 to June 2000.

Study Population:

Inclusion Criteria: All patients, clinically diagnosed or documented to have coronary artery disease, who required coronary arteriography was taken as study population.

Grouping of Study Population: were grouped into patients with CDS (CDS Group) and without CDS (non-CDS group).

Criteria for Cardiovascular Dysmetabolic Syndrome: The CDS was diagnosed by using the diagnostic criteria defined by Western Working Group, Hawaii, 1997[2] (Table-I).

Serum lipid profile was done in all patients after twelve hours fasing and the data were analyzed. Coronary arteriograms were also analyzed to document coronary artery disease.

Exclusion Criteria: Patients with hypertrophic and dilated cardiomyopathies, valvular and congenital heart diseases were excluded from the study. Fasting lipid profile was done in all the patients and the pattern of dyslipidemia was compared between the groups.

Statistical Analysis:

After processing of all available information, statistical analysis of their significance was done. The patients were grouped into those with and without CDS having coronary arteriography. All parametric values were expressed as mean±one standard deviation (mean±SD) and non-parametric values were expressed in percentage (%). The significance of differences between the two groups were determined by using unpaired Student's t test, Pearson's chi-square test and Z test where applicable. A 'P' value of less than 0.05 was considered to be significant.

Results:

In this prospective cross-sectional study 132 patients of IHD were studied among whom 101(76.5%) patients were in CDS group and 31(23.5%) patients were in non-CDS group (figure-1). Table-II shows the age and sex distribution of the study population. The CDS patients were relatively older than the non-CDS patients ($P<0.01$). The male to female ratio of total population was 10:1. The figure-2 shows the clinical presentations of both CDS and non-CDS groups. There were no significant differences in clinical presentations between the two groups ($P>0.05$). The distribution of components of CDS is shown in table-III. Dyslipidemia was the second commonest component of CDS next to obesity.

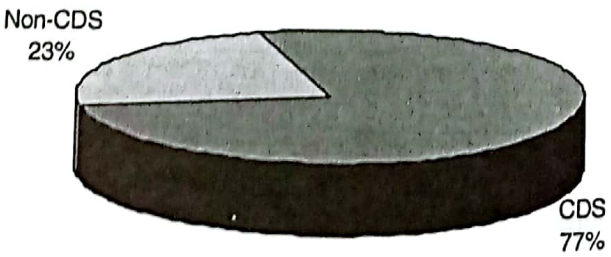

Fig.-1 : *Distribution of cardiovascular dysmetabolic syndrome (CDS)*

Table-1
Criteria for Cardiovascular Dysmetabolic Syndrome:

a) Dyslipidemia:
   i) Fasting triglycerides >140 mg/dl, OR
   ii) HDL cholesterol <40 mg/dl,
b) Insulin resistance:
   i) Fasting plasma glucose >110 mg/dl, OR
   ii) Type 2 diabetes mellitus
c) Obesity:
   i) Body mass index >25 k g/m^2, OR
   ii) Waist/Hip >0.85, OR
   iii) Waist >100 cm
d) High blood pressure:
   i) Systolic blood pressure ≥140 mm Hg, OR
   ii) Diastolic blood pressure ≥ 90 mm Hg

The presence of at least 2 of the first 3 components (dyslipidemia, insulin resistance and obesity) was required for the diagnosis of the CDS.

Analysis of fasting serum lipid shows CDS patients had significantly higher mean total cholesterol [211.30±42.91 mg/dl vs 184.52±45.47 mg/dl (P<0.01)] and triglyceride [255.13±143:36 mg/dl vs 115.39±54.22 mg/dl (P<0.001)], and lower mean HDL-cholesterol [36.15±6.97 mg/dl vs 41.77±7.31 mg/dl (P<0.001)] levels than those of non-CDS patients but no significant difference was observed between the two groups regarding LDL-cholesterol level [129.43±38.42 mg/dl vs 121.97±37.63 mg/dl (P>0.10)] (table-IV).

As expected, there were highly significant differences regarding high triglyceride and low HDL- cholesterol types of dyslipidemias between CDS and non-CDS patients (P<0.001). High total cholesterol and high LDL-cholesterol were more in CDS patients but the differences were not statistically significant (P>0.05) [table-V].

Table-VI shows that 32 (24.24%) patients had normal coronary arteriography, among them 22 (21.78%) were in CDS group and 10 (32.26%) were in non-CDS group. Insignificant coronary lesions were present in 10 (9.9%) and 3 (9.68%) respectively in both the groups. The CDS group had significant number of triple vessel disease than non-CDS patients [27 (26.73%) vs 2 (6.45%); P<0.001]. There were no significant difference between the two groups regarding normal coronaries, single and double vessel diseases [P>0.05].

Table-II
Distribution of age and sex

Parameters		CDS n=101	Non-CDS n=31	P value
Age	Mean ± SD	50.21±8.08	44.03±11.89	<0.01
	Range	24-68	20-75	–
Sex	Male	90(89.11%)	30(96.77%)	>0.05
	Female	11(10.89%)	1(3.23%)	>0.05

Table-III
Distribution of components of CDS

Components of CDS	CDS (n=101)	Non-CDS (n=31)	P value
Dysli idemia	99(98.02%)	9(29.03%)	<0.001
Insulin resistance	53(52.48%)	2(6.45%)	<0.001
Obesity	101(100%)	18(58.06%)	<0.041
High blood pressure	62(61.39%)	10(32.26%)	<0.001

Table-IV

Fasting serum lipid profile

Types of lipid	CDS (n=101)	Non-CDS (n=31)	P value
TC (mean± SD) mg/dl	211.30±42.91	184.52±45.47	<0.01
LDL-C (mean± SD) mg/dl	129.43±38.42	121.97±37.63	>0.10
HDL-C (mean± SD m /dl	36.15±6.97	41.77±7.31	<0.001
TG (mean± SD) mg/dl	255.13±143.36	115.39±54.22	<0.001

TC = Total Cholesterol, LDL-C = Low Density Lipoprotein Cholesterol, HDL-C = High Density Lipoprotein Cholesterol, TG = Triglyceride, SD = Standard Deviation

Table-V

Distribution of dyslipidemia

Types of dyslipidemia	CDS (n=101)	Non-CDS (n=31)	P value
High TG (> 140mg/dl)	89(88.12%)	4(12.9%)	<0.001
Low HDL-C (<40mg/dl)	78(77.23%)	9(29.03%)	<0.001
High LDL-C (>130 mg/dl)	49(48.51%)	13(41.94%)	>0.05
High TC (>200 mg/dl)	58(57.42%)	14(45.16%)	>0.05

TG = Triglyceride, HDL = High Density Lipoprotein, LDL = Low Density Lipoprotein, TC = Total Cholesterol

Table-VI

Distribution of number of diseased vessels

Diseased vessels	CDS n=101	Non-CDS n=31	P value
Normal coronaries	22(21.78%)	10(32.26%)	>0.05
Insignificant CAD	10(9.9%)	3(9.68%)	>0.05
SVD	23(22.77%)	9(29.03%)	>0.05
DVD	19(18.81%)	7(22.58%)	>0.05
TVD	27(26.73%)	2(6.45%)	<0.001

