Predictive Value of Plasma Prolactin Level in the Differential Diagnosis between Limbic Seizures and Psychotic Behavioral Disorders

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Abstract

This study evaluates the usefulness of serum prolactin (PRL) levels in predicting the disturbed behavior related to either epileptic or psychotic disorders. Data were collected from 112 patients consecutively admitted to Neurology and Psychiatric Departments, in a 12-month period. Those patients included 53 epileptic patients (31 had grand mal epilepsy "GM" and 22 had complex partial seizures "CPS") and 59psychotic patients (19 had Electro convulsive therapy "ECT" and 40 had no ECT). Plasma PRL concentration (P1) was assessed 20 minutes after the seizures or the abnormal behavior episodes and a second PRL level (P2) was 24 hours after (P1) as a baseline level. Using the criteria of two-fold elevation of PRL levels above the baseline value correctly differentiated behavioral disorders due to either epilepsy or psychotic disorders. The criteria was positive in 21(67.7%) of 31 patients had GME, in 11 (50%) of 22 patients had CPS of temporal lobe origin (Limbic epilepsy), in 19(100%) of 19 psychotic patients received ECT, and in only 4 (10%) of 40 psychotic patients received no ECT.

It could be concluded that two-fold increase of PRL level above the baseline has a highly predictive value in differentiating behavioral disorder due to epilepsy versus psychotic disorders. Also, this study suggests to perform further studies using one fold epileptic and psychotic behavioral disorders.

Introduction

The first step towards optimal treatment of epilepsy is accurate diagnosis. Alterations in consciousness, or behavior may be consequence of epileptic or non-epileptic episodes. (Wada, 1985).

Patients with limbic epilepsy have been particularly studied because it is the most common form of epilepsy and frequently difficult to treat. Limbic epilepsy, in particular, has been associated with psychopathology, indeed, some investigators have argued that a psychosis or thought disorder occurs with limbic epilepsy. (Bear et al, 1982; Greschwind, 1983). Many psychotic disorders may occur during complex partial seizures (CPS) or simple partial seizures (SPS) e.g. illusions and hallucinations. Emotional disturbance also, may occur in the form of sadness, loneliness, anger, happiness, fear, anxiety or even violence and aggression. A direct attack of uncontrollable rage may occurs either as part of a seizure or as an interictal phenomenon. A lesser degree of aggressive behavior as a part of a temporal lobe seizure is not uncommon. At certain periods of life, usually adolescence or early adulthood, it begins to have episodes of wild, aggressive behavior. One suspects epilepsy, but there

is no history of recognizable seizure and no interruption of consciousness, which is so typical of CPS. The Electro encephalography electrodes placed in the amygdaloid nuclear complex have recorded seizure discharges (Adams et al, 1997).

Paroxysmal psychotic behavioral disorders are clinical events that resemble limbic epileptic attacks but are not associated with physiological central nervous system dysfunction. Patients with such attacks must be evaluated very carefully because of possible misdiagnosis. The psychiatric classifications of non-convulsive, non convulsive, non epileptic seizures include some psychotic disorders such as Schizophrenia, Schizo-affective disorder, Bipolar disorder (Kenneth et al, 1995).

The EEG is useful in diagnosis, however, findings on interictal EEG studies are often negative. A blood tests able to aid in the diagnosis of epilepsy would be very valuable. Measurement of serum prolactin (PRL) levels has been partially successful in addressing this need. (Yerby et al, 1987).

Post ictal elevations of serum PRL after true seizures but not after pseudo seizures were first described by Trimble in 1978, and subsequently substantiated by numerous authors (Abbot et al, 1980; Pritchard et al, 1983). PRL appears to be released at the onset of a grand mal epilepsy (GME) or CPS and less frequently in some (SPS involving limbic cortex. It reaches a peak elevation within 15 or 20 minutes, then followed by a decline to baseline values by 60 minutes post ictus (Sperling et al, 1986).

Serum PRL levels typically increase many fold after generalized seizures induced by Electro convulsive therapy (ECT) in man. Such elevation lasts for about an hour. (Balldin, 1982).

The criteria for a significant elevation of PRL vary with individual investigators. Some use an upper limit of 23 mg/ml beyond which PRL is considered positive for epilepsy. Others have used statistical methods of significance testing, i.e. student, test, to compare mean values. Most researchers will consider an elevation of at least two times baseline an indicative of true epilepsy. Baseline values are usually obtained 60 minutes after episode. (Yerby et al, 1987).

Many factors affect PRL release. In temporal lobe seizures, amygdalar activation alone is not sufficient and therefore hippocampal activation is also required. Widespread, intense activation of medial temporal limbic structures is followed by an increase in PRL levels with CPS (Sperling et al, 1986). Sleep, stress, antiepileptic drugs, and surgery may affect PRL secretion (Noel et al, 1972). PRL levels do not rise after convulsive or non convulsive status epilepticus (Malkowicz et al, 1995) or after absence seizures (Bilo et al, 1988).

Therefore, the aim of this study is to determine the clinical usefulness of plasma PRL as a diagnostic aid in the differential diagnosis between limbic epilepsy (CPS) and psychotic behavior disorders.

