Results

The study comprised fifty children with bronchial asthma (32 males and 18 females) aged from 3 to 12 years (with a mean of 6.36 ± 2.0), they were subdivided into:

-**Group I**: 25 asthmatic children were selected from the Allergy Clinic coming to our hospital for follow up (inbetween attacks).

-**Group II**: 25 asthmatic children were selected from emergency department coming to our hospital with acute exacerbation of asthma.

The study also included 40 healthy age and sex matched children with no history of current or previous atopic and/or upper or lower chronic airway disease as a control group.

Our results were tabulated and presented in tables (1-11) and figures (1-15) respectively.

Table 1: Sex distribution among the studied groups:

Sex	Patients (n=50)		Controls (n=40)		χ^2	P value
	no	%	no	%		
Male	32	60.0	16	40.0	3.56	>0.05
Female	18	40.0	24	60.0		

It is clear that the percentage of asthma is higher in males than females.

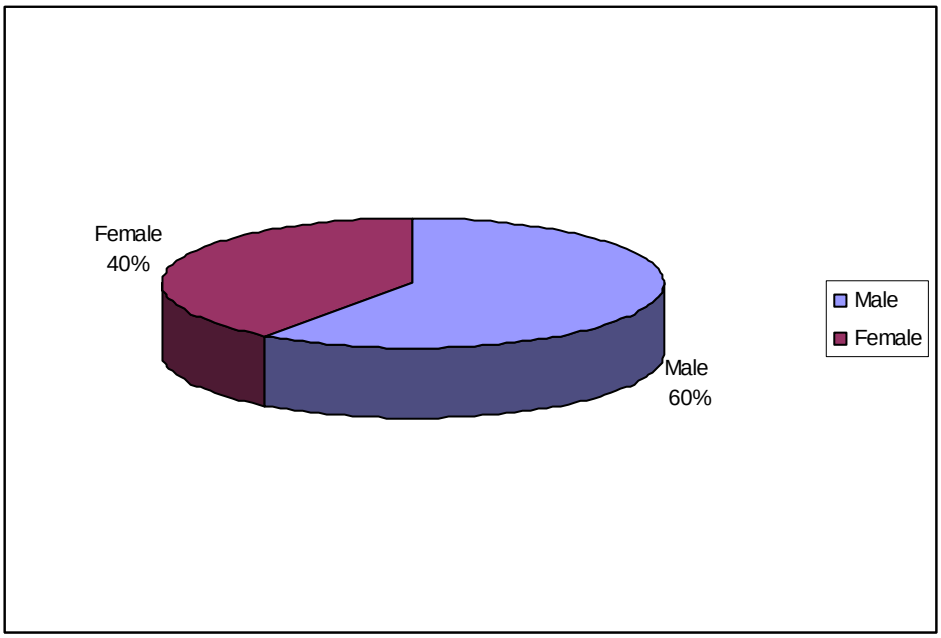


Fig 1: Sex distribution in asthmatic patients

Table 2: Socio-economic standard of the studied groups:

	Patients (n=50)		Controls (n=40)		χ^2	P value
	no	%	no	%		
Residence						
Urban	**35**	**70.0**	**19**	**47.5**	**4.69**	**<0.05**
Rural	**15**	**30.0**	**21**	**52.5**		
Socio-economic condition (SEC)						
High	**19**	**38.0**	**24**	**60.0**	**4.31**	**<0.05**
Low	**31**	**62.0**	**16**	**40.0**		

This table shows that regarding residence, urban residence was significantly higher among patients (70%) than among controls (47.5%) (P<0.05). Also it shows that low SEC was significantly higher among patients (62%) than

