ORIGINAL RESEARCH PAPER | Physiology | Volume : 6 | Issue : 11 | November 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

Effect of Aerobic Excersice and Other Variables on Bronchial Hyper Reactivity in Apprantly Healthy Young Adolescents of Central India

KEYWORDS: EIBHR- Excersice induced bronchial hyper reactivity, BMI- Body mass index, PEFR- Peak expiratory flow rate

PRACHI ARYA
Department of Physiology, G.R. Medical College and assoc. Jaya Arogya Hospital, Gwalior

MILIND SHIRALKAR
Department of Physiology, G.R. Medical College and assoc. Jaya Arogya Hospital, Gwalior

ABSTRACT *Exercise inducible bronchial hyperactivity (EIBHR) is a reversible airway narrowing to wide variety of stimuli like physical exertion and characterized by repeated episodes of dyspnoea, wheezing, chest tightness, cough and phlegm. EIBHR can occur in healthy people who do not have chronic asthma. Exercise is the only stimulus for their asthma symptoms which most likely reflects a different pathophysiologic event than chronic asthma.*

A prospective observational study conducted in the Department of Physiology, G.R. Medical College, Gwalior. Apparently healthy teenage medical students aged 17-19 yrs were studied to assess lung function parameters and prevalence of unrecognised EIBHR. General information obtained from all the subjects with special reference to factors affecting lung functions such as history of parental smoking, family history of asthma/atopy and personal history of smoking and atopy.

PEFR and baseline lung functions were evaluated as per standard technique thereafter adolescent were instructed to undertake aerobic exercise on cycle ergometer for 6-8 minutes. Exercise intensity was kept at a level so as to achieve 70-80% of maximum HR (220 age in years). PEFR and spirometry was recorded at 5 minute interval for next 30 minutes. 15% or more fall in PEFR from baseline was defined as exercise induced bronchial hyper reactivity (EIBHR).

An overall prevalence of 14% exercise induced bronchial hyperreactivity (EIBHR) was found in this study. This increased prevalence of EIBHR was found in urban dwellers and male sex. Family history of atopy was found to have significant negative correlation with baseline vital capacity, PEFR and FEV1.

BMI had positive correlation with PEFR only particularly in male sex. Other family and personal variables did not have significant difference on lung function parameter. Body surface area corrected baseline lung function parameters was statistically highly significant in both sex in without EIBHR (p=0.008, p\0.001 and =0.0001 for PEFR, FEV and FEV1 respectively) but was not statistically significant in both sex with EIBHR.

INTRODUCTION

Exercise inducible bronchial hyperactivity (EIBHR) is a reversible airway narrowing to wide variety of stimuli like physical exertion and characterised by repeated episodes of dyspnoea, wheezing, chest tightness, cough and phlegm[1].EIBHR can occur in healthy people who do not have chronic asthma. Exercise is the only stimulus for their asthma symptoms which most likely reflects a different pathophysiologic event than chronic asthma.

EIBHR can also occur in people who have chronic asthma and may not be aware that their symptoms during exercise are not a manifestation of asthma as the treatment for EIHBR and chronic asthma are not different[2].

Pathophysiology of EIBHR depends upon water loss theory which states that exercise causes decrease airway humidity through more rapid ventilation, causing increased mucosal osmolarity which further results in release of mediators and bronchoconstriction[3,4]. Another is heat exchange theory which states that once exercise ceases, the bronchial vasculature dilates to rewarm the airways, this rebound hyperaemia narrows the airways[5].

EIBHR occurs in almost 90% of people who have chronic asthma and 40% of individuals who have allergic rhinitis. Prevalence among children is 7% of the general population[6]. Prevalence of EIBHR in athletes is 10-15%. In patients of EIBHR after 6-8 min. of exercise, bronchoconstriction occurs (immediate response) instead of bronchodilatation and after 6-8 hrs. mast cell mediators are released which causes further bronchoconstriction[7].

The purpose to diagnose EIBHR is to diagnose excersice induced asthma (EIA), to treat EIA and maximise patients ability to participate actively in aerobic activity, work related activity and increase sense of self work socialization[8].