CAD = Coronary Artery Disease, SVD = Single vessel disease, DVD = Double Vessel Disease, TVD = Triple Vessel Disease

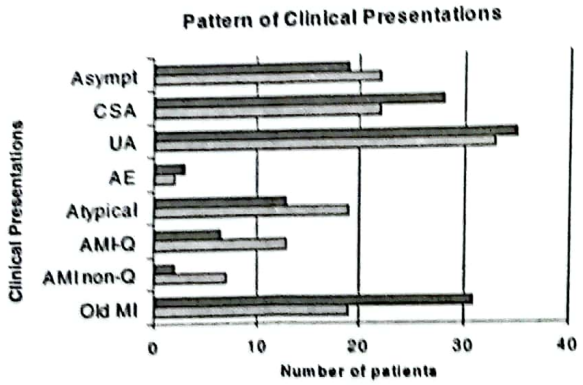

Fig.-2 : *Distribution of pattern of clinical presentations in CDS and non-CDS patients.*

Discussion:

South Asians have more CHD than Europeans despite apparently lower levels of risk factors. It is thought that there is high prevalence of cardiovascular dysmetabolic syndrome (CDS) in the Indian subcontinent as suggested by several studies on immigrants in Europe, South Africa and USA[6]. The reason behind the pathogenetic mechanism for CAD in this ethnic population seems to be the CDS[7]. Furthermore, in the people of South Asian origin the CAD frequently occurs at an early age and that it is diffuse and severe[6,8].

In this study, the mean age of CDS and non-CDS patients were 50.21±8.08 (range = 24-68) years and 44.03±11.89 (range = 20-75) years respectively, reflecting that CDS patients are relatively older than the non-CDS patients and the difference was significant (P<0.01). Haque et al[9] found the mean age of their IHD patients to be 53.33±13.21 years, Safiuddin et al[10] reported to be 48.98±8.37 years of their CAD patients and Uddin et al[11] showed to be 50.49±10.79 years in Bangladeshi population, which support the finding of the presenting study. In the comparative studies of CAD between immigrant South Asians and white population the mean age shown by Hughes et al[8] were 50.7 vs 55.9 years, Enas et al[7] showed 48.0±5.5 vs 53.2±9.5 years, and Bhopal et al[15] found 50.84±13.1 vs 54.17±13.1 years. Cianflone et al[12] found the mean age in Italian population to be 54±8 years. So, the population of this study was younger than the Western people as well. The male to female ratio of total population was 10:1. Haque et a1[9], Safiuddin et al° and Uddin et al[11] also found similar sex ratios. There was no significant difference of sex distribution in CDS and non-CDS patients (P>0.05) in the present study.

Among the 132 patients of the present study, 101 (76.5%) patients had CDS and 31 (23.5%) patients were non-CDS. The clinical presentations were similar in both CDS and non-CDS groups. The presentations were asymptomatic [19.80% vs 22.58% (P>0.05)], chronic stable angina [33.66% vs 32.26% (P>0.05)], angina equivalent [3.96% vs 3.23% (P>0.05)], atypical chest pain [14.85% vs 19.35% (P>0.05)], Q wave acute myocardial infarction (AMI) [5.94% vs 12.9%(P>0.05)], AMI non-Q wave [1.98% vs 6.45% (P>0.05)] and old MI [30.69% vs 19.35% (P>0.05)]. Malik et al[13] found AMI in 57.76% and angina pectoris in 42.24% of patients. Solymoss et al 14 found 52% of their patients having old MI. Cianflone et al[12] demonstrated stable angina pectoris in 24.5%, unstable angina pectoris in 24.5%, AMI in 51% of patients with IHD. Hughes et al[8] showed atypical chest pain in 51% of Asians and 39% of white patients.

Regarding components of CDS, dyslipidemia was most common in this study. It was significantly higher in CDS group than in non-CDS patients [98.02% vs 29.03% (P<0.001)]. Haque et a1[9] reported 26.19% of IHD paitents to be hypercholesterolemic and Haque et al[15] ʸ found 42% in AMI patients to be dyslipidemic in Bangladeshi population. Next to dyslipidemia was obesity and was significantly higher in CDS patients than in non-CDS group [100% vs 58.06% (P<0.001)]. Haque et a1[9] demonstrated 19.40% of patients and Bhopal et al[5] showed 66% in South Asian immigrants at Newcastle, UK. In the present study, hypertension was also significantly higher in CDS patients than in non-CDS group [61.4% vs 32.3% (P<0.01)]. Safiuddin et al[10] observed 54% in IHD patients, Haque et a1[15] showed 42% in AMI patients and Haque et al[9] demonstrated 23.80% of patients to be hypertensive in Bangladesh. Enas et al7 found 12% in general population of immigrants Asian Indians at USA where as Bhopal et al[5] reported 14% in general South Asian population at UK. In this study, insulin resistance was least common among the components of CDS and it was significantly higher in CDS group than non-CDS patients [52.5% vs 6.5% (P<0.001)]. Bhopal et a1[5] found 21% in South Asian immigrants at UK. In the comparative studies between immigrant Asian Indians and Caucasians showed hypertension and insulin resistance are higher in immigrants than in Caucasians. Bhopl et al[5] reported higher level of blood pressure in South Asians (14% vs 12%) and also insulin resistance (21% vs 8%), where as Enas et al[7] showed no difference between the two ethnic groups regarding hypertension [11.8% vs 11.9% (P=0.96)].

Analysis of fasting serum lipid profile showed significant difference between CDS and non-CDS groups regarding mean total cholesterol [(211.30±42.91 vs 184.52±45.47) mg/dl; P<0.01], HDL-cholesterol [(36.15±6.97 vs 41.77±7.31) mg/dl; P<0.001], and triglyceride [(255.13±143.36 vs 115.39±54.22) mg/dl P<0.001] but mean LDL-cholesterol did not differ between the two groups [(129.43±38.42 vs 121.97±37.63) mg/dl; P>0.10]. These findings are in consistent with that of Uddin[16] where he found mean total cholesterol, LDL-cholesterol, HDL-cholesterol and triglyceride 217.66±38.27 mg/dl, 139.06±38.06 mg/dl, 38.54±7.76 mg/dl and 216.52±91.78 mg/dl respectively in his patients with CAD. Hughes et al[8] showed that total cholesterol concentrations [(223.51 vs 240.53) mg/dl], LDL-cholesterol concentrations [(187.93 vs 200.31) mg/dl] and HDL-cholesterol concentrations

[(35.58 vs 40.22) mg/dl] were lower in Asian immigrants than those of white patients with AMI, whereas triglyceride was higher in Asians than that of white patients [(170.06 sv 146.15) mg/dl]. In the present study high triglyceride (>140 mg/dl), low HDL-cholesterol (<40 mg/dl), high LDL-cholesterol (>130 mg/dl) and high total cholesterol (>200 mg/dl) were observed in 88.1%, 77.2%, 48.3% and 57.4% of patients respectively in CDS group and 12.9%, 29%, 41.9% and 45.2% of patients respectively in non-

CDS group. There were significant differences between the two groups regarding high triglyceride and low HDL-cholesterol (P<0.001) but no significant differences were observed regarding high LDL-cholesterol and high total cholesterol levels (P>0.05). Comparative studies between South Asian immigrants and white population abroad showed similar high triglyceride and low HDL-cholesterol in South Asians than the white people. Enas et al[7] reported high triglyceride (18.5% vs 11.3%) in Asians than in whites. Bhopal et al[5] found high triglyceride in 55% of South Asians and in 37% of Europeans; and low HDL-cholesterol in 30% of South Asians and in 13% of Europeans.

