RESULTS

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Twenty female SLE patients were included in our study. The mean of their age was 21.65 ± 6.63, the mean age of onset was 19.79 ± 6.19, and the mean disease duration was 1.86 ± 1.17.

According to disease activity index score, our patients were divided into 3 groups:

Group I: Mild included 6 (30%) patients.

Group II: Moderate included 10 (50%) patients.

Group III: Severe included 4 (20%) patients.

The activity score was ranging from 5-35 and it's mean was 14.4 ± 6.98.

As regards disease manifestations: all patients showed general manifestation [17 (85%)] showed fever, 12 (60%) showed fatigue and 5 (20%) showed weight loss].

Regarding muscloskeletal manifestation arthritis was seen in 16 (80%) patients and arthralgia in 3 (15%) patients.

Mucocutaneous manifestations were found in all patients [18 (90%) with malar rash, 13 (65%) with mucosal ulcer, 16 (80%) with alopecia and 1 (5%) with erythema nodusum].

Central nervous system involvement was present in 2 (10%) patients as lupus headache.

Renal manifestations were present in 10 (50%) patients (10 patients with proteinurea and 1 patient with urinary cast).

Respiratory manifestations were presented in the form of pleurisy in 4 (20%) patients and pleural effusion in 2 (10%) patients.

Vasculitis was seen in 2 (10%) patients (spinter haemorrhage and ulcer tip of finger).

Cardiac involvement was present as palpitation 1 (5%), pericardial effusion 1 (5%), pericarditis 1 (5%) and tachycardia with gallop in 1 (5%) patients.

Only 1 (5%) patients was having abdominal manifestation in the form of ascities.

As regards drugs used in the treatment of our patients: 16 (80%) patients were using corticosteroid tablets, 12 (60%) patients were using chloroquine, 5 (25%) patients were using Azathioprine, 4 (20%) patients were using cyclophosphamide pulse and only 1 (5%) patients was using corticosteroid pulse.

The prevalence of hyperprolactinemia in our patients was 20% as 4 SLE patients among a total of 20 patients were hyperprolactinemic.

2 (50%) of these hyperprolactinemic patient were reciving corticosteroid tablets as a treatment, 3 (75%) were reciving cyclophosphamide pulse, 1 (25%) was receiving Azathioprine tablet. None of them was treated with chloroquine tablet.

Table (2): Clinical data prevalent among the studied cases.

Clinical picture	N.	%
General:		
* Fever	17	85
* Fatigue	12	60
* Weight loss	5	20
Muscloskeletal:		
* Arthritis	16	80
* Arthralgia	3	15
Mucocutanous:		
* Malar rash	18	90
* Mucosal ulcer	13	65
* Alopecia	16	80
* Erythema nodusum	1	5
Renal		
* Proteinuria.	10	50
* Urinary cast	1	5
C.N.S.		
* Headache	2	10
Respiratory:		
* Pleurisy	4	20
* Pleural effusion	2	10
Vasculitis:		
* Splinter hemorrhage	2	10
* Ulcer at tip of finger	2	10
Cardiac manifestation:		
* Palpitation	1	5
* Pericardial effusion	1	5
* Pericarditis	1	5
* Tachycardia with gullop	1	5
Abdomen:		
* Ascitis	1	5

N. = Number

CNS = Central nervous system.

Table (3): Laboratory results of the studied cases.

Parameter	Range	Mean ± S.D.
ESR. mm/h	45-150	72.5 ± 26.78
Hb g/dl	7-9.8	8.31 ± 0.75
RBCs /mm^3	2.70 -3.70	3.21 ± 0.27
WBCs /mm^3	2700-4300	3520 ± 597.01
Platelets /mm^3	120-220	184.5 ± 27.04
PRL (ng/ml)	3.6-27.6	14.89 ± 5.78

ESR = Erythrocyte sedmintation rate.

Hb = haemoglobin.

RBCs = Red blood corpusles

WBCs = White blood cells.

PRL = Prolactin

Table (4): Prevalence of hyperprolactinemia among the studied patients.

Prolactine level (ng/ml)	N.	%
Hyperprolactinemia	4	20
Normoprolactinemia	16	80
Total	20	100

N. = Number.

Table (5): Prolactin level according to the age of the studied patients.