Pathophysiology of Bronchial hyperactivity: It is attributed to one or more of the following abnormalities -

1. Defect in the airway,
2. Abnormal neural control of the airway
3. And bronchial inflammation.

It is suggested that imbalance between excitatory (Cholinergic alpha adrenergic and non- Cholinergic) and inhibition mechanism (B adrenergic & non adrenergic) increase bronchial reactiveness [9]. Bronchoconstriction results from increase cholinergic activity causing bronchial smooth muscle to contract and bronchodilation results from non- adrenergic system activation and endogenous catechominess acting on Beta adrenergic receptors as well as Prostaglandin E2. VIP (Vasoactive intestinal peptides) relax smooth muscle of bronchi while substance P increase smooth muscle tone, mucus hypersection and microvascular leakage[10]. Airway Inflammation is considered to be the basic pathology in bronchial hyperreactivity. This is initiated by degranulation of mast cells. Newly synthesized and stored mediators (histamine, Leukotrienes, Prostaglandin,

Volume : 6 | Issue : 11 | November 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

Platelet activating factor and bradykinin) are released from degranulated mast cells following non-specific stimulation and binding of allergens to specific mast cell associated immunoglobulin IgE, which initiated bronchoconstriction , mucosal edema and immune response . Inflammatory mediators also influence reactivity via neural mechanisms. Reduced adrenaline production and reduced no. of Beta receptor contributed to pathogenesis of exercise induced Asthma (EIA).

Factors contributing to EIA are -

Genetic: - The frequent clinical observations that BHR occur in successive generation suggest that there may be genetic determination of BHR. Bronchial liability and atopy was demonstrated to be more frequent in families of infants with wheezy bronchitis than in families of normal children. Sputum Eosinophils were shown to be significantly increased in the group with exercise induced asthma. (8.1 +/- 13.9% to 18.3 +/- 20.2%, P= 0.001) as compared in the control group (0.9 +/- 9% to 1.5 +/- 15%)[11] .BHR has been shown to have multifactorial origin, allergies contributing 55% infection 23% and functional 4%[12].

Exercise:- Exercise induced airway narrowing is a common feature of children who develop bronchial hypersensitivity with prevalence reported to be around 6.9%[13] . The response to exercise challenge however, varied according to other factors associated eg. Allergen challenge, baseline bronchial reactivity and temperature. Exercise is commonly associated with asthma with a frequency of around 82-90 %. While accompanies allergic rhinitis in 40-50% , atheletes 14% and general population 12%[14].

Allergens - It is speculated that BHR develops when airway of sensitized subjects are repeatedly exposed with allergens. Common allergens being house dustmite (Dermatophagoides pteromyssious &D. favinae), animals fur of cat, dog, horse, pollens of grass and moulds (atlernaria teanis and aspergillus fumigatus)

Infection- There are also evidence that viral infection in childhood may predispose to the development of bronchial hyperreactivity in later life.[15] Prevalence of BHR in Fijian children was found to be consistent with greater burden of respiratory infection (15.6%) in a study by Flynn et al[16]. Both respiratory syncitial virus infection and croup may be followed by persistent wheezing and hyperreactivity . Such infections also preceed sensitization with common allergens in atopic children. The virus usually responsible are influenza, rhinovirus, Respiratorysyntitial Virus, bacteria mycoplasma pneumonia. The effects involved is the production of viral specific IgE, upregulation of leukocyte inflammatory activity, altered autonomic nervous system activation and damage to the airway epithelium[17].

Air Pollution – It may also provoke exacerbations of BHR. Higher prevalence of BHR has been reported among subjects living in the industrial area than agriculture area (i.e. 57.26% & 41.4%) in a study by forasttere et al[18]. Also exposure to low concentration of airway irritants during early childhood is associated with an increase prevalence of BHR in school children[19].

Smoking- Parental smoking one of the important etiological factor in symptomatology of children with bronchial hyperactivity was evident in 74% subjects in a study by Roizin et al[20].

An increase in Bronchial hyperreactivity after cigarette smoke challenge in asymptomatic subjects suggest that prolonged subclinical airway inflammation can occur in the absence of demonstratable change in airway caliber on exposure to environment tobacco smoke (ETS).[21.]

Psychological factor- Events such as shock, bereavement or excitement may trigger symptoms. The psychological effects on airway reactivity are presumably mediated by autonomic nervous system.

Gastro esophageal factor – It has been postulated that repeated episodes of microaspiration of gastric juice into trachea triggers bronchospasm.