Materials and Methods

Subjects were in-patients at Almana General Hospital, Dammam, within the period from January 1st, to December 31st, 1997 via Emergency room (ER) and Out patient department. The patients with ages ranged from 15-45 years, median 32 years were considered for the study. During this age, PRL levels become stable after puberty

for both males and females and before menopause for females. One group of patients (53) consecutively admitted to the department of Neurology with a diagnosis of epileptic seizures, and the other group of patients (59) admitted to the department od psychiatry with a diagnosis of behavior changes due to psychotic disorders.

The patients with epileptic seizure were (29) males and (24) females. A first sample plasma PRL level (P1) was drawn within 20 minutes after cessation of the seizure if the attacks occurred while the patient was in the ER or in the ward. A second sample (P2) was drawn after 24 hours post ictus as a baseline PRL level. An interictal EEG was done to detect patients with either GME or CPS of temporal lobe origin. Scalp electrodes were placed according to the international 10-20 system. Seizures types were defined according to the Commission on Classification of Terminology of the International League Against Epilepsy 1981 guidelines. A contrast enhanced computed tomography (CT) scan of the brain was performed in All patients with proven epileptic. The epileptic patients were divided into convulsive group (GME) and non convulsive group (CPS).

The patients with psychotic behavioral disorders were 34 males and 25 females. They were diagnosed according to ICD_{10}. The patients mainly presented with: bouts of impaired awareness, aggressive disturbed behavior, bizarre delusion, and hallucinatory experience. They were divided into ECT and non ECT subgroups. The ECT group were (19) patients comprised (7) paranoid schizophrenia, (2) severe depression, (2) bipolar affective disorder – currently manic, (3) bipolar affective disorder – currently mixed, (2) undifferentiated schizophrenia, (3) affective disorder-manic. The psychotic group were (40) patients included: (11) paranoid schizophrenia, (5) undifferentiated schizophrenia, (4) schizo-affective disorder,(3) affective disorder manic episodes, (2) bipolar affective disorder currently depressed, (6) bipolar affective disorder currently manic, (1) acute psychotic disorder, (1) bipolar affective disorder currently mixed, (7) sever depression. For ECT subgroup, the patients did not receive any major tranquilizers for 4 weeks prior to the time of admission and were kept only on minor tranquilizers to control their disturbed behavior and sleep difficulties for 24-36 hours until first ECT session. Minor tranquilizers don't affect basal prolactin release (Wilson, 1979). For all patients, ECT consent was taken and the patients were starved overnight. The first blood sample (P1) was drawn after ECT within 20 minutes post-ictally, to evaluate the effect of generalized seizure induced by ECT on PRL level. The second blood sample (P2) for PRL was drawn after 24 hours following ECT stimulation as a base line level. The patient received Thyropentoin (100mg. to 300 mg.) and Succinycholin (30mg. to 50 mg). ECT was administered using an Thymatron TM DGx Computer-Assisted Seizure Monitor Output and End –of – Treatment Printed Report (EEG and EMG) machine set at waveform 1:This delivered waveform: bipolar brief pulse square wave; Current: 0.9 amps constant for up to 500 ohms impedance; Voltage: proportional to dynamic impedance, max. 450v; Frequency standard: 70Hz at 55-100% energy. Induction of a seizure was confirmed by inflating a sphygmomanometer cuff to 200 mm Hg just before administration of the muscle relaxant: the convulsion was thus observed unmodified in the forearm distal to the cuff

and recorded by EMG and also brain wave recorded by EEG. For the Psychotic non ECT subgroup, a first blood sample (P1) for PRL was drawn on admission and a second sample (P2) in the next 24 hours, while the patient on the minor tranquilizer and following alleviation of acute symptoms as baseline PRL level. EEG was done for each patient to detect abnormal discharges.

The blood sample was placed in a tube and immediately sent to the laboratory. Plasma PRL concentration was determined by usual radio-immune assay methods and expressed in nannograms per milliliter. The criteria for exclusion of patients were all patients who had abnormal endocrine, liver and kidney functions. Female patients who were menstruating, lactating or pregnant at the time of taking blood sample. Subjects received medication known to influence PRL secretion as neuroleptics for less than 4 weeks except the usual antiepileptic drugs for the epileptic groups. Also, we excluded patients who had brain tumor including pituitary adenoma or other brain lesion.

We considered an elevation of PRL level at least two times baseline s indicative of true epilepsy. Each subject served as his or her own control. We used a statistical method of significance testing (i.e. students "t" test) to compare mean values between the different convulsive subgroups, (ECT vs. GME) and between the non convulsive subgroups (Non ECT vs. CPS).

Results

Data were obtained from 112 patients, 53 had epileptic seizures (31 had GME and 22 had CPS), and 59 had psychosis (19 received ECT while 40 did not). All patients with GME had spontaneous tonic-clonic seizures, where as patients with CPS had spontaneous abnormal behavioral attacks. The ECT subgroup had induced seizures after ECT stimulation, whereas the non-ECT psychotic patients had bouts of behavioral changes. Serum PRL levels were measured 20 minutes after identified seizures (GME and ECT) or episodic behavioral changes (CPS and Non ECT). The EEG focus for the epileptic group was temporal in CPS patients and centrencephalic in GME.

The 20 minute post ictal PRL levels (P1) obtained for GME subjects ranged from 6.1 to 116 ng/ml; for CPS patients it ranged from 8.2 to 138 ng/ml; for ECT subjects it ranged from 37.8 to 200 ng/ml. and for non ECT psychotic patients it ranged from 1.4 to 46.4 ng/ml.