In the present study, 32 (24.24%) patients had normal coronary arteriography, and there was no significant difference between CDS and non-CDS groups [21.78% vs 32.26% (P>0.05)] though normal coronaries were little higher in non-CDS group. Non-significant coronary lesions (<50% stenoses) were also not significantly different between the two groups [9.9% vs 9.67% (P>0.05)]. CDS patients had significant number of triple vessel disease (TVD) than non-CDS patients [26.73% vs 6.45% (P<0.001)] but no significant difference were found regarding single vessel disease (SVD) and doubly vessel disease (DVD) [22.77% vs 29.03% (P>0.05) and 18.81% vs 22.58% (P>0.05) respectively]. There were also no correlation between the presence of number of components and the number of diseased vessels (P>0.05). Dortimer et al[17] found normal coronaries in 11% vs 27% (P<0.1) of patients with or without diabetes, SVD in 14% vs 24% (P>0.1), DVD in 32% vs 24% (P>0.3) and TVD in 43% vs 25% (P<0.1). Uddin[16] showed in his diabetic and non-diabetic patients that normal coronaries, SVD, DVD and TDV in 0% vs 6% (P>0.05), 18% vs 26% (P>0.05), 24% vs 30% (P>0.05), 58% vs 38% (P<0.05) respectively. Hughes et al[8] compared the numbered of diseased vessels in Asians with those of white people and observed SVD in 20% vs 42% (P<0.01), DVD in 27% vs 37% (not significant) TVD in 54% vs 21% (P<0.001). So, Bangladeshi people and other South Asians have more TVD. Safiuddin et al[18] contradicted this finding by demonstrating more SVD (54%) and less TVD (12%) in unheralded AMI patients. Bogaty et al[18] also showed similar result where SVD, DVD and TVD were 61.8%, 18.2% and 10.9% respectively in AMI patients.

Thus, in conclusion, there were highly significant differences regarding high triglyceride and low HDL-cholesterol types of dyslipidemias between CDS and non-CDS patients but total cholesterol and LDL-cholesterol were similar in both the groups. This reflects that high triglyceride and low HDL-cholesterol types of dyslipidemias are more important as causative factors than the serum level of total cholesterol and LDL-cholesterol in the patients of Bangladesh.

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REVIEW ARTICLES

Sleep Apnea Syndrome -A Review

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[Chest & Heart Journal 2005; 29(2) : 124-126]

Sleep Apnea is defined as the presence of an abnormal number of breathing cessations (apneas) or reductions in ventilation during sleep[1]. Sleep apnea syndrome is characterized more precisely as sleep apnea associated with nocturnal or daytime symptoms. By convention, apneas of at least 10 seconds duration have been considered important but in most patients the apneas are of 20 to 30 seconds duration and may be as long as 2 to 3 minutes. By the time most of the patients come to attention they have at least 10 to 15 events per hour of sleep. In the Wisconism sleep cohart study, the prevalence of OSA syndrome was 4% in men and 2% in women[2].

Sleep apneas have been classified into three types:

Central
Obstructive
Mixed

Central Sleep Apnea (CSA) - The neural drive to all the respiratory muscle is transiently abolished. In obstructive sleep apnea (OSA) - airflow ceases despite continuing respiratory drive because of occlusion of the oropharyngeal airway.

Mixed apneas that consist of central apnea followed by an obstructive component, is a variaril of OSA.

The definitive event in USA is occlusion of the Upper airway usually at the level of oropharynx. The resulting apnea leads to progressive asphyxia until there is a brief arousal from sleep. The patient then returns to sleep and sequence of events is repeated often up to 400 to 500 times per night, resulting in fragmentation of sleep[3].

The obstruction most commonly, occurs in the oropharynx or hypopharynx or both. In the oropharynx, the tongue and uvula prolapse posteriorly and there occurs invagination of lateral and posterior pharyngeal tissues. Similarly in the hypopharynx, the root of the tongue and epiglotti's prolapse posteriorly.

Many patients with obstructive apneas have some degree of narrowing of the upper airway from a variety of causes, such as.

Obesity-. Significantly overweight individuals are likely to have obstructive sleep apnea, some workers have shown that collections of adipose tissue reduce the caliber of the upper airway.

Adenotonsillar hypertrophy: More commonly a factor in children.

Macroglossia: Most frequently attributed to hypothyroldism and myxedema

Mandibular deficiency: Micrognathia and retrognathia can result in posterior displacement of the tongue and compromise the upper airway lumen.

This may explain the tendencies of OSA be inherited in some families,

Rhinitis

Upper Airway Tumors

The immediate factor leading to collapse of the upper airway in OSA is the generation of a critical sub atmospheric pressure during inspiration that exceeds the ability of the airway dilator and abductor to maintain the airway stability'. In sleep, generalized reduction in muscle tone causes narrowing of the upper airway even in healthy individuals. Any factor that reduces airway size, increases upper airway resistance, decreases muscle tone.

Clinical Features:

The sensitivity and specificity of history and examination for the presence of sleeps disordered breathing are only 50 -70%.

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History: The classic symptoms of OSA are heavy snoring, excessive daytime sleepiness and witnessed apnea. When all three are present, OSA syndrome is more than 90% likely. A history from bed partners and family members improve detection of symptoms. Other symptoms are

- Nocturnal choking and gasping.
- Nocturnal reflux
- Nocturnal enuresis
- Morning headache
- Dry throat
  - Unrefreshed sleep
  - Restless sleep, frequent awakening.
  - Impaired cognitive function
  - Mood change
  - Reduced libido and impotence

Complications of obstructive sleep apnea:

Cardiac Arrhythmia's: Bradycardia, sinus pauses with various degrees of A-V blocks are also seen.

Systemic hypertension: Obstructive apneas are frequently associated with transient elevations of systemic blood pressure.

Myocardial infarction, Stroke, Pulmonary hypertension and corpulmonaie. Obesity hypoventilation syndrome.

Management : Because mortality is increased in patients with an apnea index of more than 20, most clinicians treat this group regardless of symptoms.

Behavioral Treatment:

- Reduction of body weight
- Sleep on side
- Avoidance of sedatives prior to sleep
- Avoidance of sleep deprivation
- Elevation of head during sleep
- Colds and allergies should be treated promptly
- Avoidance of large meal prior to sleep
- Cessation smoking

Medical Treatment: Pharmacotheraphy - Tricyclic antidepressant may increase the upper airway muscle tone.