Dietary habits- Atopic BHR subject may occasionally notice that their symptoms are provoked by certain food or drinks. The foods most suspectable are cold substances, milk, eggs, fish, cereal, nuts and chocolates. Food induced allergic reaction can increase airway reactivity.

Climate- Reactivity to perineal allergens is predictive of bronchial responsiveness in both winter and spring22.

Screening protocol for BHR - An account of episodic wheezy breathlessness or cases of cough, chronic, episodic, nocturnal, persistant, laugh, or exercise induced cough, interspersed with periods of normalilty is sufficient evidence on which to suspect BHR. Hopp RJ observed that airway responsiveness was not necessarily associated with questionnaire based respiratory symptomatology. Assessment of pulmonary function before and after administration of an aerosol bronchodilatation is valuable. An increase of atleast 10% in PEFR or FEV, after aerosol therapy is indicative of reversibility of airway obstruction and strongly suggestive of BHR.

MATERIAL AND METHODS

The study was undertaken in the Department of Physiology, G.R. Medical College & Jaya Arogya Hospital, Lashkar, Gwalior. It comprised of 50 apparently healthy 17-19 years old medical students who agreed to participate in the study. Students were enrolled after taking informed consent. Those with history of asthma like symptoms or who had undertaken treatment or were taking treatment for asthma were excluded.

Of the 50 students who participated, 31 were boys and 19 girls.. The aims and study protocol was explained to them. Information regarding family history, personal habits and allergies was elicited & recorded. Height & Weight was taken as per standard technique and these parameters were used to calculate BMI (Body mass index) and BSA (Body surface area).

General Information i.e. Age, Sex, Place of Living – rural urban, family history of atopy, Asthma and smoking, personal history of atopy, asthma and smoking were obtained and recorded in a pretested proforma. Detailed physical examination was done to rule out clinical illness.

PEFR was recorded with the help of wrightrs peak flow meter. Average of 3 readings was taken as baseline PEFR. Lung function parameters was assessed by spiroexcel (PC

Volume : 6 | Issue : 11 | November 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

based spirometer), Medicaid systems ISO 9001:2000 Company. Baseline measurement was taken after the student had become conversant with the technique of blowing into the spirometer . After recording baseline spirogram, the students were requested to undertake aerobic exercise on cycle ergometer at an intensity to achieve 70-80% of max. Heart rate in 2 minute and then to maintain same level of activity for next 6 to 8 minutes.

PEFR, FEV_1, FEV_1 / FVC, FEV_{25-75} calculated at baseline. The exercise challenge test was administered on cycle ergometer at ambient temperature between 25-28°C in the laboratory. After exercise, PEFR & spirometry was recorded immediately and at 5 minute interval for next 30 minutes.

Any drop from baseline in PEFR greater than or equal to 15% was taken as evidence of EIBHR.

The procedures were in accordance with the ethical standard of experimentation of the Institution and with the Helsinki Declaration of 1975.

STATISTICAL ANALYSIS

The data collected was complied and subjected to statistical analysis using SSPS 10 software. Values of baseline lung function parameters were calculated in the form of per square meter body surface area and intra group comparison was done between boys and girls and between those with EIBHR and without EIBHR [Table 12].

The baseline data was categorised on the basis of age, sex and height .

Impact of personal, physical and family variables on baseline parameter was assessed using Pearson correlation and X^2 analysis.

DISCUSSION

A prospective observational study was conducted in the Department of Physiology, G.R. Medical College, Gwalior to assess lung function parameters and prevalence of unrecognized EIBHR in apparently healthy teenage medical students. Out of 50 adolescents of 1st year MBBS students enrolled for the study. Out of these, 31 were males (62%) and 19 subjects were females (38%)[Table 1] . 9 adolescents i.e. 5 boys and 4 girls (Table 1) had BMI of < 18 kg/m2 and 40 subjects (25 boys and 15 girls) had BMI in 18 to 24 kg/m2 range [Table 4]. One boy had BMI in overweight category i.e. 25 to 29.9 kg/m2. Body surface area of male adolescent ranged from 1.40-1.9 persqm and in female adolescent it ranged from 1.30-1.5 persqm [Table 6]. History of parental smoking was positive in 17 adolescents and family history of atopy and bronchial asthma was positive in 15 and 5 adolescent respectively. In all 17 adolescent gave history suggestive of atopy and 2 gave history of smoking. Parental smoking is reported in 70% of subjects with BHR and parental history has been concluded to be one of the important etiological factor in the symptomatology of children with BHR.