The 24 hour post ictal PRL level (P2) ranged for m4.1 to 89.4 ng/ml for GME, 4.7 to 114 ng/ml for CPS, 3.3 to 52.5 ng/ml for ECT and 5.3 to 17.5 ng/ml for non ECT patients.

The mean (± SD) post ictal serum PRL level (P1) for those with GME was 53.7± 38.9 ng/ml versus 65.9 ± 42.9 ng/ml for GME subgroup, it decreased to a mean post ictal (P2) peak value of 17.3 ± 14.8 for GME subgroup and 13.3 ± 14.2 ECT subgroup. The mean post ictal PRL values for induced seizures after ECT were not significantly higher than the mean values for spontaneous GM seizures.

The mean (+/-SD) post ictal serum PRL level (P1) for those with CPS was 44 +/- 40 ng/ml, versus 12.7 +/- 9.4 ng/ml for non ECT psychotic patients with a highly significant value ($P<0.001$). It decreased to a mean post ictal peak value (P2) 23.2 ± 31.6 ng/ml for CPS subgroup and mild decrease for the non-ECT psychotic subgroup without significant statistical value (Table 1).

Comparing the mean value of PRL levels between subgroup of either epilepsy or psychotic patients (Table 2), we found that there was no significant increase mean values for GME than for CPS (P1 & P2), but the mean value of PRL (P1) for ECT subgroup was significantly higher than mean values for non ECT psychotic subgroup ($P < 0.001$).

Comparing the mean value of post ictal and baseline levels for each subgroup revealed highly significant increasing values ($P<0.001$) for GME, ECT and CPS subgroups but not for non ECT psychotic subgroup (Table 3 and figure 1&2).

PRL value 20 minutes post ictally (P1) was above normal level (>23 ng/ml), in 20 (64.5%) GME patients while it was abnormal in all 19 (100%) ECT patient but without a statistically significant difference (($P < 0.580$). It was abnormal in 16 (72.7%) of CPS subgroup while it was abnormal in only 4(10%) of non-ECT psychotic subgroup with a highly statistical significant value ($P < 0.001$) (Table 4).

In comparing the PRL level of (P1) after seizure or episodes between the subgroups of either epilepsy or psychosis it resulted in, no significant difference between GME and CPS subgroups whereas there was a high significant value ($P<0.001$) between ECT and non ECT psychotic subgroups (Table 5).

Regarding the positive PRL value for the subgroups, PRL level of (P1) was more than (P2) by at lest one-fold after GME in 31 (100%) patients, and after ECT in 19 (100%) patients without significant statistical value. Also, the increase of PRL after CPS in 22 (100%) patients, and with non-ECT psychotic episodes in 4 (10%) patients had high significant statistical value ($P<0.0007$). PRL value (P1) increased at least twofold over baseline value (P2) after GME in 21 (67.7%) and after ECT in 19 (100%) also without apparent significant statistical value. It increase after CPS in 11 (50%) and with non-ECT psychotic episodes in 4 (10%) with significant statistical value $P< 0.07$). PRL values (P1) increased at least three -fold over baseline value (P2) after GME 10 (32.3%), and after ECT 17 (89.5%) with no apparent statistical value. It increase in CPS 5(22.7%) and in non ECT psychosis 3 (7.5%) also with no apparent statistical value (Table 6).

Comparing the positivity between GME and CPS as epileptic subgroups revealed no significant statistical values, but the comparison between ECT and non ECT psychotic subgroups revealed highly significant statistical values at one, two or threefold increase of P1 over P2 ($P<0.0004$, and ($P< 0.0003$ respectively) (Table 7).

The 4 cases of the non-ECT psychotic subgroup had abnormal PRL levels detected within 20 minutes of the abnormal behavioral episodes (P1). The abnormal levels were (46.3, 42.5, 33.5 and 31.5 ng/ml) respectively. Those 4 cases had positive increase of PRL levels by the criteria of elevation of (P1) above 23 ng/ml, one-fold elevation and two-fold elevation above baseline (P2). The EEGs of the non ECT psychotic subgroup (40 cases) were abnormal in 5 (12.5%) cases. Three cases out of the 4 patients who had abnormal PRL (P1) had abnormalities in the form of epileptic discharges in the temporal region in 2 cases and in fronto-temporal region in one case. The other 2 cases which had normal PRL (P1) had generalized slowness in one case and non specific dysrhythmia in fronto –temporal region in another case.

Table (1): Mean values of prolactin levels in the convulsive and non-convulsive subgroups.