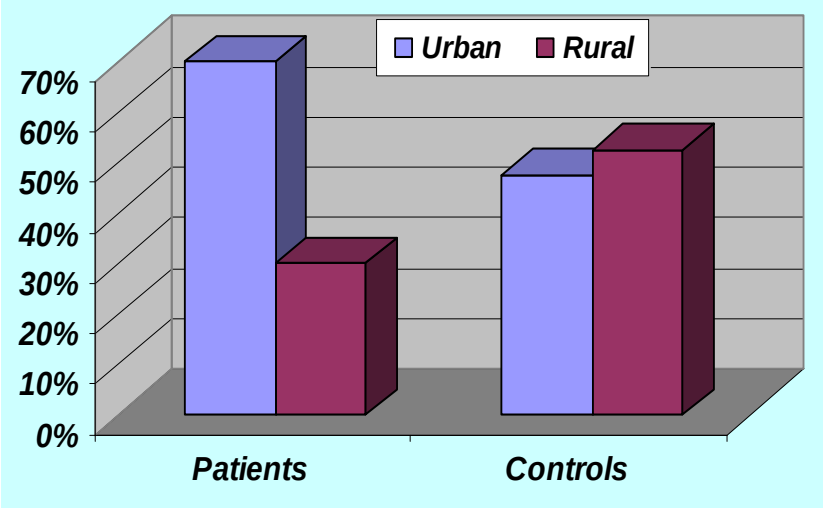


Fig 2: Residence distribution among patients and controls

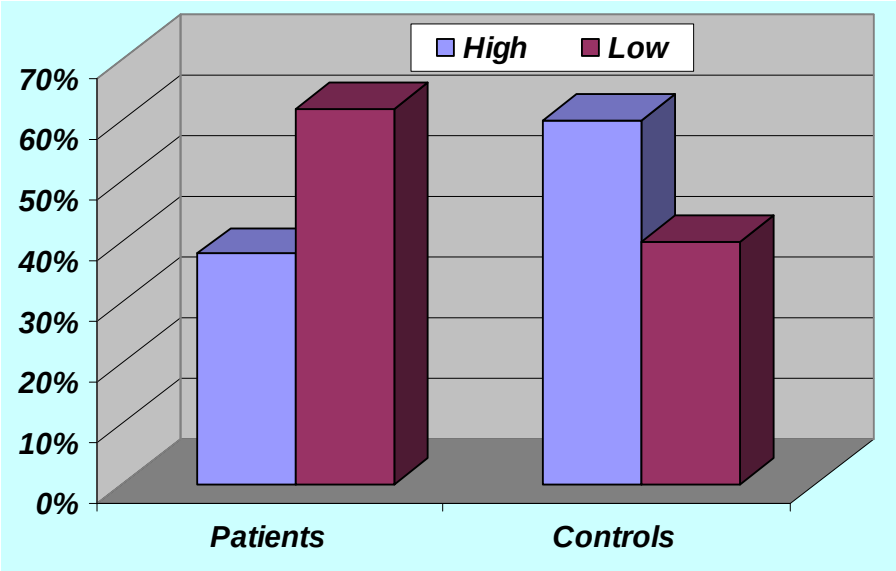


Fig 3: Socio-economic standard distribution among patients and controls

Table 3: Frequency of different precipitating factors in studied patients:

Precipitating factors	%
None	6%
Smoking	36%
Infection	6%
Pets and domestic animals	2%
Food allergy	4%
Dust	1%
Smoking & infection	25%
Cold air& Smoking	15%
Psychological	3%
Infection& food	2%

This table shows the frequency of the different precipitating factors of asthma in patients. The most frequent factor was passive smoking.