Prolactin level (ng/ml)	Juvenile patients		Adult patients	
	N.	%	N.	%
Hyperprolactinemia	1	12.5	3	25
Normorpolactinemia	7	87.5	9	75
Total	8	100	12	100

N = Number

Table (6): Comparison of mean prolactin level between patients and control.

Prolactin level (ng/ml)	Cases (N=20)	Control (N=10)	t-test	P	Sig.
All patients	14.4 ± 5.78	10.6 ± 3.23	2.17	0.03	Sig.
Adult patients	13.26± 6.65	8.78 ± 3.14	2.04	0.04	Sig.
Juvenile patients	15.98 ± 5.14	11.43± 2.65	2.40	0.03	Sig.

P < 0.05 = Significant
N = Number
t = Student t-test value.

Table (7): Demographic and clinical variables of patients with hyper prolactinemia and normal prolactin level.

Character	Hyperprolactinemia Mean ± S.D. (N = 4)	Normal prolactin level Mean ± S.D. (N = 16)	t P Sig.
Age (years):			1.68
Range	14-34	13-35	0.11
Mean ± S.D.	26.0 ± 8.76	20.56 ± 5.83	N.S.
Age of onset (years):			1.37
Range	12-31	12-31	0.19
Mean ± S.D.	23.5 ± 8.58	18.87 ± 5.4	N.S.
Disease duration (years):			1.24
Range	2.0 –3.0	0.08 – 4.0	0.23
Mean ± S.D.	2.5 ± 0.58	1.7 ± 1.24	N.S.
Total activity score:			6.30
Range	14-33	5-15	0.23
Mean ± S.D.	25.75 ± 6.4	11.56 ± 3.24	Sig.

N = Number
P < 0.05 = Significant
NS = non significant
SD = Standard deviation
t = Student t-test value

Table (8): Comparison of clinical presentation between hyperprolactinemic patients and normoprolactinemic patients level.

Clinical picture	Hyperprolactinemic		Normoprolactinemic		Z-test	P	Sig.
	N.	%	N.	%			
General:							
* Fever	4	100	13	81.3	0.939	0.174	N.S.
* Fatigue	1	25	11	68.8	1.598	0.055	N.S.
*Weight loss	1	25	4	25	0.00	0.50	N.S.
Muscloskeletal:							
* Arthritis	4	100	14	87.5	0.745	0.228	N.S.
* Arthralgia	0	0	3	8.8	0.939	0.174	N.S.
Mucocutanous:							
* Malar rash	3	75	15	93.8	1.118	0.132	N.S.
* Mucosal ulcer	4	100	9	6.3	1.64	0.05	Sig.
* Alopecia	4	100	14	87.5	0.745	0.228	N.S.
*Erythema nodusum	0	0	1	6.3	0.51	0.30	N.S.
Renal:							
* Proteinuria	4	100	6	37.5	2.236	0.013	Sig.
* Urinary cast	1	25	0	0	2.052	0.020	Sig.
C.N.S.							
* Headache	2	50	0	0	2.98	0.001	Sig.
Respiratory:							
* Pleurisy	2	50	1	6.3	2.192	0.014	Sig.
* Pleural effusion	1	25	1	6.3	1.118	0.132	NS
* Interstitial fibrosis.	1	25	0	0	2.052	0.020	Sig.
Vasculitis:							
* Splinter hemorrhage	2	50	0	0	2.98	0.001	Sig.
* Ulcer at tip of finger	0	0	2	12.5	0.745	0.228	NS
Cardiac manifestations:							
* Palpitation	0	0	1	6.3	0.51	0.30	NS
* Pericardial effusion	0	0	1	6.3	0.51	0.30	NS
* Pericarditis	1	25	0	0	2.052	0.020	Sig.
* Tachycardia with gullop	0	0	1	6.3	0.51	0.30	NS
Abdomen:							
* Ascitis	0	0	1	6.3	0.51	0.30	NS

P < 0.05 = Significant NS = Non significant
Sig. = Significance N. = Number
t = Student t-test value Z = test of percentage

Table (9): Comparison of mean laboratory findings between hyperprolactinemic and normoprolactinemic.