Positive EIBHR was defined as 15% or greater disease in PEFR from baseline at one or more of 5 minutes time interval recordings after exercise. 7 adolescents had 15% or greater decline in PEFR. Thus, the prevalence of unrecognised EIBHR was 14% in the present study [Table 8]. Family history of asthma was positive in two of these seven adolescents and family history of atopy was negative in all the 7 adolescents.

Parental history of smoking was positive in 1 and personal history of smoking was negative in all.

Personal history of atopy was present in 2 cases. All 7 adolescents had BMI in the normal range i.e. 18-24.9 kg/m2[Table 5,Fig.2].

Thus, prevalence of EIBHR was 16% in boys and 10.5% in girls. 1 boy and 1 girl came from rural area [Table 2,Fig1]. The maximum decline in PEFR was 24.5% from baseline. In 5 adolescents, the maximum decline in PEFR was at 15 min after exercise and in one it was at 5 min and in another at 20 min after exercise. In these 2 adolescents decline in PEFR was 10% and 3% from the baseline at 15 min time interval.

The effect of short term exercise on pulse rate and peak expiratory flow rate in healthy Libyan school children was studied by F. Sagher et al, it was found that exercise markedly increased the pulse rate and markedly reduced peak expiratory flow rate. In 10% children, reduction in PER was > or = 15% from the baseline [Table 8].

In our study, the maximum decline in FEV1 ranged from 19.64 to 31.7% from baseline. In 4 adolescent maximum decline occurred at 15 minutes, in 2 at 20 min and in one at 5 minute after exercise (Table 9).

It was also reported that maximum fall in FEV1 after 85% exercise load was 8.84% vs 25.11% after 95% ($p<0.001$). Fall of > or = 10% in FEV1 was seen in 40% (9 subjects) of the subjects after 85% exercise load while it became 100% (i.e. in all 20 subjects) after 95% exercise load. The baseline FEV1/VC ratio in these 07 adolescents ranged from 80-90%.

In our study, FEV1/VC ratio showed maximum decline of 7.6%-19.5% in these 7 adolescent. In 1 adolescent, the maximum decline in FEV1/VC ratio was at 30 min after exercise, in 2(two) adolescent at 20 minutes and 15 minutes after exercise, in other two at 20 minutes only and in one each at 15 minutes and 5 minutes after exercise (Table 10).

In a study of falls in the ratio of forced expiratory volume in one second (FEV1) to vital capacityi.e. FEV1/VC ratio was significant at 5 and 15 minute after exercise in obese boys while in controls it was significant only at 5 minutes after exercise. Also the pulmonary function changes after the exercise had had significant negative correlation with body mass index and subscapular and biceps skin folds.

The maximum decline in FEV 25-75 ranged from 15.6% to 26.4% of baseline (Table 11). In 3 adolescent the maximum decline was at 15 minutes and in one, it was at 20 minutes while in other two it was at 25 minutes after exercise and in one at 30 minutes after exercise. Both of these adolescents also had comparable degree of decline in FEV25-75 at 15 minutes interval.

The impact of family variables (i.e. parental smoking, family history of atopy) and personal variables on baseline lung function parameter i.e. PEFR, VC, and FEV1 is shown in Table 13. Family history of atopy was found to have significant negative correlation with the baseline vital capacity, FEV1 and peak expiratory flow rate particulary in females. BMI had significant positive correlation with PEFR particularly in male sex [Table 13].

Volume : 6 | Issue : 11 | November 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

OBSERVATION

Table – 1

Showing distribution of study sample by sex

Sex	No. of sample	Percentage (n=50)
Male	31	62%
Female	19	38%

Table 2

Showing distribution of adolescents by place of living

Residence	Male n=31	Female n=19	Total n= 50
Urban (n = 38)	22 (57.89%)	16 (42.10%)	38 (76%)
Rural (n = 12)	9 (75%)	3 (25%)	12 (24%)

Majority of the study subject came from urban area (76%) and most of the male sex (57.89%).

