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Brief Communications

SCHIZOPHRENIA – ELECTROLYTE PROFILE & THE EFFECT OF TREATMENT

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Introduction

Schizophrenia is a common psychiatric disorder and various hypotheses have been put forward to explain its aetiopathogenesis. Recently, much of the efforts have been made to explain the nature of the illness on the basis of biochemical studies. Several workers (Gjessing 1938, Katzenebogen et al 1943, Coopen 1965, Yassa et al 1979) have studied serum electrolytes in Schizophrenia but with equivocal results. This has inspired us to take up the present study.

Material and Methods

60 female Residual Schizophrenics, chronic type (DSM-III 1980) (mean duration of illness 6 years; age range 18-55 years; mean age 32.62 years) were taken up for the study who came to hospital in a relapsed stage and were not on the treatment for last six weeks. Those, who were suffering from diabetes, malabsorption, dehydration or other metabolic disorders which affect serum electrolytes, from the study, after doing clinical and relevant biochemical investigations. 60 normal healthy volunteers (age & sex matched) were taken as control after taking a written consent.

The Schizophrenics (Group S) were divided groups into two – 30 patients (Group S_1) were given phenothiazines for 4 weeks and 30 patients (Group S_2) were treated with ECT's for 4 weeks and in both groups (S_1 & S_2) the doses of phenothiazines (Trifluoperazine in dosage range – 15 to 30 mg and Trihexyphenidyl in dosage range – 4 to 8 mg) and ECT (average number 4-6) were adjusted to achieve the control of symptoms.

The overnight fasting serum sodium, potassium, chloride, bicarbonate, calcium, magnesium and phosphorus were estimated in normal subjects and in Schizophrenics (before, and 4 weeks after treatment with drugs or ECT) by flame Photometer (Gradwohl 1980).

Results

There were significant increase in the concentrations of serum, sodium, potassium, chloride, calcium and magnesium in

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Table

Serum electrolytes in normal and schizophrenics (untreated and treated). (Mean ± S.D.)

Group	Sodium	Potassium	Chloride	Bicarbonate	Calcium	Magnesium	Phosphorus
Normal (N=60)	137.23 ± 2.62	3.68 ±0.35	98.68 ±3.5	20.96 ± 2.68	8.97 ±0.63	1.06 ±0.36	2.97 ±0.77
Untreated Schizophrenics (N=60)	143.82* ± 9.32	4.34* ±0.37	106.97* ± 3.98	19.14* ± 3.83	9.67* ± 0.69	2.29* ± 0.38	3.49 ±0.90
After drug treatment (S_1)	144.92 ± 13.62	4.48 ±0.42	107.60 ± 4.52	19.12 ± 3.78	8.04* ±0.89	2.02* ±0.52	3.56 ±0.92
After ECT's (S_2)	143.89 ± 13.37	4.49 ±0.45	107.56 ± 4.49	19.10 ± 3.82	8.12* ± 0.90	2.06* ±0.53	3.53 ±0.90

p value ~ *<0.001

Schizophrenics as compared to controls. A decrease was observed in serum bicarbonate whereas phosphorus levels were unaffected.

The results before start of treatment, 4 weeks after drugs or ECT's are shown in Table. There were significant fall in serum calcium and magnesium while other serum electrolytes were unaffected after treatment with drugs or ECT'S.

Discussion

In our study, we have found increased serum levels of sodium, potassium, calcium, magnesium and chloride in schizophrenics in comparison to normal subjects and the differnce was statistically significant ($p < .001$). The serum bicarbonates were low in schizophrenics in comparison to normal subjects and the difference was again statistically significant ($p < 0.001$). However there was no difference in serum phosphorous levels in schizophrenics and normal subjects ($p > .05$). This electrolyte difference shows some alteration in metabolism in schizophrenic patients. Lowett Doust (1951) showed that peripheral hypoxia in schizophrenics can result in hyperventillation and in turn cause respiratory alkalosis. This can explain the low bicarbonate levels in blood of schizophrenics. Sethi and Sethi (1971), found the increased levels of serum potassium, chloride and decrease in bicarbonate which they explained on the basis of decreased cortisol secretion.

Hoagland (1955) reported the alteration in the adrenocortical function resulting in increase in urinary sodium and potassium excretion. Alexander et al. (1978), found increased calcium levels in catatonic schizophrenics, which became normal after the treatment with fluphenazine. This is compatible with our results. Carmen et al. (1976) found a decrease in CSF and serum calcium after a successful electroconvulsive therapy (ECT) as seen in our study. Steven (1984) also found moderate disturbances in serum calcium and magnesium in children with schizophrenia and depression.

With this, it is evident that moderate degree of electrolyte imbalance is found in schizophrenics and serum electrolytes come to normal after treatment. Long term follow up study on serum electrolytes to observe any correlation between course and prognosis of schizophrenia to serum electrolyte levels would be useful.

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