- 0_2
- Nasal CPAP
- Bilevel CPAP
- Oral appliance to maintain airway.

Nasal CPAP (Continuous Positive Airway pressure)[8,9]. It is applied via a nasal mask with a soft plastic cushion to provide an airtight seal & held in head by head gear and has Velcro straps fastened around the back or the head. Positive pressure is maintained in the upper airway by the CPAP apparatus, physically hold the airway open despite insufficient dilator muscle tone.

CPAP improves daytime sleepiness, daytime cognitive function, mood and quality of life CPAP decrease the incidence or traffic accidents in OSA population. It can reverse or significantly improve daytime hypertension and corpulmonale. CPAP may reduce the mortality with OSA syndrome. Compliance is better in those who experience significant relief of daytime symptoms. Various cheap, light and automated machines are now available[10].

Mandibular advancement device: Are intra-oral devices that displaces the jaw anteriorly increasing the anteroposterior diameter of upper airway and reducing collapse when worn at night. Specialist orthodontic collaboration is needed[12].

Surgery: Nasal surgery is seldom useful in OSA. Adeno-tonsillectomy can be Curative in children & adolescents.

Uvulo-palato-pharyngoplasty (UPPP): Involves removal of tonsils, uvula and parts of the soft palate and pharyngeal folds. It is the most commonly performed surgical procedure in OSA, It has significant morbidity. It is useful in less than 50% of Patients.

Complex maxillofacial surgery- Success rates are more. Surgery is performed in two stages[13].

- Genioglossal advancement via mandibalar osteotomy with uvulo-palato-

  pharyngo plasty and nasal surgery
- Maxillomandibular osteotomy

  Tracheostomy : This is the last resort in patient with seveie or complicated OSA who can not tolerate CPAP.

CSA (Central sleep Appea)[14].

- Management of patients whose CSA arises from and instability of respiratory drive is more difficult. Patients with hypoxernia usually respond favorably to nocturnal supplemental Oxygen. Others have responded to acidification

with acetazolarnide. Nasal CPAP is also much effective.

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The Role of Exercise in Cardiac Rehabilitation after Uncomplicated Myocardial Infarction.

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Abstract

Literature is reviewed on the role of exercise in cardiac rehabilitation (CR) for patients after uncomplicated post myocardial infarction (MI). Exercise plays a role in each of the four phases of CR. Meta-analysis demonstrated a 20% reduction in mortality following an exercise programme but no significant reduction in recurrence of MI.

Physiological benefits include increased aerobic capacity with reduced exercise heart rate and blood pressure, reduced angina and ST segment depression, increased levels of high density lipoprotein and sometime increased myocardial contractility. The extent of these changes depends on the amount of exercise undertaken.

Depression and anxiety, where diagnosed, may be reduced by exercise although other factors such as group support may contribute.

Recent studies report benefits following two to four months exercise starting three to six weeks after MI. Non-exercising controls generally reach a similar stage at one year after MI. The role of exercise in such programmes may be one of encouraging motivation to exercise rather than to achieve significant increases in aerobic capacity. Home exercise programmes, correctly prescribed, are a real possibility for suitable low-risk patients and may facilitate long term adherence to exercise.

Strategies to reduce drop-out from CR and encourage underrepresented groups to participate should be formulated.

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Introduction:

Cardiac rehabilitation (CR) comprises a multifactorial programme of regular physical exercise, advice and education on risk factor modification and stress management. This type of approach gives optimum benefit to a wide variety of cardiac patients as it provides opportunity for physical, psychological, social and vocational needs to be addressed.'" The exclusion criteria in this uncomplicated MI category are the patients who have suffered MI but have no subsequent evidence of congestive heart failure, unstable angina, significant arrhythmias and less than 35% of the left ventricle affected by ischaemia[3,4,5]. Other criteria that exclude patients from this category are the physical limitations imposed by severe obesity, orthopaedic and peripheral vascular disease, stroke, chronic lung disease and low exercise tolerance.

The review aims to identify the role of exercise in the rehabilitation of patients after uncompliated MI from phase 1 to phase 4 of rehabilitation. The benefits of physical exercise will be reviewed. Different types of exercise programme will be discussed, together with the amount of exercise required to be of benefit.

Four phases of cardiac rehabilitation may be identified which vary in length depending on the rate of recovery of individual patients. They are described[6] as phase 1 - inpatient stay; phase 2 -

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convalescent period at home, phase 3 - intermediate post discharge and phase 4 - long term maintenance. Phases 1 and 2 involve gradual mobilisatons of patients within their own limitations. Generally they would be able to climb stairs at about five to seven days post MI. Over next two to six weeks (phase 2) patients are encouraged to walk progressively further until a daily distance of two miles is achieved. During phase 3 patients may attend a formal, supervised out-patient programme of exercise and participate in various psycho-social interventions that may include relaxation, counseling or continued education and advice on risk factor modification. The exercise should be prescribed for each patient following a graded exercise test. This will ensure a safe and effective level of physical activity. As patients progress into phase 4, they should be encouraged to undertake move unsupervised exercise of a suitable frequency, intensity and duration to maintain the increased fitness achieved in the supervised programme.

Benefits of Exercise:

Early randomised controlled trials of exercise based CR showed a trend in reduction of mortality and non-fated recurrence of MI, although results failed to reach statistical significance because samples were too small[7,8],. Meta-analyses of exercise based CR showed a significant reduction of 20% in mortality upto three years after an acute cardiac event but no significant change in morbidity[9,10]. The authors of these metanalyses also suggest that the results may not be entirely due to exercise as some of the programmes included in the studies involved education and advice as well as prescribed physical activity.

As huge numbers of patients are required to demonstrate statistically significant changes in morbidity and mortality, more recent research has measured specific variables, such as maximal oxygen consumption (VO_2 max), sub-maximal heart rate and blood pressure[11,12] contractile force of myocardium[3,13,14], angina and lipid levels[13,15,16].

Physiological Effects of Cardiac Rehabilitation Programmes

Lower HR and BP: - A reduced heart rate and blood pressure for a given work intensity in MI patients have been demonstrated[11,14,15] and reported following three to four months of regular exercise training. The rate pressure product (RPP=heart rate x Systolic blood pressure), an indication of myocardial oxygen consumption, fell in a group of 100 MI patients following a three- month controlled exercise programme[17]. A control group showed no change in rate pressure product (RPP). Conversely, a trial by Leizorovicz" et al found no significant fall in RPP with exercise. They suggest this might show an habituation effect of exercise rather than an aerobic training effect. Their exercise programme lasted only six weeks, however, which might have been too short to allow a training effect to develop.

After one year of exercise training, Froelicher et al[18] found a reduction in heart rate and blood pressure at a given workload. This was partly due to a direct improvement in ventricular function. Subjects were selected individuals with stable coronary heart disease. Vo_2 max was increased and myocardial perfusion also improved.