Table – 3

Showing distribution of adolescents with EIBHR by place of living

Residence	Male (n=5)	Female (n=2)	Total (n=50)
Urban (n=5)	4 (80%)	1 (50%)	5 (71.42%)
Rural (n=2)	1 (20%)	1 (50%)	2 (28.57%)

Table – 4

Showing sex wise distribution of BMI in adolescents

BMI (kg/m^2)	Male N = 31	Female N = 19	Total (n=50)
< 18	5 (16.12%)	4 (21.05)	9 (18%)
18-24.9	25 (80.6%)	15 (78.94%)	40 (80%)
25-29.9	1 (3.22%)	0	1(2%)
>30	0	0	0

Table – 5

Showing sex wise distribution of BMI in adolescents with EIBHR

BMI (kg/m^2)	Male (n=5)	Female (n=2)	Total (n=7)
< 18	0	0	0
18-24.9	5 (100%)	2 (100%)	7 (100%)
> 25	0	0	0

All seven adolescents with EIBHR had normal range of BMI.

Table – 6

Showing sex wise distribution of body surface area in adolescents

BSA (per sqm)	Male (n=31)	Female (n=19)	Percentage (n=50)
< 1.30	0	1 (5.26%)	1 (2%)
1.30 – 1.39	0	13 (68.42%)	13 (26%)
1.40-1.49	2 (6.45%)	3(15.78%)	5(10%)
1.50 – 1.59	10 (32.25%)	2 (10.52%)	12 (24%)
1.60 – 1.69	8 (25.80%)	0	8 (16%)
1.70 – 1.79	8 (25.80%)	0	8 (16%)
1.80 – 1.89	2 (6.45%)	0	2 (4%)
> 1.90	1 (3.25%)	0	1 (2%)

Table – 8

Showing change in PEFR during exercise in adolescents with EIBHR

SN	Peak expiratory flow rate (PEFR) (L/m)						
	0 min (100%)	5 min (%⇩)	10 min (%⇩)	15 min (%⇩)	20 min (%⇩)	25 min (%⇩)	30 min (%⇩)
1.	525	465 (11.42)	445 (15.2)	440* (16.19)	480 (8.57)	795 (5.71)	500 (4.7)
2	550	430 (21.8)	440 (20)	415* (24.54)	420 (23.63)	430 (21.8)	440 (20)
3	600	540 (10)	525 (12.5)	510* (15)	515 (14.16)	535 (10.83)	540 (10)
4	390	330* (15.38)	340 (12.8)	350 (10.25)	330 (15.38)	340 (12.8)	360 (7.69)
5	400	360 (10)	352 (12)	340* (15)	350 (12.5)	360 (10)	350 (12.5)
6.	300	255 (15)	265 (11.6)	275 (8.3)	250* (16.66)	265 (11.6)	255* (15)
7	550	495 (10)	485 (11.8)	465* (15.45)	478 (13.89)	465 (15.45)	495 (10)

* Percent decline from baseline.

Table – 10

Showing change in FEV1/VC ratio during exercise in adolescents with EIBHR

SN	FEV_1 /VC %						
	0 min (100%)	5 min (%⇩)	10 min (%⇩)	15 min (%⇩)	20 min (%⇩)	25 min (%⇩)	30 min (%⇩)
1.	83.9	75 (10.6)	77.8 (7.27)	73.8 (12.03)	67.5* (19.5)	77.9 (7.15)	69 (14.9)
2	80.9	77.9 (3.7)	75.6 (6.55)	75 (7.29)	77.9 (3.7)	78 (3.58)	66* (18.41)
3	80	77.8 (2.75)	74.8 (6.5)	69.1* (13.62)	75.1 (5.25)	79.8 (0.25)	73.9 (7.62)
4	90	74.9* (16.7)	74.95 (16.7)	75.9 (15.66)	75 (16.66)	77.9 (13.4)	75.1 (16.5)
5	90	75.9 (15.9)	75 (16.6)	74.9* (16.77)	74.9* (16.77	75 (16.6)	75.94 (15.6)
6.	84	75.9 (9.6)	75.9 (9.6)	75* (10.7)	75* (10.7)	77 (8.3)	77.99 (7.15)
7	83.9	77.9 (7.15)	75 (10.6)	75.9 (9.53)	73.98* (11.82)	77.7 (7.3)	75 (10.6)

* Percent decline from baseline.