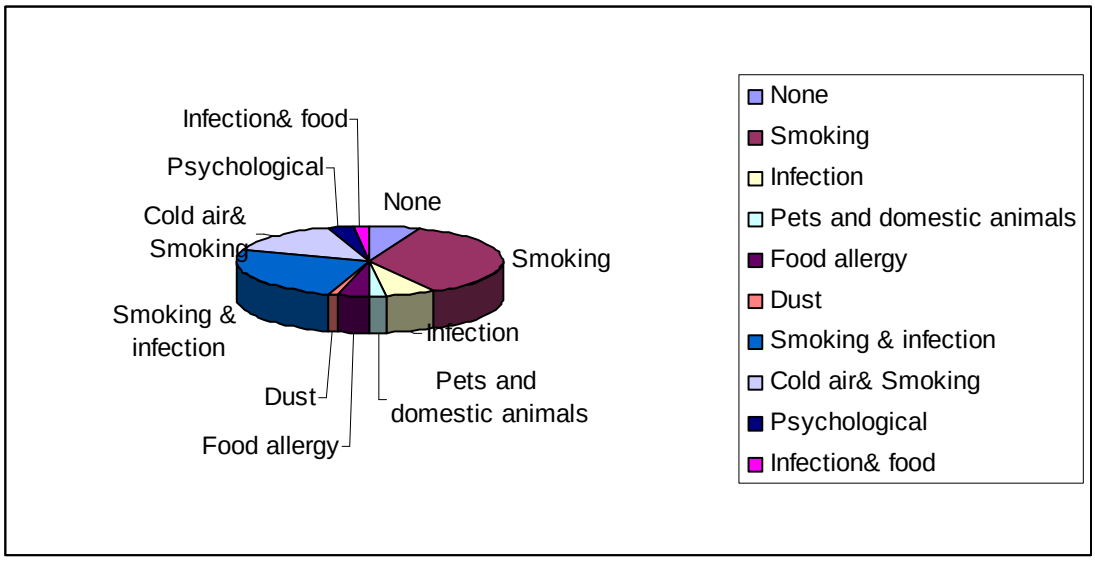


Fig 4: Frequency of different precipitating factors in studied patients

Table 4: passive smoking as the most important precipitating factor in studied patients:

	Patients (n=50)		Controls (n=40)		χ^2	P value
	no	%	no	%		
Passive smoking						
Yes	**38**	**76.0**	**16**	**40.0**	**12.0**	**<0.01**
No	**12**	**24.0**	**24**	**60.0**		

This table shows that history of passive smoking was significantly higher among patients (76%) than among controls (40%) (P<0.01).

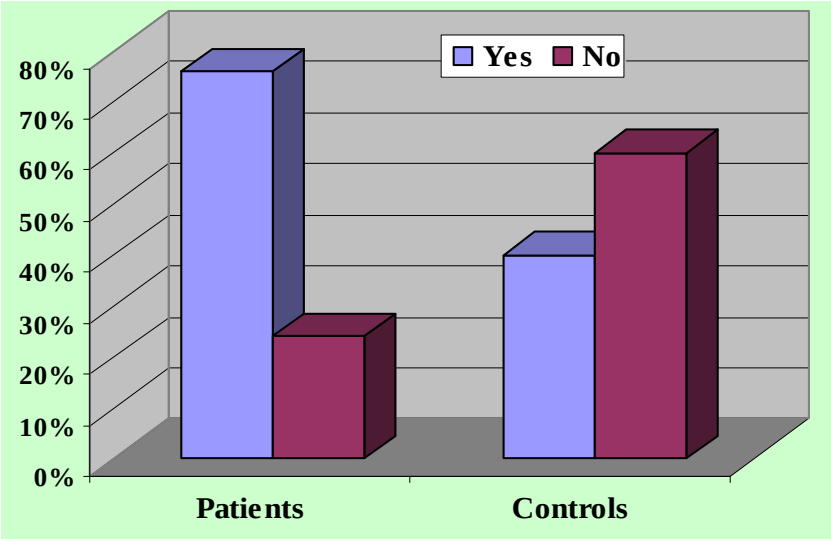


Fig 5: History of passive smoking distribution among patients and controls

Table 5: The family history among studied groups

	Patients (n=50)		Controls (n=40)		χ^2	P value
	no	%	no	%		
Family history of asthma						
Positive	**36**	**72.0**	**0**	**0.0**	**48.0**	**<0.01**
Negative	**14**	**28.0**	**40**	**100.0**		
Consanguinity						
Positive	**18**	**36.0**	**15**	**37.5**	**0.02**	**>0.05**
Negative	**32**	**64.0**	**25**	**62.5**		

This table shows that regarding positive family history of asthma, it was significantly higher among patients (72%) than among controls (0.0%) (P<0.01) but no statistically significant difference as regard Consanguinity.