Group / PRL (ng/ml)		Convulsive				Non convulsive			
		GME N=31	ECT N=19	"t"	2-tail sig	CPS N=22	NON ECT N=40	"t"	2-tail sig
P1 (20min	Mean ±S.D	53.7 38.9	65.9 43.0	1.10	0.287	44.0 40.0	12.7 9.4	4.30	0.000
P2 (24hr)	Mean ±S.D	17.3 14.8	13.3 14.3	0.81	0.428	23.2 31.6	11.7 2.5	1.95	0.060

PRL: Prolactin level; P1: Prolactin level 20 minutes after seizures; P2: Prolactin level 24 hours after seizures; GME: grand mal epilepsy; CPS: Complex partial seizures; "t": test; SD: Standard deviation.
ECT: Electro convulsive therapy;

Table (2): Mean values of prolactin levels in epileptic and psychotic subgroups

Group / PRL (ng/ml)		Epilepsy				Psychosis			
		GTC N=31	CPS N=22	"t"	2-tail sig	ECT N=19	NON ECT N=40	"t"	2-tail sig
P1	Mean ±S.D	53.7 38.9	44.0 40.0	-1.23	0.233	65.9 43.0	11.7 10.2	5.16	0.000
P2	Mean ±S.D	20.1 10.8	20.7 29.7	0.08	0.937	13.3 14.3	12.6 2.4	0.24	0.814

Table (3): Mean values of prolactin levels in each subgroup.

PRL Level	PRL (P1) 20 min.	PRL (P2)24 hr.	"t"	2-tail sig
Subgroup	**Mean ± SD**	**Mean ± SD**		
GME	53.7 ± 38.91	17.3 ± 14.8	5.28	0.000
ECT	65.9 ± 42.98	13.3 ± 14.3	7.52	0.000
CPS	44.0 ± 40.04	23.2 ± 31.6	5.76	0.000
NON ECT	12.7 ± 9.35	11.7 ± 2.5	0.62	0.536

Table (4): Correlation between Convulsive and Non Convulsive groups regarding PRL level 20 minutes post ictus.

Group / PRL 20 min Post ictal	Convulsive				Non convulsive			
	GME N=31	ECT N=19	X2	P	CPS N=22	NON ECT N=40	X2	P
Normal up to 23 ng/ml	11 (35.5%)	0 (0%)	1.72	0.189	6 (27.3%)	36 (90%)	4.69	0.030
Abnormal > 23 ng/ml	20 (64.5%)	19 (100%)	0.31	0.580	16 (72.7%)	4 (10%)	10.33	0.001

Table (5): Correlation between Epilepsy and Psychosis regarding PRL level 20 minutes post ictus.

Group	Epilepsy				Psychosis			
PRL 20 min Post ictus	**GME N=31**	**CPS N=22**	**X2**	**P**	**ECT N=19**	**NON ECT N=40**	**X2**	**P**
Normal up to 23 ng/ml	11 (35.5%)	6 (27.3 %)	0.03	0.864	0 (0 %)	36 (90%)	7.66	0.005
Abnormal > 23 ng/ml	20 (64.5%)	16 (72.7%)	0.05	0.955	19 (100 %)	4 (10%)	12.41	0.0004

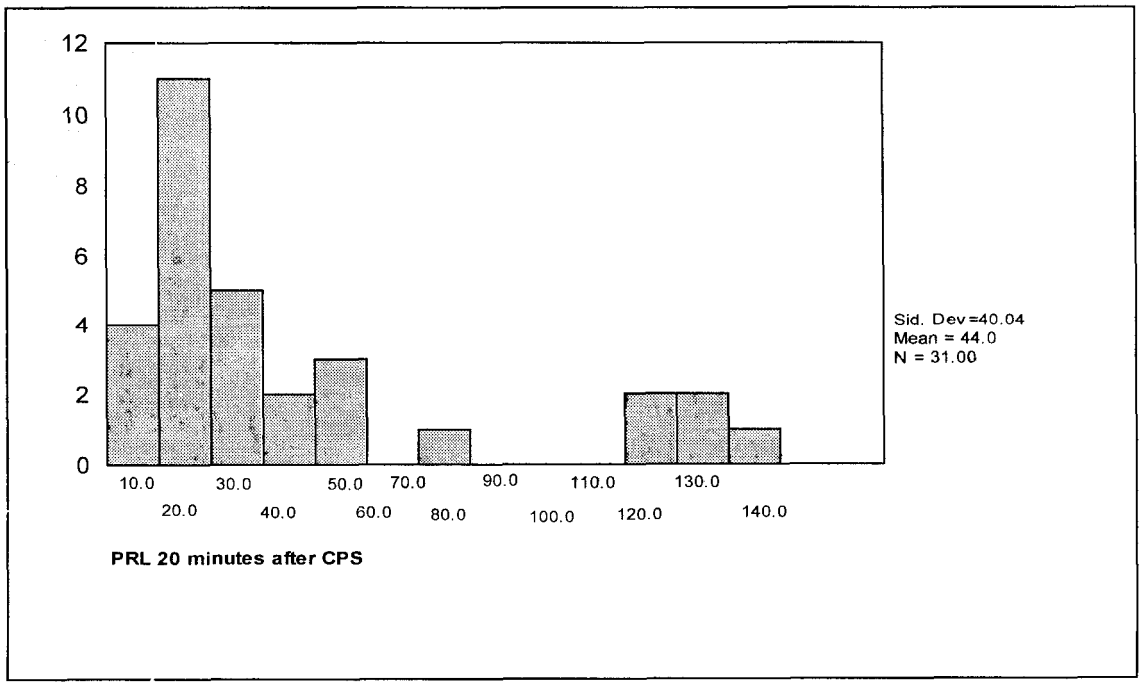
Table (6): PRL values in convulsive and non-convulsive groups