There is a 37% improvement in Vo_2 max with significant reduction in exercise heart rate, blood pressure and ST segment depression[13,19]. The result were achieved following a year of regular aerobic activity, the last nine months of which was performed four or five times weekly at 70-80% of a patient's Vo_2 max. Sophisticated measurement techniques showed an increase in left ventricular ejection fraction reflecting a direct improvement in ventricular contractile function. Other studies demonstrate more modest but significant improvements in physical work capacity (PWC) following three or four months exercise training beginning six to eight weeks after MI[11,17]. Authors studying the effects of short-term exercise programme soon after MI consider improvements in PWC to be largely due to skeletal muscle adaptation[17,20]. This is based on work by Detry et al[21] (1971) who found an increase in Vo_2 max of 22.6% in a group of 12 patients with coronary heart disease following three months exercise training. The stroke volume during sub-maximal exercise was unchanged and the exercise heart rate reduced. The arteriovenous oxygen difference was increased implying that increased peripheral oxygen extraction was the reason for improved physical work capacity. This is due to the increased enzyme activity in the trained muscles that was

demonstrated by Gollnick et al (1973)[22,23]. Increased enzyme activity enables muscle mitochondria to produce a higher respiratory rate, thereby increasing local aerobic capacity, and reducing demands on the myocardium.

Reduce Angina: Exercise training can also reduce angina[3]. Bethell and Mullee[25] found a 10% reduction after three months training compared to a 60% increase in a non exercising group. Angina was also reduced in some of the patients following 12 months training[26]. This may be due to peripheral adaptation or improved myocardial function or both.

Lipid Levels: High density lipoprotein (HDC) are recognized to have a protective influence against heart disease whereas low density lipoproteins may accelerate atheroselerosis[25,26]. Studies of Ehsani et al (1986)[13] and Blumenthal Rajeski et al (1988)[15] demonstrate increase in HDL, following 12 and three months' training respectively. Heath et al (1983)[27] found an 8% reduction in total cholesterol, a 9% reduction in low density lipoprotein and 11% increase in high density lipoproteins. However, this study included only ten patients exercising at high intensity for six months.

Effects of Early Short-term Programmes: Some authors have found that untrained groups have caught up with short-term trained groups when cardiovascular function has been re-evaluated six months to one year after an acute cardiac event[28,29]. The role of exercise in such short-term cardiac rehabilitation programmes is primarily to motivate patients to resume their previous levels of activity. This implies that the amount of exercise undertaken is less important than the patient's psychological attitude forwards activity during the recovery period. However, increasing the rate of recovery and restoring patients to a fuller life earlier should allow them to enjoy a better quality of life for longer and, if appropriate, an earlier return to work. A five-year study carried out by Hedback and Perk (1987)[28] did show continued benefit to an exercise group compared with a non-exercise group. However, the exercise group continued with a monthly supervised exercise session which may have provided the motivation to maintain increased fitness.

Psychological Effects of Exercise: Physical activity is reputed to reduce depression and anxiety, to enhance mood in general[ly], compared three groups of MI subjects undergoing an exercise programme, group counseling, or usual medical care by a physician. Subjects were all diagnosed with depression or anxiety. Results showed that a 12 week aerobic exercise programme reduced fatigue, anxiety and depression, promoted independence and sociability and considerably increased work capacity. Group counseling over 12 weeks resulted in substantial reductions in depression, promoted sociability and facilitated coping with interpersonal relationships. The usual care group showed no changes in any of the measured parameters. It appears from this study that an exercise programme with its apparent combined physical and psychological benefits is the optimum feature of cardiac rehabilitation. However, at 12 months there were no significant differences between any of the groups as the psychooocial status of the us usual care group had improved to a compared level.

Newton et al (1991)[29] studied 22 MI subjects and the psychological effects of an exercise-based cardiac rehabilitation programme. Depression and mood status were assessed in all subjects at the beginning and end of a ten week rehabilitation period. Compared with a control group of 'usual medical care' both groups improved their physical work capacity but the exercise group also showed reduced depression. The authors suggest that an exercise based programme appears to have psychological benefits, but they also propound other possible reasons for this benefit. They suggest the effect of social interaction, peer support, or being supervised by professional. Education and relaxation sessions included in the programme may also have contributed to the improved psychological status.

Exercise Programmes: Each subject must have an individual prescription based on a symptom -limited graded exercise test:-

Frequency: Studies that have demonstrated a training effect included exercise at least three times a week[1,30,31]. An exercise frequency of four or five times weekly may pose a challenge for less committed patients or create conflict with their other interests. Some authors have prescribed

twice weekly formal exercise with daily walking on the remaining days[32, 33]. A few studies describe daily exercise regimes comprising home-based walking programmes[34,35]. Though these studies are rather isolated in their approach, perhaps they deserve more consideration, particularly for patients who do not adhere well to a more formal exercise programme.

Intensity: Exercise intensity should be prescribed for each patient according to the results of a stress test to ensure that the level is safe and effective. Blumenthal et al[30] studied the effects of low (< 45% Vo_2 max) versus high intensity exercise (65-75% Vo_2 Max) in patients eight weeks after MI. After 12 weeks both programmes produced a similar increase in PWC. The authors concluded that in the short term a low intensity exercise programme was as effective as a high one for increasing Vo_2 max.

Prescription of target heart rate range is based on the results of exercise testing. Although in prior years, patients were advised to exercise to a target heart rate range between to 70 and 85 percent of the highest level safety achieved at exercise testing,[39] exercise intensities in the 50 to 70 percent heart rate range have produced comparable improvement in functional capacity and endurance and may provide greater safety because of the lower risk of cardiovascular complications with unsupervised exercise[30,33,34]. These lower rates are less likely to produce discomfort that may deter long-term exercise adherence. The documented efficacy of lower intensity exercise training to improve aerobic capacity has increased both its applicability and acceptance. An alternative method for calculating target heart rate involves 70 to 85 percent of the difference between peak exercise test heart rate and resting heart rate, added to resting rate. This method may be advantageous in patients whose heart rate is attenuated by (ß blocking or other drugs.

Kugler et al (1990)[34] describe a mainly home based walking programme which proved comparable to an entirely hospital-based cycle ergometry programme in terms of increased physical work capacity. They do not describe the intensity of the walking, or report whether the home based exercise intensity was monitored for consistency. The results offer positive implications for financing of programmes and patient convenience in attending hospital only once a week. If similar benefits may result from a lower intensity exercise, it seems preferable to use this rather than a higher intensity which carries greater risk of musculoskeleted injury, causes some patients considerable subjective discomfort, and has an element of greater cardiovascular risk[30].

Timing: Duration of exercise sessions is closely linked to the frequency and intensity of exercise. With uncomplicated MI, an exercise programme may begin as early as three weeks after the events[5,33]. These early programmes generally consist of aerobic sessions of 30 minutes at an individually prescribed heart rate. This is gradually progressed to 45 minutes and training is carried out three or four times a week[1,5].

In exercise programmes starting soon after MI, most of the training effect occurs in the first six to eight weeks[36, 37]. The minimum length of a programme may be six weeks[32] to eight weeks and some may last 12 weeks[14,30]. A few studies report results after one year.

