SERUM PROLACTIN LEVEL AFTER ELECTRO-CONVULSIVE THERAPY, IN STATES OF DEPRESSION AND SCHIZOPHRENIA

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20 depressive patients (Major type) and 13 schizophrenic patients were studied as regards the quantitative rise of serum prolactin following electro-convulsive therapy. 4 of the schizophrenic group were given neuroleptics before E.C.T.

Serum prolactin increased 9.6 times its basal level in depressive patients, 6.9 times in schizophrenia without medication, after E.C.T. In schizophrenics who received neuroleptics alone, serum prolactin reached 7 times its basal value. Schizophrenics who received both E.C.T. and neuroleptics, showed rise of serum prolactin 18.4 times its basal level.

Depressive patients and schizophrenic patients who received both E.C.T. and neuroleptics showed rapid and better improvement.

Degree of serum prolactin rise may be considered as a pointer to better response and improvement.

Introduction

The total amount of hypothalamic stimulation induced by an E.C.T. seizure is presumably reflected by the total quantity of prolactin released (Swartz and Abrams, 1984).


Gregory and Shawcross (1985), postulated that unilateral E.C.T. is probably slower than that of bilateral. They concluded that these differences remain a matter of speculation.

Neuroleptic drugs elevate plasma prolactin by blocking dopamine receptors.

It was found that associating E.C.T. with pharmaco-therapy in schizophrenia
gave better results than giving either neuroleptics or E.C.T. alone (Tailor and Fleminger, 1980).

The aim of this study is to investigate whether there is a difference between depressive and schizophrenic patients in their prolactin response after E.C.T. Also to study the quantitative increase of prolactin of schizophrenic patients previously having neuroleptics. Such investigation is a trial on the way to know the possible mechanisms of action of E.C.T. on the human brain.

Material and Methods

20 patients suffering from Major Depression and 13 patients suffering from schizophrenic disorders were selected according to D.S.M. III* criteria for diagnosis. All patients were drug free at least one month before the trial. Basal serum prolactin was taken before applying the E.C.T. therapy or giving any drug. Serum prolactin was determined by a specific double antibody radio-immunoassay using I₂₅ as a tracer (Reagent Kits were obtained from D.P.C. «Diagnostic Product Corporation» U.S.A.).

All depressed patients (20) as well as 9 schizophrenic patients were given bilateral E.C.T. 4 schizophrenic patients were given neuroleptic treatment equivalent to 300 mg. chlorpromazine per day before starting the course of E.C.T. The base line blood samples were drawn before giving anaesthesia.

Bilateral E.C.T. was administered through standard bi-frontotemporal electrodes. In each case a pulsed bi-directional squarewave stimulating current of 70 Hz and 1.5 ms pulse width was passed for two seconds, resulting in a fully generalized bilateral tonic-clonic grand mal seizure.

The second blood sample for prolactin was obtained 15 minutes after termination of visible muscular convulsive activity. Results were subjected to statistical analysis.

Results

Depressive patients were 20 with a mean age 28.8 (S.D. = 14.27) and mean duration of illness 1.67 years (S.D. = 1.76). Schizophrenic patients were 13. Their mean age was 31.23 (S.D. = 7.1) and mean duration of illness was 3.63 (S.D. = 2.34). 6 patients were of chronic type and 7 patients were in relapse with previous history of recurrent episodes. (Table 1).

Serum prolactin increased significantly (P < 0.0005) above basal level in cases of depression after E.C.T. (Table 2).

In cases of schizophrenia there was as well a significant increase of serum prolactin (P < 0.0005) after E.C.T., as well as after giving neuroleptic and before giving E.C.T. (Table 3).

Those schizophrenic patients who were on neuroleptics and given as well E.C.T., showed another significant rise (P < 0.01) of serum prolactin over the already high value due to neuroleptics.

Table 4, showed that rise of serum prolactin in cases of depression after giving

* DSM III : The third edition of the American Psychiatric Association's Diagnostic and Statistical Manual of Mental Disorders.
E.C.T. reached 9.6 times its basal value, while that in cases of schizophrenia without medication reached only 6.9 times its basal value. Those schizophrenic patients who received neuroleptics and before E.C.T. showed a rise of serum prolactin 7 times its basal value.

Schizophrenic patients who received both a neuroleptic and E.C.T. showed rise of serum prolactin 18.4 times its basal level.

### Table 1)

**Description of Patients**

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Age Mean (S.D.)</th>
<th>Duration Mean (S.D.)</th>
<th>Other Clinical Data</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>20 (28.8)</td>
<td>14.27 (1.67)</td>
<td>1.76</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>13 (31.23)</td>
<td>7.11 (3.63)</td>
<td>2.34</td>
</tr>
</tbody>
</table>

4 Patients received neuroleptic treatment equivalent to 300 mg. chlorpromazine before E.C.T.

### Table (2)

**Serum PRL (ng/ml) In Cases of Depression Before and After ECT**

<table>
<thead>
<tr>
<th></th>
<th>Before E.C.T.</th>
<th>After E.C.T.</th>
</tr>
</thead>
<tbody>
<tr>
<td>No.</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Mean</td>
<td>5.78</td>
<td>55.30</td>
</tr>
<tr>
<td>S.D.</td>
<td>4.54</td>
<td>45.64</td>
</tr>
<tr>
<td>S.E.M.</td>
<td>1.015</td>
<td>10.205</td>
</tr>
</tbody>
</table>

\[
t_{df} = 4.8287/18 \\
P < 0.0005
\]
Table (3)
Serum PRL (ng/ml) in Cases of Schizophrenia Before & After ECT