Finding	Hyperprolactinemic (N =4)	Normoprolactinemic (N =16)	t-test	P	Sig.
ESR mm/h	88.75 ± 19.31	68.44 ± 27.31	1.39	0.18	NS
WBCs/mm^3	2900 ± 182.27	3675 ± 562.73	4.62	0.000	Sig.
RBCs /mm^3	3.03 ± 0.05	3.26 ± 0.28	3.09	0.000	Sig.
Hb g/dl	7.38 ± 0.48	8.54 ± 0.61	4.08	0.007	Sig.
Platelet/mm^3	142.5 ± 15.0	195.0 ± 17.13	6.08	0.002	Sig.

ESR = Erythrocyte sedmintation rate

WBCs = White blood cells.

RBCs = Red blood corpusels.

Hb = Haemoglobin

$P < 0.05$ = Significant

NS = Non significant

t = Student t-test value

Table (10): Comparison of the disease grading between hyperprolactinemic and normoprolactinemic.

Grading	Hyperprolactinemic		Normoprolactinemic		X^2	P	Sig.
	N.	%	N.	%			
Mild	0	0	6	37.5	11.73	0.006	Sig.
Moderate	0	0	10	62.5			
Severe	4	100	0	0			
Total	4	100	16	100			

$P < 0.05$ = Significant

X^2 = Qui-square test

Table (11): Correlation between serum prolactin level and clinical variables in SLE patients.

Variable	r*	P	Sig.
Age of onset (years)	+ 0.348	0.13	NS
Disease duration (years)	+ 0.106	0.66	NS
Total activity score	+ 0.628	0.003	S

P < 0.05 = Significant
NS = Non significant
r = Correlation coefficient

Table (12): Correlation between serum prolactin level and laboratory variables in SLE patients.

Variable	r*	P	Sig.
ESR mm/h	+ 0.192	0.417	NS
WBCs /mm^3	- 0.387	0.09	NS
RBCs /mm^3	- 0.063	0.79	NS
Hb g/dl	- 0.205	0.38	NS
Platelet /mm^3	- 0.551	0.012	Sig.
Anti nuclear antibody	- 0.139	0.56	NS

P < 0.05 = Significant
NS = Non significant
r = Correlation coefficient

Fig. (1): Disease grading of the studied cases.