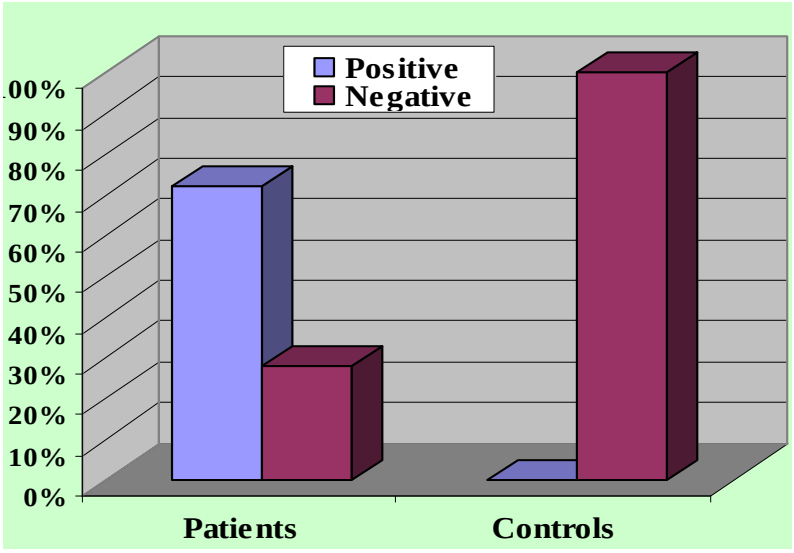


Fig 6: Family history distribution of asthma among patients and controls

Table 6: Grading of asthma in studied patients

Grading	count	%
Intermittent	7	14
Mild persistent	12	24
Moderate persistent	18	36
Sever persistent	13	26

This table demonstrates the distribution of patients according to severity of disease guided by GINA classification. Intermittent asthma was diagnosed in 7 patients (14%) while mild, moderate and sever persistent asthma was diagnosed in 12 patients (24%), 18 patients (36%) and 13 patients (26%) respectively.

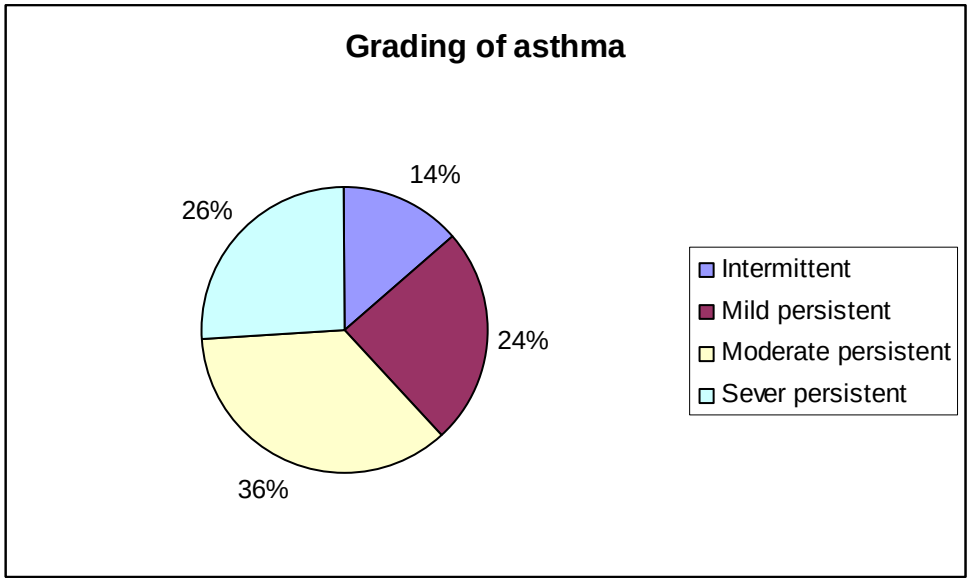


Fig 7: Grading of asthma in studied patients

Table 7: Comparison between patients of group I (inbetween attacks) and group II (during attack) regarding characteristics of asthma:

	Patients				χ^2	P value
	group I (n=25)		group II (n=25)			
Other allergic diseases						
Yes	**19**	**76.0**	**17**	**68.0**	**0.39**	**>0.05**
No	**6**	**24.0**	**8**	**32.0**		
Diurnal variation						
Night	**25**	**100.0**	**17**	**68.0**	**9.52**	**<0.05**
Morning	**0**	**0.0**	**8**	**32.0**		
Seasonal variation						
Winter	**19**	**76.0**	**25**	**100.0**	**6.82**	**<0.05**
Spring	**6**	**24.0**	**0**	**0.0**		

This table shows that regarding night diurnal variation, it was significantly higher among group I patients (100%) than among group II patients (68%) ($P<0.05$). But it shows that winter seasonal variation was significantly lower among patients of group I (76%) than among patients of group II (100%) ($P<0.05$).

