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DEXAMETHASONE SUPRESSION TEST IN DEPRESSIVES TREATED WITH ECT.

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SUMMARY

A cohort of endogenous depressives and normal controls were studied to examine the role of DST in depressives treated with ECT. Weekly DST estimation was done and depression was assessed on HRS-D. 73.6% patients were found to be non suppressors as compared to controls in whom 16.7% were non suppressors. 60% DST positive patients showed clinical improvement while 50% DST negative did not show improvement. This proves the notion that DST is a state dependent biological marker of endogenous depressed state.

Introduction

The advancement in biological psychiatry has led to many exciting developments; the most important till date has been the premise to diagnose some melancholics by a simple laboratory test: the Dexamethasone Suppression Test (DST). The pioneer work in this field was done by D.B.J. Carroll who had described it as a laboratory marker of endogenous depressed state (Carroll et al 1980). The evidence for its state relationship is fairly strong. Response to DST is non suppressive during melancholic episode and suppressive during euthymion (Caroll 1972, Greden et al 1980.)

DST can be used in the monitoring of antidepressant treatment (Greden et al 1983), predicting new episodes (Carroll 1987), diagnosing affective disorders in early childhood (Greden and Carroll 1979), masked depression and pseudodementia (Grunchams et al, 1983).

The DST tends to normalize as depression subsides (Albala et al 1981, Devanand et al 1987) and may even normalize well before clinical improvement which may have a predictive value for good treatment response.

A favourable electroconvulsive therapy (ECT) response was predicted when an abnormal DST result was noted at discharge (Coryell 1982). However, Coryell and Zimmerman (1983) suggested that ECT may be having immediate effect on hypothalmic structures which invalidate use of DST as a predictor of response.

The present study was done to find out the relationship between DST and clinical improvement of depressives treated with ECT.

Material and Methods

The patients of the experimental group comprised of 30 patients (drawn from In-patients Department of Psychia-

try, K.G's. Medical College, Lucknow) diagnosed as Manic Depressive Psychosis – depression (ICD) and fulfilling the Research Diagnostic Criteria (Spitzer et al 1978) for Major Depression. The sample were between the ages of 17 – 55 years and were evaluated on a present selection criteria. Thirty normal controls were also studied.

The patients of the experimental group were evaluated on Hamilton Psychiatric Rating Scale for depression (HRS-D) (Hamilton 1960) and those having a score of 17 or more were further studied. The patients were given biweekly direct ECT's upto a maximum of 10 treatments. However, the treatment was stopped when HRS-D levels equalised 5 or fell below it.

DST estimation was done by giving 1 mg of dexa-methasone to the patients at 11 p.m. followed by sampling of blood at 4 p.m. on the following day. The plasma was separated and cortisol levels was estimated by spectro-flurometric method (Mattingloy 1962). The DST (cut off 13 mg/dl) was done one day and, thereafter on day 7, 14, 21, 28 and 35. The ECT was stopped when the patient recorded 5 or less on HRS-D. The DST was estimated 72 hours after the last ECT so as to exclude any possibility of an increased cortisol levels due to ECT itself.

The control group consisted of 30 normal subjects, who were evaluated clinically, as well as, on Cornell Medical Index, to exclude any possibility of psychiatric illness. Only one point DST of the control group was done.

Apart from giving nitrazepam on an SOS basis no extra medication was given to the patients.

Results

Thirty patients each of the experimental and control group completed the study. The mean age of the experimental group was 38.6 years (range 20 – 55 years) while it was 32.9 years (range 20 – 49 years) for the controls. In majority of the patients the illness started between 26 and 45 years when they had their first episode. 60% patients had no past history of affective illness. The mean plasma cortisol levels of the depressed and control group was 18.9 and 11.9 mg/Dl respectively. No correlation was found between age and plasma cortisol levels (Table 1).

Table 1
DST in Experimental and Control Group

	Experimental Group N = 30		Control N = 30	
	No.	%	No.	%
Suppressors	8	26.7	25	83.3
Non Suppressor	22	73.3	5	16.7

Thirty depressed patients and thirty controls were compared for DST. 73.3% of depressed patients were non suppressors prior to treatment while only 16.7% of controls were non suppressors. 26.7% of the patients were suppressors of DST, as compared to 83.3% that of controls.