Mode of exercise: Hall et al 38 suggest cardiac patients may benefit best from a circuit-interval type training in the early phase of rehabilitation, progressing to a continuous conditioning type after about three months. The advantage of circuit-interval training is that patients get variety in modes of exercise, using cycle and arm ergometers, bench stepping and rowing machines, working on each piece of equipment for a few minutes. A rest period can follow each station if required and this may be particularly important in the early stages of recovery. Various authors have demonstrated significant improvements in physical work capacity following circuit-interval training[1,17,33]. A further advantage of a circuit is that most large muscle groups of the body can be exercised. This is important as exercise is task-specific. Therefore, to enable patients to gain optimum recovery and functional capacity, exercise of the arms should be incorporated, as many occupations and activities of daily living involve use of the upper limbs.

Continuous conditioning further increases aerobic capacity. The patients perform one type of activity-usually walking, jogging or cycle ergometry-at a constant intensity for a gradually increasing period. There is no rest until the exercise time is completed.

Location of Exercise programmes: Cardiac rehabilitation programes may be based in hospitals, community centers or at home De Busk et al [5,40] suggest that for appropriate patients home-based exercise training at an individually prescribed intensity can be just as safe and effective as supervised exercise.

In a randomized controlled trial of low risk MI subjects, De Busk et al[5] showed an increased work capacity of 0.9 NETS in subjects who undertook prescribed exercise at home with monthy telephone contact with a cardiac nurse compared with a 'usual care' group.

Low risk patients are those who have had an uncomplicated MI and in whom the following signs are absent on a symptom-limited graded exercise Test (SLG x T): ischaemic ST segment depression, angina, significant arrhythmias such as frequent premature ventricular complexes and exercise hypotension. In addition, patients physical work capacity must be greater than 5 METS on the SLG x T[41,42,43].

Home based exercise programmes are an attractive option in the present economic climate and offer advantages to patients in terms of convenience, abolition of traveling costs, and the opportunity to become independent and responsible for regaining their own fitness. They may also facilitate progress to phase 4 of rehabilitation.

Conclusion:

Regular physical exercise can reduce mortality after MI. Following an exercise programme physical work capacity is increased, sub-maximal heart rate, blood pressure and angina are reduced and high density lipoprotein levels may be increased. In selected patients, exercise of sufficiently high intensity and frequency over a year can increase myocardial contractility and stroke volume. Many shorter-term exercise programmes attribute improvements in physical work capacity to musculoskeletel adaptation. These types of programmes serve to accelerate the rate of recovery but non-CR patients will reach a similar physical work capacity after one year.

With the current economic constraints on services, home-based exercise programmes may become more popular. As exercise is individually prescribed, it would lend itself to a home-based programme for suitable, uncomplicated MI patients. This would free facilities for supervised exercise for higher-risk patients. However, such decision should based on correct identification of low risk patients through clinical assessment and discussion with relevant members of the multi-disciplinary team.

Other components of the CR programmes such as stress management or dietary advice are essential to ensure an holistic approach to achieving and maintaining a healthy lifestyle. Education of patients and relatives to encourage a change in attitude towards healthy living is very important if phase 4 of CR is to be successful. This includes developing a positive attitude towards regular exercise. Therefore, a CR programme must include this encouragement even if exercise is home-based.

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CASE REPORTS

Tracheal Tumour and Asthma Mimic Each Other - Report of Three Cases

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Abstract

Three case reports are presented in this article. Out of three cases, two were being treated as bronchial asthma initially. Ultimately, these two cases were diagnosed as endotracheal tumour - one was leiomyoma and the other was carcinoid. In contradistinction, the third one was considered to be a case of obstructive sleep apnoea syndrome or endobronchial/endotracheal lesion. Interestingly, it was established as a case of severe persistent asthma and the patient improved with the conventional management of bronchial asthma.

It is obvious that tracheal tumors and asthma mimic each other. The rarity of the cases and comparatively better prognosis following surgery inspired us to report these cases.

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Introduction:

Benign tumours of the lung account for about 2% to 5% of intrathoracic tumours[1]. In order of frequency, the tumours are found in the peripheral lung, small bronchi, central bronchi and trachea. The clinical picture depends primarily on the location of the lesion. Symptoms, signs and roentgenologic features differ among benign tumours that arise in trachea, the bronchi and the lung parenchyma. Endotracheal benign tumors may be asymptomatic, but may manifest with wheezing, cough, dyspnoea or haemoptysis. The leiomyomas are the most common soft tissue tumors found in the lungs[2].

Careful examination of the tracheal air shadow by standard roentgenography may help to detect the tumour but confirmation by computed tomography (CT) is required. Bronchoscopy is usually diagnostic and may even permit adequate tumor removal by forceps or with the aid of laser technology. Surgical resection, however, is treatment of choice. Leiomyoma, leiomyosarcoma and carcinoids have all been reported in the trachea, presenting with signs of respiratory obstruction. This may be misdiagnosed as asthma. Careful history, clinical examination, FOB and relevant investigations, especially. HRCT of chest might be helpful in the diagnosis of leiomyoma and bronchial carcinoid[3].

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Case Report-1

A 36-year-old lady presented to a pulmonologist with severe respiratory distress, occasional cough and haemoptysis for one month. Breathlessness was associated with occasional wheeze and was gradually increasing in severity. Cough was unproductive and haemoptysis was scanty. Since her illness, she was on treatment with Tab. Aminophylline, Tab. Betnelan etc., but with no improvement. She had no personal or family history of bronchial asthma or atopy. She is neither hypertensive nor diabetic. On examination she was alert with respiratory distress with prominence of accessory of muscles of respiration. Breath sound was vesicular and of diminished intensity with occasional rhonchi in both the lung fields. Urgent high resolution CT scan (HRCT) of chest was recommended. HRCT revealed a sessile polypoid mass measuring 19.9X16.0 mm within the trachea, about 5 cm above the carina. The lesion was attached to the right posterior wall of the trachea, which almost obliterated the tracheal lumen. The impression was *intra-tracheal polypoid mass*. Following the diagnosis, the patient was attended by an ENT Surgeon and urgently transferred to a specialized hospital in Bangkok for further management where urgent surgery was recommended. Resection of the tumour and trachea (four rings) with end-to-end anastomosis was done. The histopathological diagnosis was Epitheloid Leiomyoma (Benign Leiomyoblastoma). Immunohistochemical study was negative for cytokeratin and CAM 5.2 and positive for vimentin and actin which makes an epithelial tumor unlikely and supports the smooth muscle nature of the tumor. At present the lady is in good health with no respiratory distress and leading a normal life.