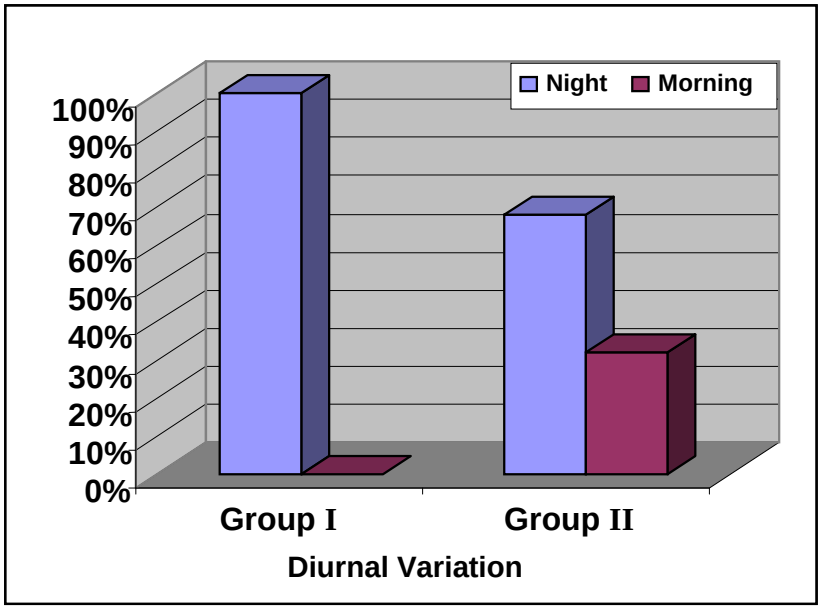


Fig 8: Diurnal variation distribution among group I (inbetween attacks) and group II (during attack) patients

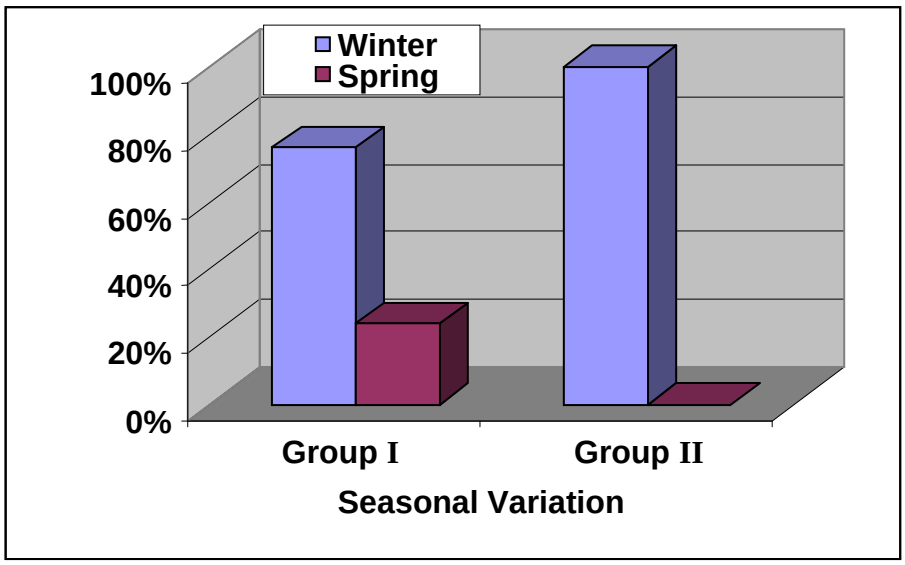


Fig 9: Seasonal variation distribution among group I (inbetween attacks) and group II (during attack)

Table 8: Forms of allergic diseases among the studied children

Atopic diseases	Patients (n=50)		Controls (n=40)		P value
	no	%	no	%	
None	14	28%	32	80%	<0.05
atopic dermatitis	16	32%	2	5%	
allergic rhinitis	11	22%	5	12.5%	
Conjunctivitis	6	12%	0	0%	
food allergy	3	6%	1	2.5%	
Total	50	100%	40	100%	

The table shows that there is statistically significant difference between patients and controls as regard the history of atopic diseases.