76.9% patients were males while 23.1% were females and in both groups about one third were suppressors on DST which shows that DST is not dependent on sex. When DST was compared with the clinical improvement (HRS-D) in the improved group at the beginning and at the end of the study it was found that 82.6% of the patients were non suppressors on DST and their mean HRS-D was 29.1 and at the end 30.4% were non suppressors (Table 2).

When the above comparison was done in the non improved group it was found that 57.1% patients were suppressors on DST with the corresponding mean HRS-D of 28.7 while at the end of the study

Table 2
DST and clinical improvement of the patients of improved and non improved group

Group		Improved Group N = 23		Non Improved Group N = 7	
		Pre Tt. %	At the end No.	Pre Tt.	At the end No.
Suppressor	No.	4(17.4%)	16(69.6%)	4(57.1%)	7(100%)
	HRS-D (Mean)	20.5	3.5	28.7	13.2
Non Suppressor	No.	19(82.6%)	7(30.4%)	3(42.9%)	0
	HRS-D (Mean)	29.1	14.1	26.2	

100% patients were suppressors with mean HRS-D of 13.3

26.7% patients were suppressors (13.3% each of improved and non improved group) before starting the treatment and they remained suppressors till the end of the study. Whereas 73.3% were non suppressors prior to treatment out of which one sixth were in the non improved group while at the end of the study 23.1% were still non suppressors and they were those patients who belonged to the improved group.

23 patients of the experimental group improved with ECT while 7 did not improve. 15 patients (50%) required 5 – 6 ECTs for improvement, 6 patients (20%) required 7 – 8 ECTs while 2 patients required 9 – 10 ECTs. 7 patients (23.3%) however, did not show improvement even after 10 ECTs.

Discussion

There are several reports relating to DST and depression but only few reports are available relating to DST, depression and ECT especially in India. Most of the studies have evaluated DST on depressives treated with antidepressant drugs. As endogenous depressives show good response to ECT, even quicker than antidepressant drugs, we had planned to study the role of DST in depressives treated when direct ECTs only.

Several methods of cortisol estimations are available, like calorimetry, flurometry, radio immunossay and competitive protein binding methods. Spectrofluometric methods was employed by Carroll et al (1968) and Carroll and Davies (1970). The present study also employed spectro-fluorometric method as it was the only available method till then, in the local laboratories. We have taken 13 mg/dl as cut off point for DST (Varma 1985).

There was no significant correlation found between age and post dexamethasone plasma cortisol level in this study. Similar findings have been reported by other workers. (Greden et al 1983, Carroll and Mendels 1976, Carroll et al 1981) which indicate that patients of depression could be identified irrespective of age. A positive correlation was observed between age and DST in patients above the age of 50 years (Sachar et al 1975) leading to a possibility that they could be hypersecretors of cortisol.

It was also found that the number of episodes of depression (single and multiple) do not have a significant bearing on the DST which was also reported by

Schlesser et al (1980) and Nelson et al (1982). This findings supports the notion that DST is a state dependent rather than trait dependent biological marker.

83.3% of the normal controls were suppressors on DST while only 16.7% were non suppressors. This is in agreement with several studies (Carroll et al 1976). Amserdam et al (1982) who have found non suppressor DST in 4 – 16% of the normal population which suggests that they may be hypersecretors of cortisol due to factors other than depression. On the other hand in the depressed group 73.3% patients were non suppressors suggesting an hyperactive H-P-A axis activity. This finding is supported by many studies.

When both the improved and non improved group patients were evaluated, it was found that 73.3% were non suppressors prior to treatment while 23.1% were still non suppressors at the end of the study and they were those patients who belonged to the improved group (Table 2). Out of the 26.7% who were suppressors, 13.3% went on to show improvement and still they remained suppressors in the end also. These findings go on to prove a point that atleast 60% patients who were DST positive initially, will show reversal of DST on clinical improvement thus confirming the notion that DST is a state related biological marker. Moreover, there is a possibility that, a prediction could be made in about two-third of the patients of non suppressor group, that they will show clinical improvement.

Among the suppressor group about 50% patients did not show clinical improvement, nor change in DST status. This also proves the above point by a negative statement that about 50% DST negative patients would not show any change in their clinical or DST status.

Summarising the findings we can say that DST is a useful state dependent biological marker in about 50-60% patients of endogenous depression but its predictive value about treatment response awaits more research.

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