Case Report-2

A 15-years-old boy presented with severe respiratory distress for four months which was gradually increasing in severity. It was associated with cough, which was productive, purulent and profuse. He had no personal or family history of bronchial asthma, atopy or allergy. He was being treated with conventional medicines used for asthma including nebulised bronchodilators and steroids, both oral and inhaled. On examination, patient was in respiratory distress with prominence of accessory muscles of respiration. On auscultation of left side there were features of diminished air entry peripherally with bronchial breath sound centrally. On the right, auscultatory findings were suggestive of resolving pneumonia. Chest X-ray (PA view) revealed non-homogeneous opacities in the right lower lung field. High resolution CT scan (HRCT) of chest revealed a small nodular swelling measuring about 1.5X1.5 cm, on the left tracheal wall just above the carina, partially occluding the opening of the left principal bronchus. Bronchoscopy and biopsy was not done pre-operatively for the risk of uncontrolled haemorrhage. The patient was directly prepared for surgical intervention. Per-operatively, the tumor was identified and HRCT finding was confirmed. The tumor bleed on touch and its base extended from lower tracheal ring to adjacent areas. It was excised by wedge resection. The post-operative period was uneventful. The histopathological diagnosis was *pulmonary carcinoid tumor*. After the diagnosis, opinion of the oncologist was sought; no chemotherapy / radiotherapy was suggested. They advised periodic monitoring of the patient clinically, radiologically and endoscopically for any recurrence of the tumor.

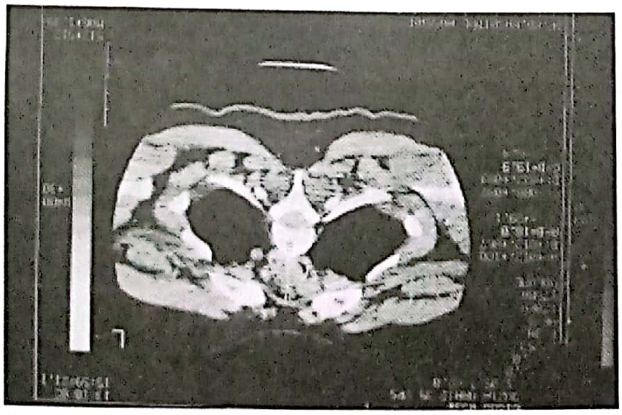

Fig-1 : *HRCT scan of chest*

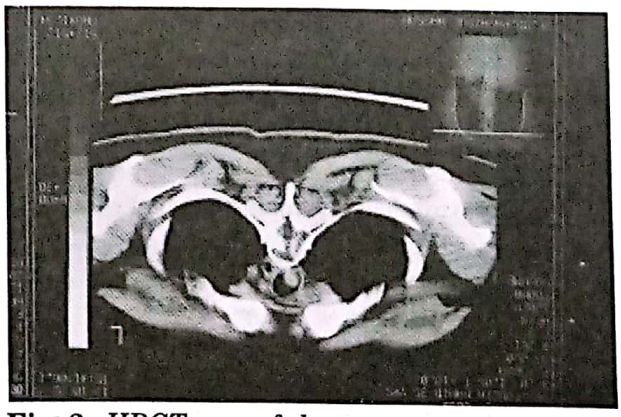

Fig-2 : *HRCT scan of chest*

Post-operative high resolution CT scan (HRCT) of chest was advised which revealed no abnormality.

After 6 (six) weeks, the patient was observed and found healthy without any further attack of breathlessness and advised again to report after three months.

Surgical Procedure

After induction, anaesthesia was maintained with laryngeal mask without endotracheal intervention. Right posterolateral thoracotomy was done through the uper border of 5th rib. The basal part of the right lower lobe was found to be consolidated. Right principal bronchus and trachea was mobilized. A vertical incision about 3cm in length was made from lower part of trachea, extending upto the beginning of right principal bronchus. The tumor was identified and thick purulent exudate was found coming from both right & left principal bronchus, which was thoroughly sucked out. Immediately, left lung was intubated and anaesthesia was shifted from laryngeal mask to that tube and one lung anesthesia (left lung) was maintained. The Right lung got collapsed and thereby proper field was obtained for surgical procedure. The tumor was identified and HRCT findings were confirmed. The tumor bled on touch and its base extended from lower tracheal ring to adjacent areas. It was excised by wedge resection. The area bled profusely which was controlled with diathermy coagulation and palpated for any residual tumor. After ensuring absence of residual tumor and absolute homeostasis, anesthesia was again shifted to laryngeal mask. The tracheal wall was closed in usual way after bronchial toileting of both sides. Subsequently, expansion of the right lung was ensured and the consolidated area assumed more or less the same consistency. Then the chest was closed in the usual way leaving a water-seal drainage tube to the pleural cavity. Post-operatively the patient was observed for any respiratory distress or haemoptysis but the period was uneventful. The histopathological diagnosis was carcinoid tumor.

Case Report: 3

Severe persistent asthma is a serious and complicated type of asthma and sometimes it mimics other obstructive airway disease.

We present a 8 year-old student who developed high grade fever, breathlessness and productive cough for 3 days. Despite the bronchodilators and analgesics, he developed severe respiratory distress and was admitted to Dhaka Shishu Hospital where he was treated with nebulized Salbutamol, injectable steroid etc. After returning home, he again developed severe breathlessness and was brought to a physician where he was diagnosed as a case of bronchial asthma and prescribed Ketotifen and bronchodilators etc. to which he did not respond; subsequently he was taken to a cardiologist. The cardiologist found nothing wrong with the patient and referred him to an ENT specialist. The ENT surgeon thought it might be due to a foreign body in the trachea and advised to consult a thoracic surgeon for possible bronchoscopy. But his parents refused to undergo bronchoscopy. He was again taken to a cardiologist where an Echocardiogram was performed. The report was normal. The parents then brought the child to a homoeopath doctor but with no improvement. The child came across various physicians, pediatricians, chest specialists and other doctors in Bangladesh.

Then the patient was taken to a hospital in Kolkata. Hematological examinations, chest X-Ray, sputum for AFB and other relevant investigations were done, but all were normal. Patient was getting bronchodilator and nasal decongestant from India with little improvement. Fibreoptic laryngoscopy was performed with normal findings. Ultimately, he was presumed to be a case of obstructive sleep apnoea syndrome. His sleep pattern was recorded by a paediatrician of Bangladesh and the record was sent to Australia. But the diagnostic dilemma and the sufferings persisted.

In due course, he was referred to the National Asthma Centre, NIDCH, Dhaka: HRCT scan of the chest was done to rule out any endobronchial lesion and the report was normal. Sputum Eosinophil was scanty. Spirometry showed severe obstructive and restrictive defect. Therapeutic trial was started with Seretide accuhaler 50/250 and Montelukast 5 mg with an assumption of severe persistent asthma and the patient responded to treatment significantly.

Discussion

Leomyoma may present as a central bronchial or tracheal lesion, often polypoid or pedunculated and may present with pseudo- asthmatic breathlessness, cough, occasional wheeze and haemoptysis[4.5.6]. It tends to occur in children and young adults. The first patient reported with the same presentation. The very rare leiomyoma originates in airway smooth muscle. The clinical picture depends primarily on the location of the lesion.

HRCT is highly diagnostic and confirmatory in these rare cases. Resection with end-to-end tracheal anastomosis or with tracheal reconstruction with myocutaneous flaps has been associated with reported cures[1,3,7].