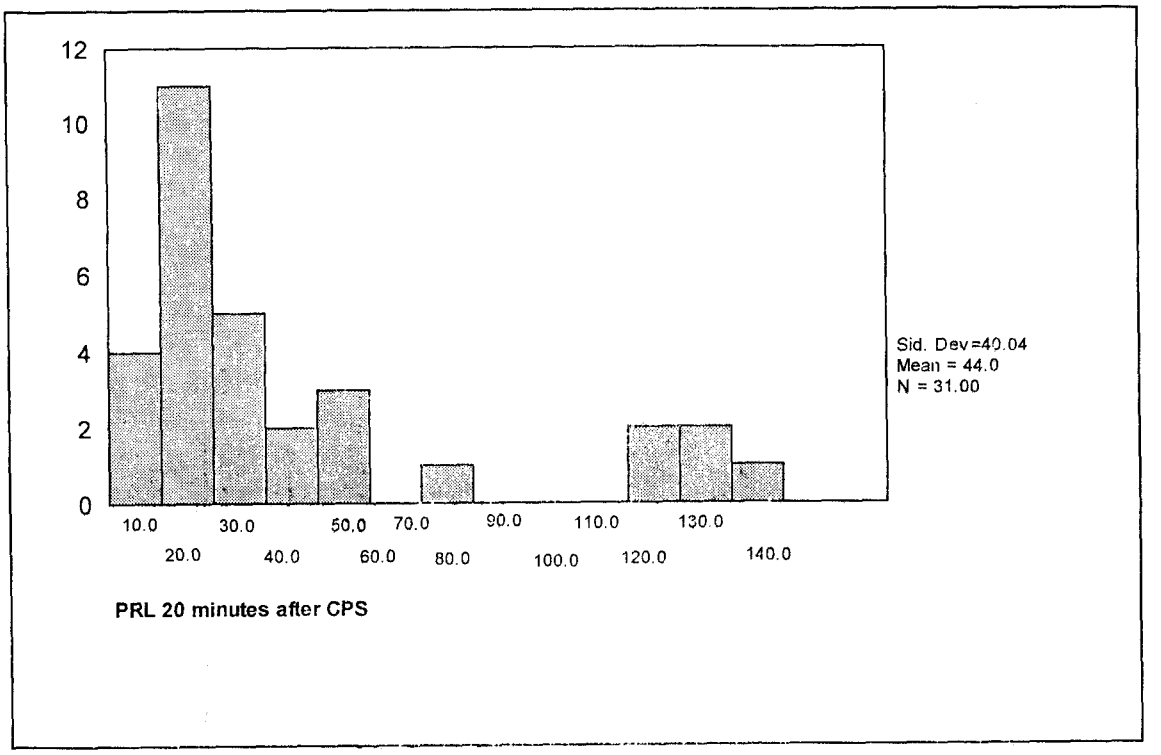
Groups	Convulsive				Non convulsive			
+ve PRL P1/P2	**GME N=31**	**ECT N=19**	**X2**	**P**	**CPS N=22**	**NON ECT N=40**	**X2**	**P**
> 1 time	31 (100%)	19 (100%)	0.01	0.942	22 (100%)	4 (10%)	15.78	0.0007
> 2 times	21 (67.7%)	19 (100%)	0.21	0.650	11 (50%)	4 (10%)	5.60	0.017
> 3 times	10 (32.3%)	17 (89.47%)	2.71	0.099	5 (22.73%)	3 (7.5%)	1.19	0.275

+ve PRL: increase P1 over P2 to indicate true epilepsy; > 1 Time: P1/P2 more than one time; >Times: P1/P2 more than 2 times; >3 Times : P1/P2 more than 3times; P:P Value; x2: Chi square.

Table (7): Positive PRL values in Epileptic and Psychotic groups.

Groups	Epilepsy				Psychosis			
+ve PRL P1/P2	GME N=31	CPS N=22	X2	P	ECT N=19	NON ECT N=40	X2	P
> 1 time	31 (100%)	22 (100%)	0.04	0.843	19 (100%)	4 (10%)	12.41	0.0004
> 2 times	21 (67.7%)	11 (50%)	0.18	0.676	19 (100%)	4 (10%)	12.41	0.0004
> 3 times	10 (32.3%)	5 (22.73%)	0.07	0.726	17 (89.47%)	3 (7.5%)	12.94	0.0003