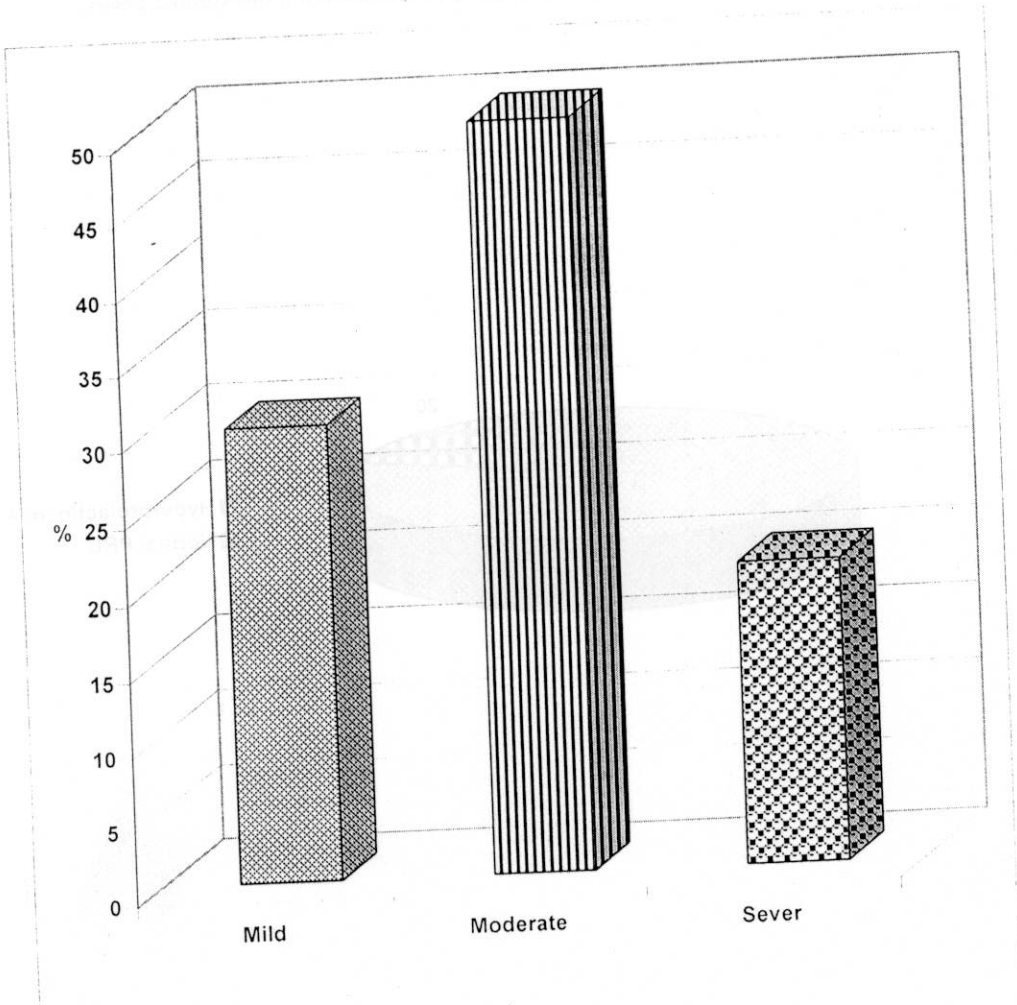


Fig. (2): Prelvanece of hyperprolactinemia among the studied cases.

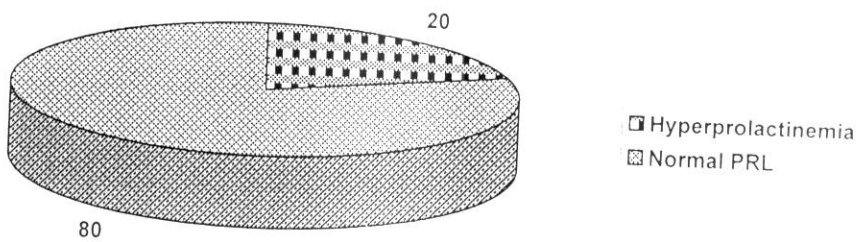


Fig. (3): Prolactine level according the age of the studied cases.