There have been many reports of pulmonary leiomyomata occurring in women with similar tumours of the uterus. Whether or not the pulmonary lesions represent "benign metastases" are subject to controversy, as low-grade smooth muscle metastases and primary leiomyomas are difficult to distinguish[8]. Nevertheless, in women with pulmonary leiomyomata, a careful pelvic examination is recommended[9]. In our patient, pelvic examination revealed no abnormal findings.

The bronchial carcinoids usually arise from a main or segmental bronchus and can often be seen bronchospopically as an intra-luminal tumour, generally covered with intact epithelium[10]. They may also arise from the trachea. It is a neoplasm of bronchial endocrine or APUD cell derived from the primitive gut. As foregut derivatives, bronchial carcinoids may produce ACTH but do not usually produce 5-Hydroxy tryptamine that is seen in midgut or hindgut carcinoid tumours[12]. They are predominantly slow growing endobronchial lesions. They occur equally in either sex usually at younger age than lung cancer. Patient may have recurrent haemoptysis, chronic cough, obstruction with atelectasis, lobar collapse, recurrent pneumonia, unilateral monophonic wheeze and signs of obstructive emphysema[1,2,9].

Surgical excision is the primary treatment for all types of bronchial adenomas, including carcinoid. Surgical excision is the treatment of choice for pulmonary carcinoid tumor. Extent of surgery is determined at operation and should be as conservative as possible. Often bronchotomy with local excision, sleeve resection, segmental resection or lobectomy is sufficient. 5-years survival rates after surgical resection are 95%, decreasing to 70% if regional nodes are involved[2,11,12].

On the other hand, severe persistent asthma may mimic endotracheal/ endobronchial lesions or obstructive sleep apnoea syndrome as demonstrated in our last case.

Conclusions

1. Tumors of the airways may present as asthma.
2. High resolution CT scan can help in identifying such tumors.
3. A coordinated team approach should be adopted in such situations. The team should consist of pulmonologists, thoracic surgeons, pathologists, radiologists, oncologists and ENT surgeons.
4. Surgery is very useful for the management of these patients.

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Late Presentation of ASD With Multiple -Valvular Disease -A Case Report

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Abstract:

ASD is a common heart disease. Symptomatic ASD is uncommon in infancy. No symptom or mild symptoms are common in the first three decades of life, Patients usually present themselves in their middle age. The complications of ASD are pulmonary hypertension, congestive heart failure and arrhythmia. A 65 yrs old male presented with dyspnoea and mild chest pain. He also complained of non productive cough and feeling of upper abdominal mass. General examination revealed mild oedema, raised jugular venous pulse, clubbing and cyanosis. Systemic examination showed bilateral basal creps, wide fixed splitting of 2nd heart sound, systolic flow murmur over pulmonary area and firm well defined lumpy filling over epigastric region. His chest skiagram showed increased cardiothoracic ratio, dilated central pulmonary vessels with peripheral pruning. Features were in favor of atrial septal defect. (ASD) with Eissenmenger reaction. Ultrasonography of abdominal organs revealed enlarged liver with dilated hepatic veins (congestive hepatomegaly), mild ascites and right sided small pleural effusion. ECG was reported as: atrial fibrillation with premature ventricular contraction. Colour doppler echocardiography reported as moderate ASD(secundum) with minimal right to left shunt, dilatation of RV, RA and LA, aortic sclerosis, good LV systolic function, moderate MS, grade 2/4 MR and 3/4 TR. Finally the case was diagnosed as ASD with reverse shunt, multiple vulvular disease and cardiac failure.

[Chest & Heart Journal 2005; 29(2) : 139-140]

Introduction:

An atrial septal defect (ASD) is a through and through communication between the atria at the septal level. It is the commonest congenital heart lesion encountered in adults. It ranks fifth congenital heart disease at birth. Incidence of ASD is approximately 1 in 13,000 children and is found to occur as isolated lesion in 7 to 15% of children with congenital heart disease.' Secundum and sinus venosus defects have higher incidence in female (female: male - 2:1). Ostium primum defect has equal sex incedence.

Chest x-ray and colour flow echocardiography are very much helpful in the diagnosis of such cases.

Case note:

A 65yrs old male presented with dyspnoea and mild chest pain for last one year. Dyspnoea increased after exertion and relieved at rest. He also complained of non productive cough and feeling of upper abdominal mass for the last one year. Mass like feeling became prominent after meal. General examination revealed mild oedema, raised jugular venous pulse, clubbing and cyanosis. Systemic examination showed bilateral basal creps, wide fixed splitting of 2nd heart sound, systolic flow murmur over pulmonary area and firm well defined lumpy filling over epigastric region. His serum bilirubin was 2mg/dl. He was referred to Radiologist for x-ray chest PA view and ultrasonography of abdominal organs. Chest skiagram showed increased cardiothoracic ratio, dilated central pulmonary vessels with peripheral pruning. Features were in favor of atrial septal defect(ASD) with Eissenmenger reaction. Ultrasonography of abdominal organs revealed enlarged liver with

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dilated hepatic veins(congestive hepatomegaly), mild ascites and right sided small pleural effusion. No mass lesion was detected. Feeling of mass was due to enlarged left lobe of the liver. ECG was reported as: atrial fibrillation with premature ventricular contraction.

Colour doppler echocardiography was also performed and reported as moderate ASD (secundum) with minimal right to left shunt, dilatation of RV,RA and LA, aortic sclerosis, good LV systolic function, moderate MS, grade 2/4 MR and 3/4 TR.

Finally the case was diagnosed as ASD with reverse shunt, multiple vulvular disease and cardiac failure.

Discussion:

ASD is a common heart disease. Frequency of ASD is 3.2 per 1000 live birth. ASD accounts for 7% of all congenital heart diseases and 10% in those surviving beyond first year of life. Females are commonly affected. Female to male ratio varies from 2:1 to 4:1. Incidence of ostium primum type of ASD is 15 to 20%, sinus venosus type is 2 to 6% and coronary sinus type is less than 3 to 4%. The commonest age of presentation is second and third decade. Patients with ASD may live upto 09 decades. Oldest patient reported with this disorder was 94 years of age. Symptomatic ASD is uncommon in infancy. Symptom usually is associated with other conditions. No symptom or mild symptoms are common in the first three decades of life, patients usually present themselves in their middle age. The complications of ASD are pulmonary hypertension, congestive heart failure and arrhythmia. Besterman reported pulmonary hypertension in 16% of his patients. It is more frequent in primum defect with common AV canal and in Lutembacher's syndrome than in ordinary fossa ovalis defect. Infective endocarditis is uncommon, if it occurs usually indicates ostium primum defect. The case presented here was a male of 60yrs old with ostium secundum defect[3].

Conclusion:

ASD usually goes unrecognized for years even decades because symptoms are usually absent with subtle physical signs. Heart failure is rare in child but is common in 4th or 5^{th} decades and is usually associated with arrhythmia. Chest skiagram, ECG, colour doppler echocardieography and abdominal sonography can help in the diagnosis of ASD along with its complications.

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