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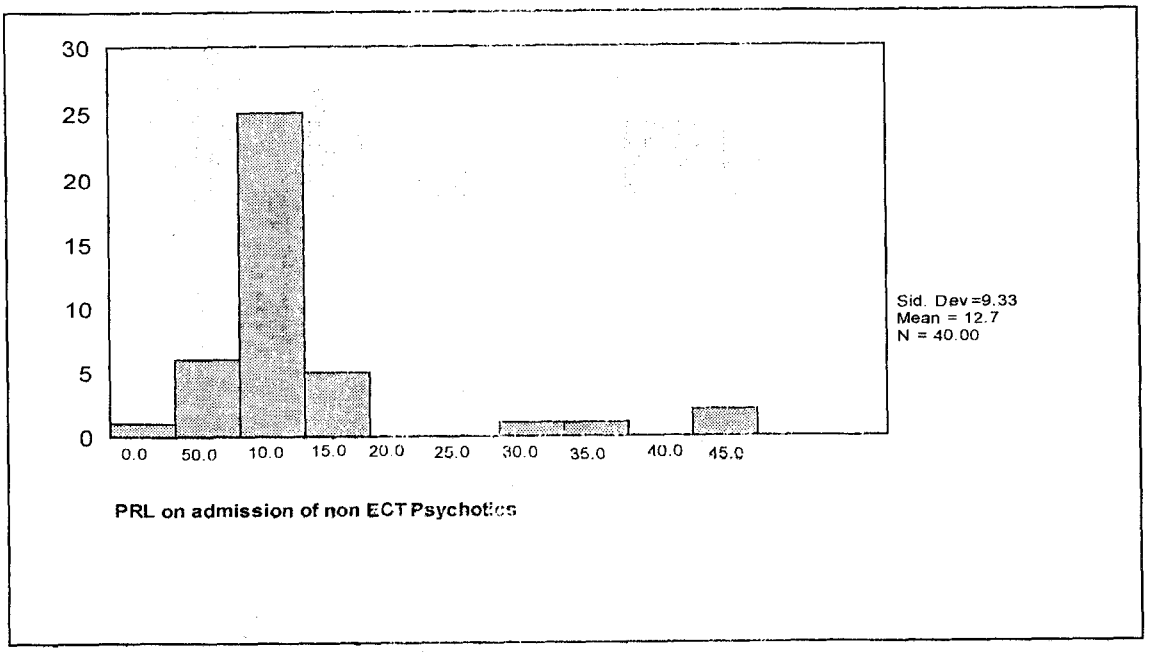
PRL 20 minutes after CPS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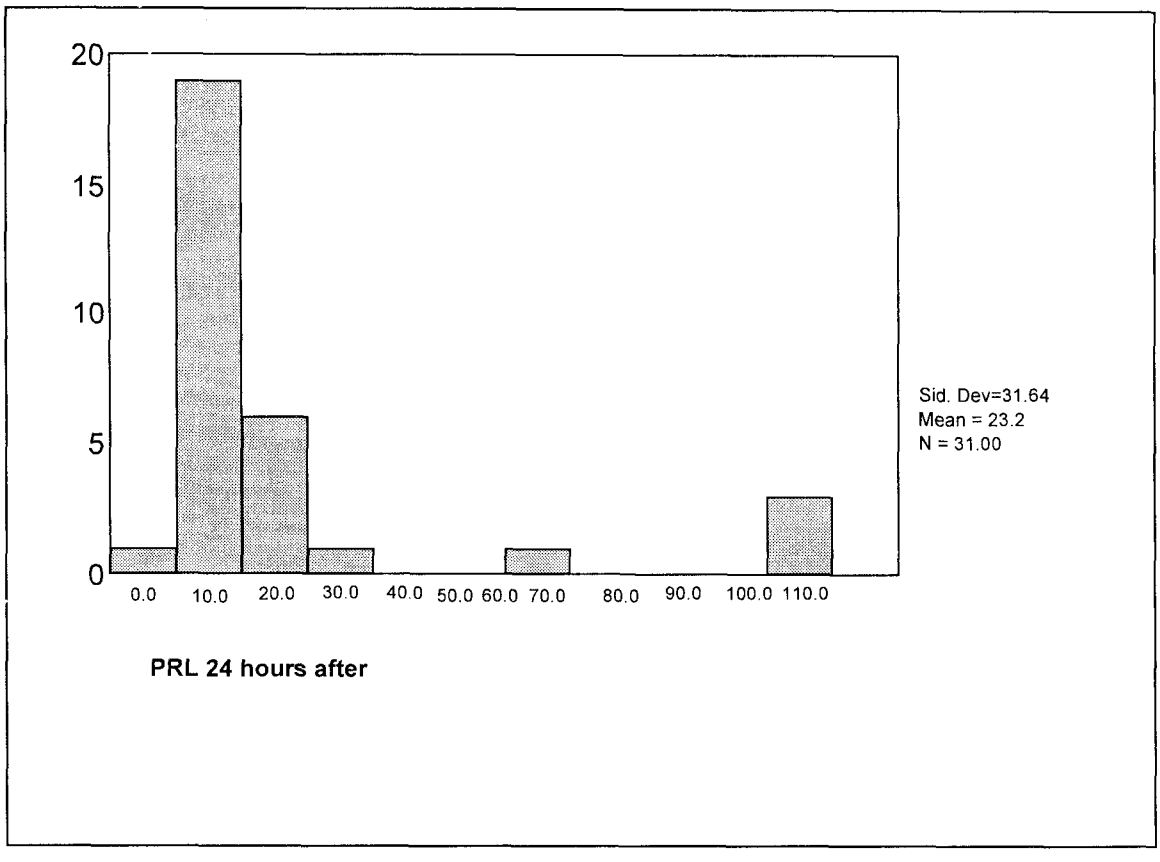
Mean = 12.7
N = 40.00
PRL on admission of non ECT Psychotics