<table>
<thead>
<tr>
<th>Group</th>
<th>Before E.C.T. No CPZ</th>
<th>Before E.C.T. +CPZ</th>
<th>After +ECT</th>
<th>Cor-rel.</th>
<th>t/df</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>No CPZ</td>
<td>9</td>
<td>4</td>
<td>9</td>
<td>4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean</td>
<td>8.12</td>
<td>57.3</td>
<td>56.08</td>
<td>149.5</td>
<td>5.937/16</td>
<td>&lt;0.0005</td>
</tr>
<tr>
<td>S.D.</td>
<td>5.54</td>
<td>13.5</td>
<td>23.575</td>
<td>56</td>
<td></td>
<td></td>
</tr>
<tr>
<td>S.E.M.</td>
<td>1.85</td>
<td>6.75</td>
<td>7.86</td>
<td>28</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table (4)
Changes of Serum Prolactin Before and After ECT in Schizophrenia and Depression

<table>
<thead>
<tr>
<th>Pathologic Group</th>
<th>Serum PRL After ECT without Premedication</th>
<th>Serum PRL After Premedication Before ECT</th>
<th>Serum PRL After Premedication + ECT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>9.6 Times</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>Schizophrenia</td>
<td>6.7 Times</td>
<td>7 Times</td>
<td>18.4 Times</td>
</tr>
</tbody>
</table>

Discussion

Electrical induction of convulsions in the treatment of psychiatric disorders was introduced 40 years ago by Cerletti and Bini, (Deakin, 1981).

The most common indication for E.C.T. today is the presence of a major depressive episode. 80 to 90% of such individuals will show marked improvement, a significantly higher figure than with pharmacological intervention, (Weiner, 1985).

E.C.T. was found also to be beneficial in acute schizophrenic patients especially when accompanied by catatonic or affective symptomatology. In chronic schizophrenia it may be less effective especially when they are drug non-responders.

Tailor and Fleminger, (1980), in a double blind controlled trial of E.C.T. with patients having paranoid schizophrenia using 8 to 12 E.C.T. treatments found significant greater improvement by the group
who received E.C.T. and neuroleptics more than the other group who received same treatments with neuroleptics alone.


Arato, et al., (1980), after giving E.C.T. to rats he found significant increase of plasma prolactin and decreased hypothalamic dopamine content. He concluded that prolactin response elicited by E.C.T. is due to monaminergic influence.

In our patients the increase of prolactin after E.C.T. in both Depression and schizophrenia was significant irrespective of the diagnosis or the duration of illness in both groups.

But the rise of prolactin in depressive disorders was 9.6 times its basal level as compared to 6.9 times in schizophrenia.

The effect of giving E.C.T. alone to one group of schizophrenic patients and neuroleptics to another group showed nearly similar rise of prolactin over basal one in both groups.

Giving both E.C.T. and neuroleptics showed marked rise of prolactin (18.4 times) that of the basal value.

This may indicate the complementary action of both drug and E.C.T. in drug responsive schizophrenics.

Klimes, (1978), found that even a 5-fold higher initial serum level of PRL fails to inhibit further hormone release.

Arato, (1980), found in two chronic hospitalized schizophrenics resistant to therapy that prolactin response to E.C.T. was negligible. He put the question of whether the prolactin response or its absence is of predictive value with respect to prognosis or effect of E.C.T.

In our study, after giving a course of 4 E.C.T., depressive patients and schizophrenic patients receiving as well neuroleptics showed marked improvement, while the group of schizophrenics who didn’t receive neuroleptics needed more E.C.T. to reach the same result.

This points to the real effect of E.C.T. on brain neurotransmitters and its value in depressive disorders (Major type) even without the use of any pharmacotherapy.

On the other hand, the effect of E.C.T. in schizophrenia is much potentiated by the use of neuroleptics in association.

The degree of rise prolactin may be a good indicator of a better response to E.C.T.

References


مستوى البرولاكتين بالفصل في حالات الاكتئاب والقصام بعد استخدام الجلسات الكهربائية

تمت دراسة عشرة من المرضى بالإكتئاب (النوع الكبير) وثلاث عشر مريضاً بالقصام وذلك لقياس معدل الارتفاع الكمي للبرولاكتين بالفصل بعد اعطاء الجلسات الكهربائية. وقد اعطي أربعة من المرضى الفصاميين أحد المديدات الفصامية قبل اعطاءهم الجلسة الكهربائية.

وقد ظهر أن زيادة البرولاكتين بالفصل في حالات الاكتئاب بعد اعطاء الجلسة الكهربائية بلغ 9.6 مرة قدر المستوى القاعدي، بينما زاد في حالات القسام بدون عقار إلى 9.6 مرة عن المستوى القاعدي. كما زاد في حالة العقار بدون جلسة كهربائية إلى 7 مرة قدر المستوى القاعدي.

وبعد اعطاء الجلسة للمرضى الفصاميين الذين استعملوا المديدات الفصامية، وجد أن زيادة بلغت 18.4 مرة قدر المستوى القاعدي.

وقد وجد أن حالات الاكتئاب وحالات القسام التي استخدمت الجلسات المصابة للعقار اظهرت تحسنًا سريعاً وكيماً.

ويمكن استنباط أن ارتفاع مستوى البرولاكتين بالفصل قد يكون إحدى دلائل سرعة الإستجابة والتحسين.