Volume : 6 | Issue : 11 | November 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

Table – 11

Showing change in FEV_{25-75} during exercise in adolescents with EIBHR

SN	FEV_{25-75}						
	0 min (100%)	5 min (%⇩)	10 min (%⇩)	15 min (%⇩)	20 min (%⇩)	25 min (%⇩)	30 min (%⇩)
1.	1420	1100 (21.42)	1134 (18.92)	1050 (26.05)	1155 (17.5)	1160 (17.1)	1030* (26.4)
2	1600	1415 (11.56)	1325 (17.18)	1195* (25.3)	1285 (19.68)	1365 (14.68)	1400 (12.5)
3	1700	1385 (18.5)	1315 (22.6)	1225* (27.9)	1390 (18.23)	1300 (23.52)	1490 (12.35)
4	1060	875 (17.45)	895 (17.92)	810 (23.5)	857 (19.15)	763* (28.0)	860 (18.86)
5	1640	1140 (30.48)	1140 (30.48)	1128 (31.21)	1090* (33.53)	1140 (30.48)	1138 (30.6)
6.	1040	895 (13.94)	890 (14.42)	870 (16.34)	905 (12.98)	850* (18.26)	900 (13.46)
7	1760	1520 (13.63)	1520 (13.63)	1485* (15.62)	1390 (21.02)	1405 (20.17)	1536 (12.72)

* Percent decline from baseline.

Table 12 Showing sex wise T test values of baseline lung function parameters (PEFR, FVC, FEV_1) of adolescents in with EIBHR and without EIBHR

Factor	Lung function parameters	T – test	d.F	P values
Without EIBHR	PEFR	2.804	41	0.008 (H.S)
Male Vs Female	FVC	5.302	41	0.0001(H.S)
	FEV1	5.744	41	0.0001 (H.S)
With EIBHR	PEFR	1.815	5	0.129 (N.S)
Male Vs Female	FVC	1.910	5	0.114 (N.S)
	FEV1	1.759	5	0.139(N.S)

HS - Highly significant

NS - Not significant

Table 13 SHOWING correlation of lung function parameters with family and personal variables

		PS	VC BASE	PEFR BASE	FAMILY	ATOPY1	BMI	SEX	FEV1 BASE
PS	Pearson Correlation	1.000	.058	0.72	-.250	-.426	.128	-133	.052
	Sig. (2-tailed)		.824	.782	.369	.088	.626	.610	.844
	N	17	17	17	15	17	17	17	17
VC_BASE	Pearson Correlation	.058	1.000	.719**	-.614**	.063	.060	-	.834**
	Sig. (2-tailed)	.824		.000	.009	.663	.681	6.36**	.000
	N	17	50	50	17	50	50	.000	50
								50	
PEFR_BASE	Pearson Correlation	.072	719**	1.000	-.600*	.052	.324*	-771**	.696**
	Sig. (2-tailed)	.782	.000		.011	.720	.022	.000	.000
	N	17	50	50	17	50	50	50	50
FAMILY	Pearson Correlation	-250	-614**	-.600*	1.000	-.426	-	.604*	-
	Sig. (2-tailed)	.369	.009	.011		.088	.049	.010	.613**
	N	15	17	17	17	17	.851	17	.009
							17		17
ATOPY1	Pearson Correlation	-	.063	.052	-426	1.000	105	124	-.041
	Sig. (2-tailed)	.426.08	.663	.720	.088		.466	.392	.779
	N	8	50	50	177	50	50	50	50
		17							

Table 13 (cont.)

BMI	Pearson Correlation	.128	.060	.324*	-.049	.105	1.00	-.284*	.108
	Sig. (2-tailed)	626	.681	.022	.851	.466	0	.046	.456
	N	17	50	50	17	50		50	50
							50		
SEX	Pearson Correlation	-.133	-636**	-771**	604*	.124	-	1.000	-
	Sig. (2-tailed)	610	.000	.000	.010	.392	.284		.789**
	N	17	50	50	17	50	*	50	.000
							.046		50
							50		
FEV1_BASE	Pearson Correlation	.052	.834**	.696**	-613**	-.041	.108	-789**	1.000
	Sig. (2-tailed)	.844	.000	.000	.009	.779	.456	.000	
	N	17	50	50	17	50	50	50	50

** Correlation is significant at the 0.01 level (2-tailed)

* Correlation is significant at the 0.05 level (2-tailed).