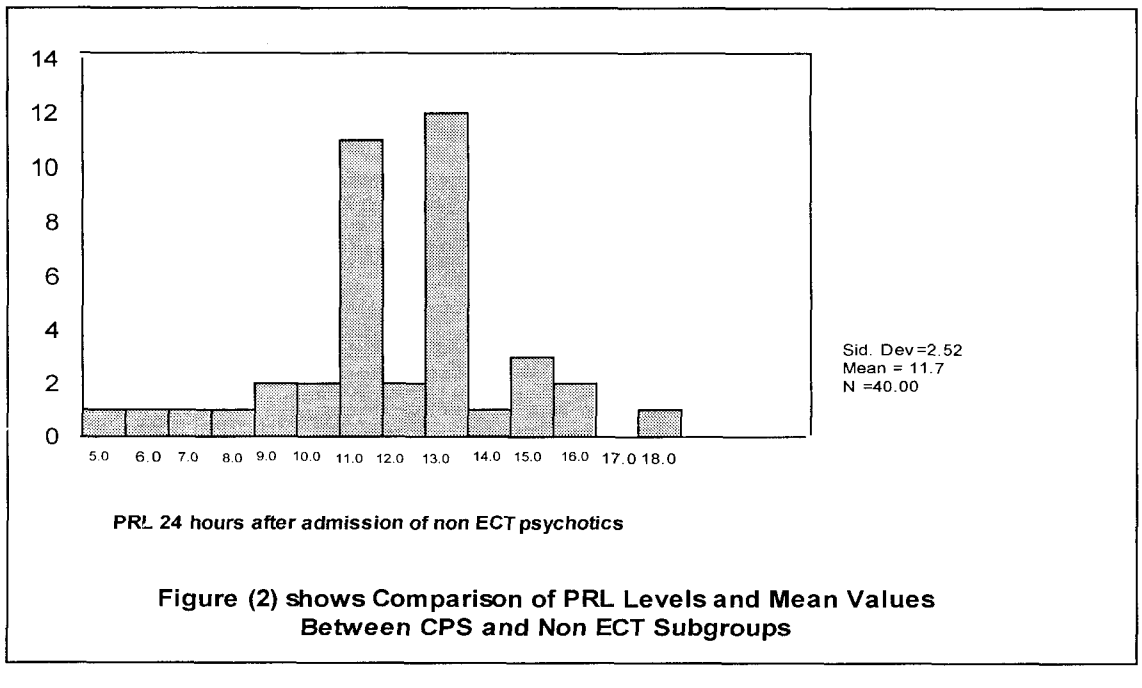


Figure (2) shows Comparison of PRL Levels and Mean Values Between CPS and Non ECT Subgroups

Discussion

Patients attending to the neuropsychiatric clinic or ER presenting with bouts of abnormal behavioral changes of psychotic nature are usually treated with anti-psychotic drugs or ECT. Our hypothesis in this study states that, some of these patients may have CPS of temporal lobe origin and can be treated by anti-epileptic drugs.

Since the initial report of Trimble (1978), many other investigators have confirmed plasma PRL increase after GME, CPS and ECT, but not after pseudo-seizures or acute stressing events. Therefore, a cut off criterion is needed to classify PRL concentration as normal or pathological (Gian, 1933).

In our study, patients had different mean PRL levels 20 minutes after seizures or episodic behavioral changes. Patients with GME had mean PRL level 53.7±38.9 ng/ml., while psychotic patients received ECT had 65.9±43 ng/ml 20 minutes after cessation of the induced seizures. The higher mean PRL level of ECT subgroup may be attributed to increase duration of physical activity of convulsive intensity during ECT.

Parra et al. (1980) have suggested that endocrine changes and prolactin release are not a function of the physical activity associated with seizure because patients with non convulsive (CPS) had post ictal elevation of PRL. But Dana et al, (1983), and Swartz, (1984), have reported that PRL response depend on the extent or intensity of the seizure, and PRL ratio may be related more to the fit itself than the amount of electricity.

Our CPS subgroup had mean PRL level 44±40 ng/ml, while non ECT psychotic subgroup had 12.7±9.4 ng/ml. our results were supported by Gian, (1993) who has reported that a blood PRL level would be directly correlated with seizure frequency and inversely correlated with measures of psychosis and thought disorders.

In this study, although we depended on the two fold increase of PRL level above baseline as an indicator of true epilepsy, we tried to compare the other cut off criteria which have been hypothesized in the previous literatures.

The PRL level was abnormal (i.e. above 23 ng/ml) in 20 (64.5%) patients of GME subgroups, in 19 (100%) patients of ECT, in 16 (72.7%) patients of CPS, while it was abnormal in only 4 (10%) patients of non ECT psychotic subgroup. These findings denote that PRL level is apparently abnormal in cases of convulsive seizures either spontaneous (GME) or induced (ECT), or even in non convulsive seizure (CPS), but not in non epileptic non convulsive behavioral condition (non ECT psychosis). This agree with Yerby et al, (1987) who proposed an upper limits of 23 ng/ml for diagnosis of epileptic seizure.

Despite the previous valuable results, the PRL levels are usually stable in a given individual but differ between individuals. Jozef (1998) had mentioned that normal serum prolactin concentrations vary considerable among individuals and it also shows pronounced circadian variation (up to four fold), peaking during sleep. It also exhibits modest seasonal variation, peaking during spring / summer S, in our study we compared the ratio between PRL level 20 minutes after a seizure or episodic behavior (P1) to the baseline PRL level after 24 hours (P2) of

the same individual. It was more than one –fold above the baseline for all patients with GME 31 (100%), with ECT 19 (100%), and with CPS 22 (100%), but it was more than one time in only 4 (10%) patient of non ECT psychotic subgroup When we applied the criterion of the two-fold increase PRL level above baseline, we found that the number of cases compatible with this rule are 21 (67.7%) of GME subgroups, 11 (50%) of CPS, while it occurred in 19 (100%) patients of ECT. But the non ECT psychotic subgroup still has the lowest number of patients as only 4 (10%) patients obey this rule. Robert et al, 1991 found that PrL levels were increased above two-fold baseline in (56%) of patients with GME and in (14%) of patients with CPS.