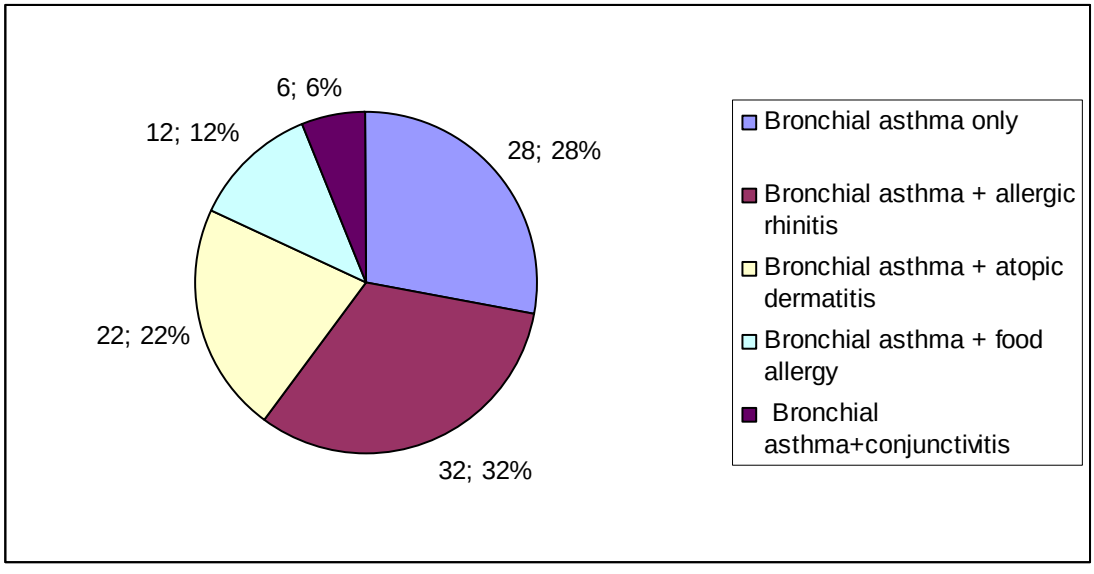


Fig 10: Forms of other allergic diseases among the asthmatic patients

Table 9: Mean absolute eosinophil count, IgE and PAI-1 in cases and controls:

Variable	Patients (n=50)	Controls (n=40)	P value
	X±SD	X±SD	
PAI	9264.0 ± 3410.5	2860.0 ±4628.4	<0.01
eosinophils	349.6±433	185±44.2	<0.01
IgE	205.5 ±272.8	31.1 ±0.1	<0.001

This table shows that mean absolute eosinophil count, IgE and PAI-1 were significantly higher among patients than among controls.

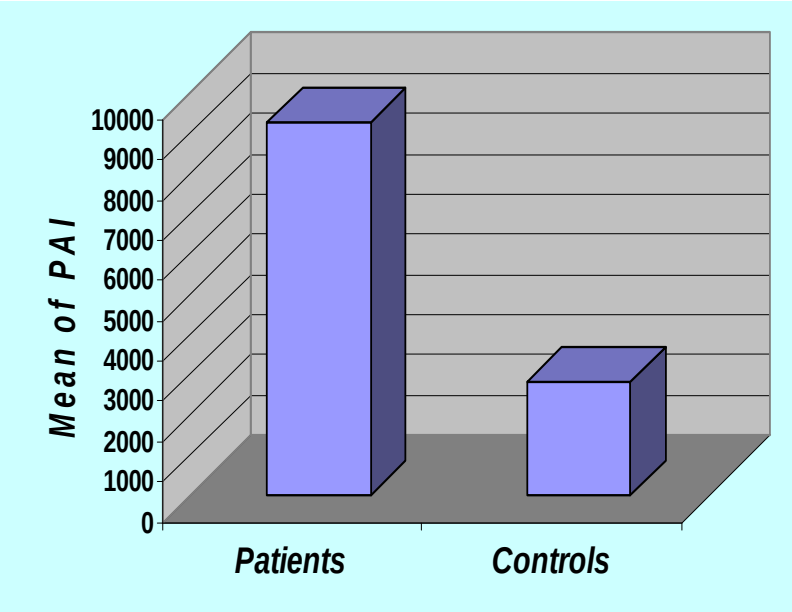
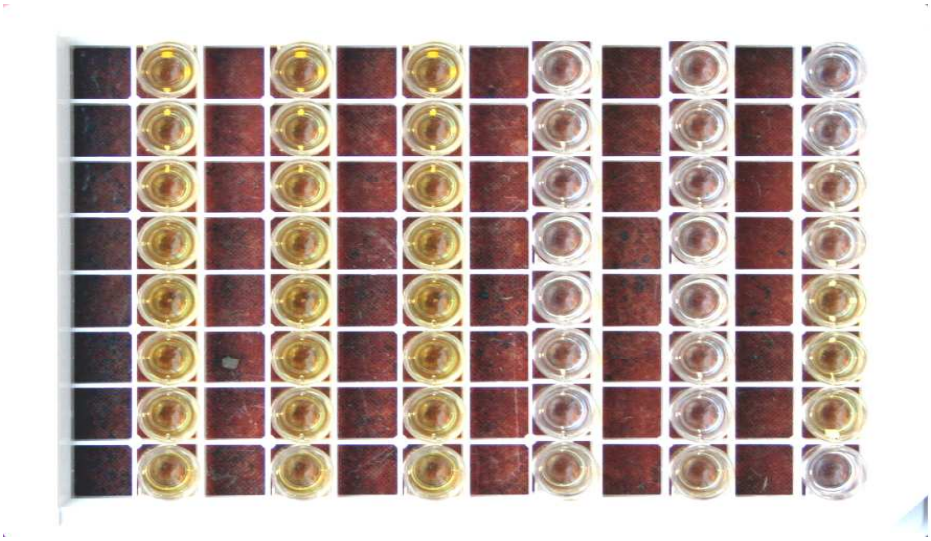