FIG: 1 SHOWING DISTRIBUTION OF ADOLESCENTS WITH EIBHR BY PLACE OF LIVING

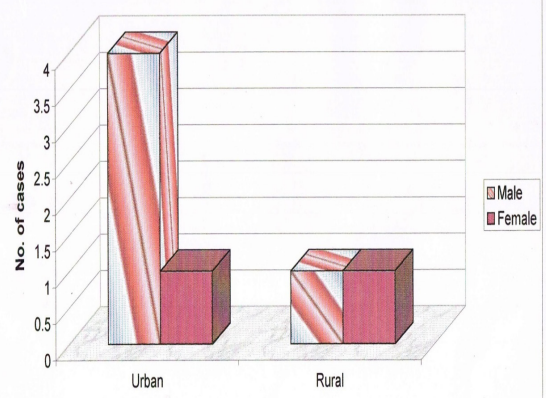
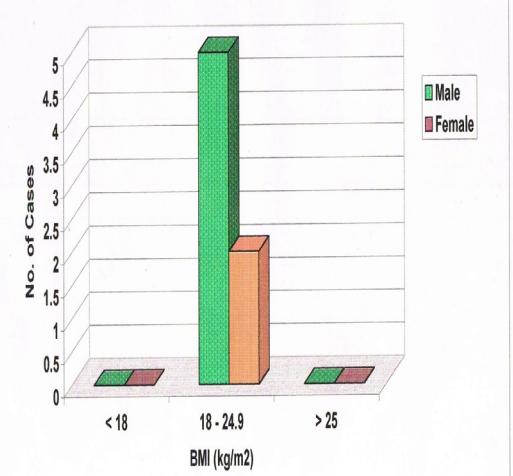
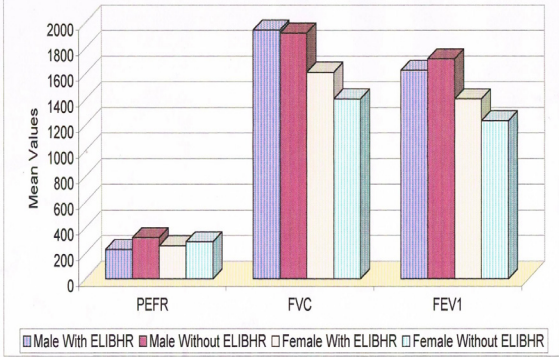


FIG : 2 SHOWING SEX WISE DISTRIBUTION OF BMI IN ADOLESCENTS WITH EIBHR



Volume : 6 | Issue : 11 | November 2016 | ISSN - 2249-555X | IF : 3.919 | IC Value : 74.50

FIG :3 SHOWING SEX WISE DISTRIBUTION OF BODY SURFACE AREA CORRECTED LUNG FUNCTION PARAMETERS (PEFR, FVC, FEV1) IN ADOLESCENTS WITH AND WITHOUT EIBHR.



RESULT

An overall prevalence of 14% exercise induced bronchial hyperreactivity (EIBHR) was found in this study. This increased prevalence of EIBHR was found in urban dwellers and male sex. Maximum percent decrease in lung function parameters were found as follows:-

(i) In PEFR, in ranged from 15%-24.5% from baseline.

(ii) Maximum percent decrease from baseline in fEV1, FEV1/VC% and FEV25-75 ranged from 19.64%-31.7%, 7.8-19.6% and 15.6% -26.05% respectively.

It was found that family history of atopy had significant negative correlation with the baseline vital capacity, FEV1 and peak expiratory flow rate. BMI had significant positive correlation with PEFR only. Parental smoking and personal history of atopy lacked significant correlation with any of the baseline lung function parameters.

Body surface area corrected baseline lung function parameter was statistically highly significant in both sexes without EIHBR (p=0.008/, p=0.001, p=0.0001 of PEFR, FVC & FEV1 respectively), but not statistically significant in both sexes with EIBHR. χ^2 test was not statistically significant in any groups.

CONCLUSION

An overall prevalence of 14% exercise induced bronchial hyperreactivity (EIBHR) was found in this study. This increased prevalence of EIBHR was found in urban dwellers and male sex. Maximum percent decrease in lung function parameters were found as follows:-

(i) In PEFR, in ranged from 15%-24.5% from baseline.

(ii) Maximum percent decrease from baseline in fEV1, FEV1/VC% and FEV25-75 ranged from 19.64%-31.7%, 7.8-19.6% and 15.6% -26.05% respectively.

Family history of atopy was found to have significant negative correlation with baseline vital capacity, PEFR and FEV1.