When we tried to apply the criterion of three-fold increase of (P1) over (P2), we found that 10(32.3%) patients with GME, 15 (89.5%) with ECT and 5 (22.8%) with CPS had the positive criteria for true epilepsy but only 3(7.5%) of non ECT psychotic patient had this positivity.

From the previous results after application of each of the three types of cut off criteria, we found that the non ECT psychotic subgroup had the lowest number of cases characterized by positivity of P1/P2 to diagnose epilepsy. For the other subgroups, there was mild difference, being higher when more than one –fold of baseline was applied and lesser when more than three-fold was applied. This agrees with Yerby et al, (1987) who has mentioned that some investigator proposed two to three-fold the baseline prolactin level s one of the criteria for the diagnosis of a seizure.

In this study, despite of 4 (10%) of 40 of non ECT psychotic patients had positive indicator of epilepsy by applying the cut off point of abnormal (>23 ng/ml) PRL level or one-fold or two-fold increase of PRL level above baseline, only 3(7.5%) patients had the positivity by using the three-fold increase over baseline. Three out of the four non ECT psychotic patients who had raised PRL level, had abnormal inter-ictal EEGs confirming the hypothesis that those patients have episodic behavior disturbance due to CPS instead of Psychotic disorders. The fourth non ECT psychotic patient who had increase of PRL level but with normal EEG may also had CPS. This agree with Adams et al, (1997) who reported that EEG during behavior episodes may show no seizure discharges. He added that, this does not exclude repeated or sustained seizure activity in the amygdala and other deep temporal lobe structures. Moreover mental state of these 4 cases on admission showed evidence of confusion, impulsivity and unprovoked violent behavior.

Another 2 out of 36 non ECT patients who had psychotic behavioral changes had abnormal EEG despite no increase of PRL level above baseline. Those 2 patients may also had CPS but of non limbic origin. This agree with Wroe et al, (1989), who has mentioned that positive serum level of prolactin is highly predictive of seizure but a negative serum PRL is not highly predictive of pseudo seizures. PRL levels are not expected to rise after seizures with brief (<10 seconds) or no impairment of consciousness.

Conclusion

It could be concluded that although in the present study, the cut off criterion (two-fold increase of PRL) was used, our

findings suggest that the cut off window of the PRL test may be extended to one-fold elevation above baseline, which increases the usefulness of the test for clinical purpose. Also, we can conclude that, a positive result is highly predictive of seizures, whereas a negative result is only weakly predictive of psychosis. This conclusion is agreed with in the literature of Yerby et al, (1987).

Our study also suggests that PRL release may be an important variable influenced by behavioral changes of epileptic nature and can be useful in differentiating between epileptic and psychotic behavioral changes. Further studies may be required to clarify the border in this neuro-psychiatric zone.

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(القيمة التنبئية لمستوى هرمون البرولاكتين في الدم
في التشخيص التفريقي بين الصرع الحوفي واضطرابات السلوك الذهاني)

أجريت هذه الدارسة لتقييم فائدة قياس مستوى هرمون البرولاكتين بالدم في التفريق للسلوك غير الطبيعي بين كل من مرضى الصرع والذهان.

تمت الدراسة على عينة من (١١٢) مريضا قد أدخلوا قسمي طب الأعصاب والطب النفسي في مدة عام. وشملت هذه العينة على (٥٣) مريضا يعانون من مرض الصرع، منهم (٣١) مريضا يعانون من حالات الصرع الكبرى ، (٢٢)

يعانون من الصرع الجزئي المركب، كما شملت العينة على (٥٩) مريض يعانون من مرض الذهان ، منهم (١٩) مريض قد عولجوا بواسطة المعالجة بالتخليج الكهربائي ، (٤٠) مريض لم يعالجوا به.

وقد تم تقدير نسبة هرمون البرولاكتين في الدم بعد ٢٠ دقيقة من انتهاء النوبة التشنجية أو نوبة الاضطراب السلوكي.. ثم تم تقدير نسبة الهرمون مرة أخرى بعد ٢٤ ساعة من العينة الأولى كعينة مرجعية (أساسية).

وبتطبيق خاصية زيادة نسبة الهرمون في العينة الأولى عن العينة الثانية بمقدار الضعف فقد تم التعرف على الحالات الصرعية عند ٢١ (٦٧,٧٪) من ٣١ حالة صرع كبرى ، وعند ١١ (٥٠٪) من ٢٢ حالة صرع جزئي مركب ، وعند ١٩(١٠٠٪) من ١٩ مريض بالذهان عولجوا بواسطة المعالجة بالتخليج الكهربائي ، وعند ٤(١٠٪) حالات من ٤٠ مريض بالذهان ولم يعالجوا بالمعالجة بالتخليج الكهربائي.

من هذه الدراسة نستنتج أن خاصية زيادة هرمون البرولاكتين بالدم ذات قيمة تشخيصية تفريقية عالية لحالات اضطراب السلوك بين كل من مرضى الصرع الذهاني.