Fig11: Mean value distribution of PAI among patients and controls



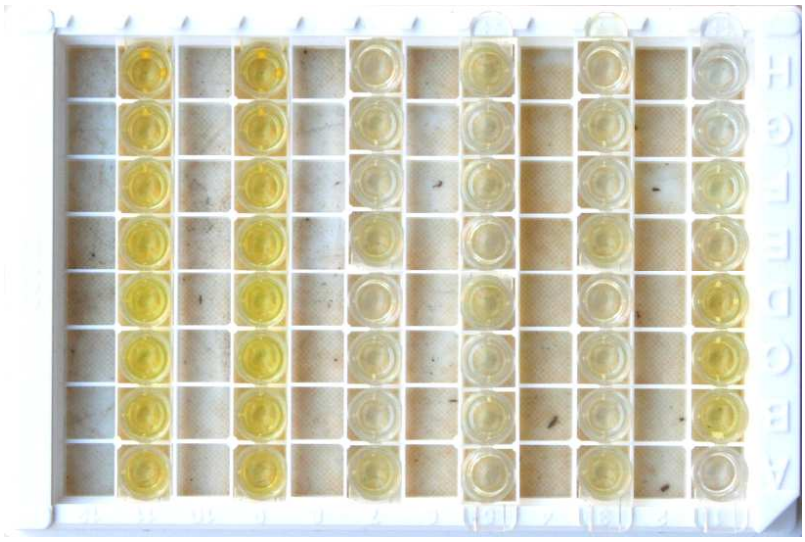


Fig12: Boster human PAI-1 ELISA Kit

Table 10: Comparison between patients in group I (inbetween attacks) and group II (during attack) regarding PAI, onset and duration of asthma:

	Patients		Kruskal-Wallis test	P value
	Group I (n=25)	Group II (n=25)		
	X±SD	X±SD		
Plasminogen activating inhibitor	6728.0 ± 2085.6	11800.0± 2449.5	7.88	<0.01
Onset of disease	0.98 ± 0.6	1.16 ± 0.5	1.75	>0.05
Duration	3.1 ± 2.3	4.8 ± 3.6	1.78	>0.05

*** t test**

This table shows that PAI was significantly lower in Group I patients (**6728.0 ± 2085.6)** than in Group II patients (**11800.0± 2449.5**) (P<0.01).