BMI had positive correlation with PEFR only particularly in male sex. Other family and personal variables did not have significant difference on lung function parameter.

Body surface area corrected baseline lung function parameters was statistically highly significant in both sex in without EIBHR (p=0.008, p\0.001 and =0.0001 for PEFR, FEV and FEV1 respectively) but was not statistically significant in both sex with EIBHR.

Most of the EIHBR is coming from urban area(76%) as compared to rural area(24%) and it more prevalent in males (57.89%) as compared to females (42.10%).

Parental Smoking and family history of smoking and Asthma have thus, been concluded to be one of the important etiological factor in the symptomatology of children with EIBHR.

REFERENCES

1. Kawabori I, et al. Incidence of Exercise – induced asthma in children. J Allergy & Clinical Immunology 1976; 56:447-55.
2. Guidelines for the diagnosis and management of asthma: expert panel report 2. Bcthesda, MD: NIH, NHLBI. April 1997. P.55-6.
3. Deal EC Jr et al. Hyperpnea and heat flux: initial reaction sequence in exercise – induced asthma. J Applied Physiology 1979; 46:476-83.
4. Strauss RH et al. Influence of heat and humidity on the airway obstruction induced by exercise in asthma. J. Clinical Investigation 1979; 69:433-40.
5. Deal EC Jr. et al. Role of respiratory heat exchange in production of exercise induced asthma. J Applied Physiology 1979; 46:467-75.
6. Strauss RH et al. Role of respiratory heat exchange in production of exercise induced asthma. J Applied Physiology 1979; 46:467-75.
7. Vincent J. et al. Exercise Induced Asthma, The physician and sports medicine November 1999 vol 27(12).
8. Godfrey S, Bar, Yishay E. Exercise induced asthma revisited. Respiratory Med 1993; 87:331-44.
9. Ghai O. P. Respiratory system. Essential Paediatrics: 4th Ed; 283-89.
10. T Kubota – The relationship of mono nuclear leucocyte beta adrenergic receptors to aerobic capacity and exercise induced asthma in asthmatic children. Arerugi 2000 Jan: 49 (1); 40-51.
11. S. Kivity – Eosinophil influx into the airways in patients with excersice induce asthma. Respiratory medicine 2000 Dec: 94(12); 1200-1205.
12. Carey OJ, Cookson JB: The effect of lifestyle on wheeze, atopy and bronchial hyperactivity in Asian and white children. Am J Respiratory Critical care Medicine 1996 Aug: 154(1); 537-40.
13. Bardagi G, Agudo A: Prevalence of exercise induced airway narrowing in school children from a meditterranean town. Am Rev Respiratory Diseases 1993 May : 147 (5) ; 1112-5.
14. J.J.S. Monje . Effect of heating on FEV1 in children with asthma challenge with exercise. Rev allergy Mexico 2000 Mar : 47 (2) : 75-79
15. Peat JK: Sensitivity to common allergens: relation to respiratory symptoms and bronchial hyperresponsiveness in chidren from three different climatic areas of Australia. Clinical Exp Allergy 1991 Sep : 21(5); 578-81.
16. Flynn MG. Respiratory symptoms, bronchial responsiveness and atopy in Fijian and Indian children. Am J Respir crit care med 1994 Aug: 150(2); 415-20.
17. Cypcar D: The impact of respiratory infections on asthma . Paediatric Clin North Am 1992 Dec: 39(6); 1259-76.
18. Forastiere F. Bronchial Responsiveness in children living in areas with different air pollution levels. Arch Environ Health 1994, Mar – Apr: 49(2); 111-8.
19. S. Y. Seth. Relation of explosive to airway irritants in infancy to prevelance of bronchial hyper responsiveness in school children. Lancet 1995 Jan: 345 (28); 217 – 20.
20. Roizin H: Atopy, bronchial hyper responsiveness and peak of low variability in children with mild occasional wheezing. Thorax. 1996 Mar: 51 (3); 272-6.
21. Menon P. Passive cigarette smoke challenge studies: Increase in bronchial hyper reactivity. J Allergy Clin Immunology. 1992 Feb: 89 (2); 560-6.
22. Studnicko MJ. Seasonal and allergic predictors of bronchial responsiveness to distilled water. Am Rev Respir Dis 1993 Dec: 148 (6);1460-6.