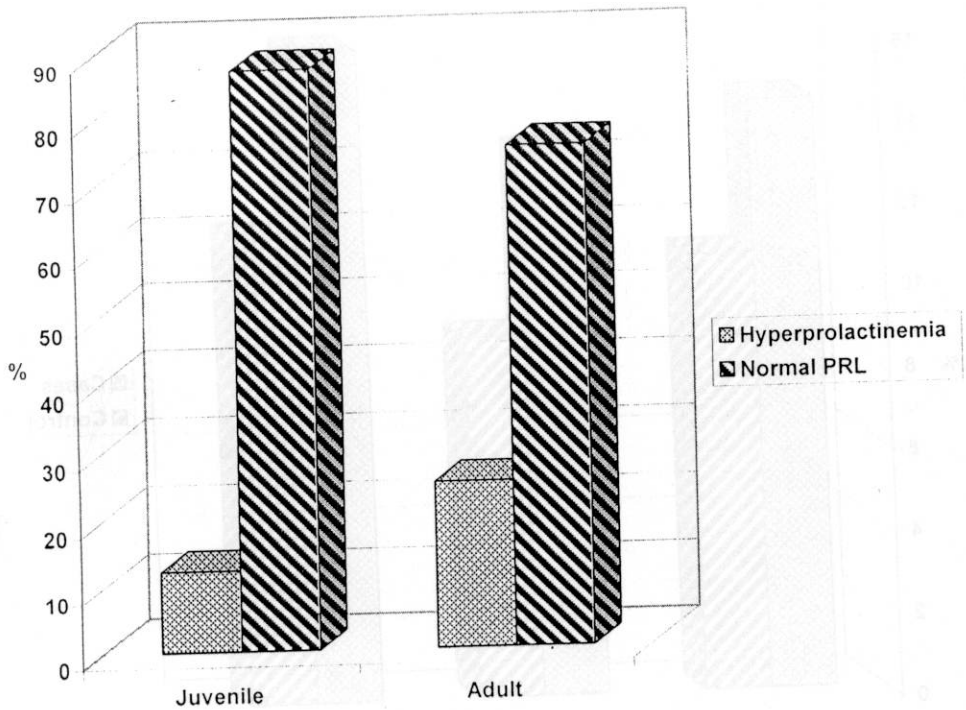
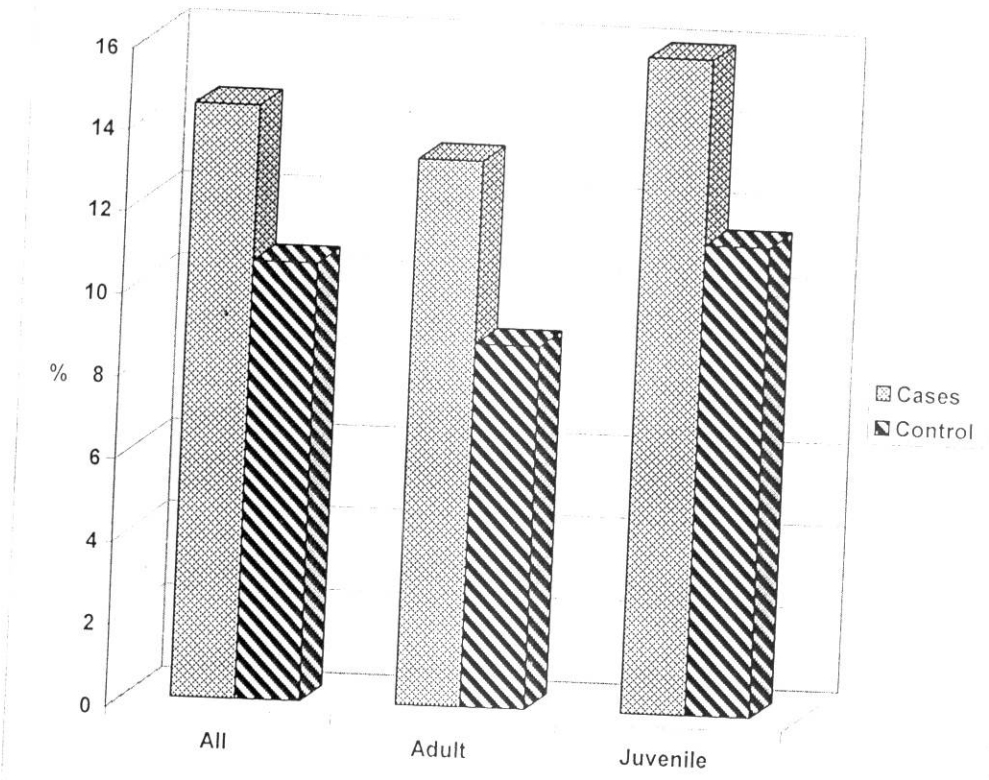


Fig. (4): Comparison of mean PRL level between patients and control.



DISCUSSION

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It is evident know that the immune response and consequently the autoimmune disease is controlled by a system of bidirectional communication between the immune and neuroendocrine systems, in which the two systems share a common set of hormones and receptors ***(Besedovsky et al., 1985).*** The immune cells posses receptors for neuroendocrine peptides (e.g.: neurotensin, substance P, PRL, GH, ACTH, TSH and VIP), also these cells can synthesize these neuroendocrine peptide. On the other hand products of immune cells (e.g., IL1, IL2, IL6, interferon alph and interferon gamma) affect the central nervous system which possess receptors for cytokines and can also synthesize them ***(Bhalla, 1989).*** In other wards, signals perceived by cells of the immune system throught their receptors are translated into language common to the immune and neuroendocrine system. In this way a unique signalling apparatus transmits exogenous antigenic stimuli to an endogenous control system involving delivery of hormone like mesengers to and from the central nervous system ***(Matera et al., 1991).***

A widely accepted and most studied model for the bidirectional communication between the immune and neuroendocrine system is the activation of hypothalamo-hyophyseal-adrenal axis by the monocyte derived IL-1 with production of ACTH and glucocorticoids which prevent inappropriate prolongation of the immune response and inflammation ***(Lumpkin, 1987).*** Also PRL is considered as an important regulator of the immune system, affecting both humoral and cell mediated system. PRL may play a role in the pathogenesis and disease expression of autoimmune disease especially SLE ***(Lavalle, 1992).***