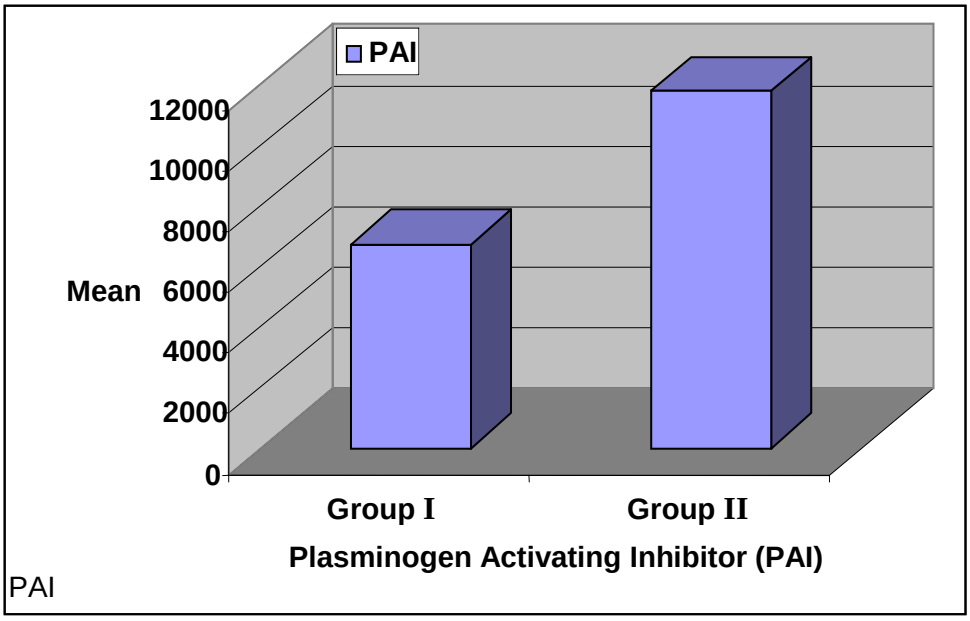


Fig 13: Mean value distribution of PAI among group I (inbetween attacks) and group II (during attack)

Table 11: Correlation between PAI and duration & grading of asthma and eosinophilia:

	Plasminogen activating inhibitor (PAI)	
	r	**P value**
Duration of asthma	**+ 0.34**	**< 0.05**
Grading of asthma	**+0.94**	**<0.001**
Esinophilia	**+0.367**	**<0.01**

This table shows that there was significant positive correlation between PAI and duration of asthma ($P<0.05$) and highly significant positive correlation between PAI and grading of asthma ($P<0.001$). Also it shows that there was highly significant positive correlation between PAI and eosinophilia ($P<0.01$)

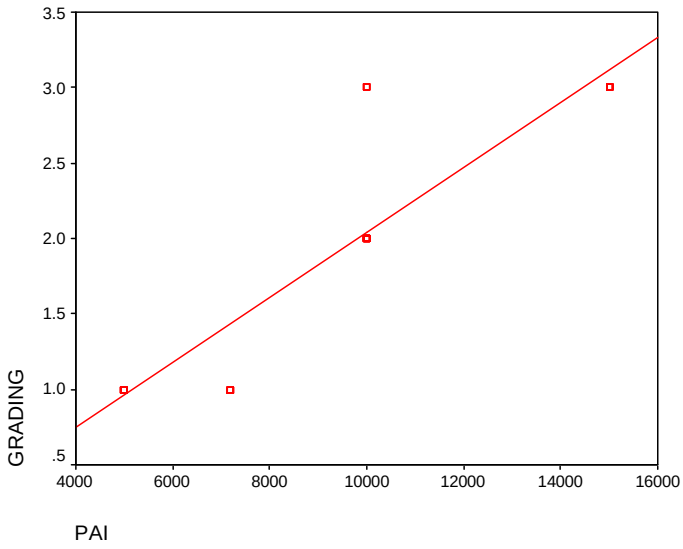


Fig 14: Correlation between PAI and grading of asthma

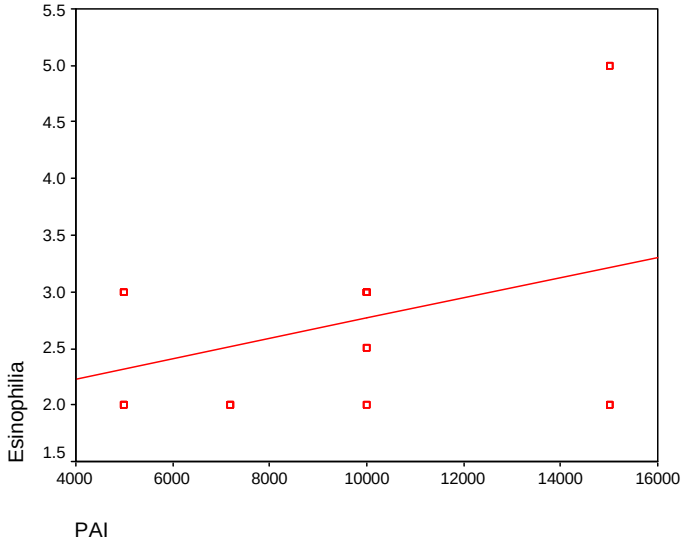


Fig 15: Correlation between PAI and Eosinophilia