ECT - side effects

CASE REPORT

Hyponatremic Seizure Following ECT*

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This report describes the investigation of a spontaneous grand mal seizure in a 55-year old woman, being treated with drugs and ECT for depression. The spontaneous seizure was due to hyponatremia caused by self-induced water intoxication, although psychotrophic medication may have contributed by lowering the seizure threshold. The diagnosis of hyponatremia is discussed.

Well recognized causes of generalized seizures among psychiatric patients include epilepsy, psychotropic drugs which lower the seizure threshold, and withdrawal states. Post-electroconvulsive treatment (ECT) states and water intoxication are less frequent causes of generalized seizures.

We describe a depressed, middle-aged woman who had a generalized seizure with prolonged post-ictal findings during a course of ECT. Our investigation of the seizure uncovered hyponatremia due to water intoxication as its cause.

Case Report

A 55-year old woman presented for admission with marked agitation, insomnia, loss of concentration and memory, dysphoria, poor appetite, and somatic complaints of abdominal burning. She was judged to be depressed (Beck Depression Inventory score 33). The patient had a lengthy history of repeated depressions requiring hospitalization. Past treatments included tricyclic and monoamine oxidase inhibiting antidepressants, neuroleptics, benzodiazepines, lithium, and three courses of ECT. Apparently, ECT had been the most effective treatment. She did not offer a history of polydipsia or polyuria.

Physical findings on admission were normal. Admission laboratory tests included: urinary specific gravity of 1.010, serum sodium 148mmol/l, normal chest and spine x-rays, and normal electrocardiogram.

There was no improvement after four weeks of treatment with trazadone (400 mg/day), L-tryptophan (2mg/day), and triazolam (.25 mg/day). She was then treated with oral flupenthixol .5mg b.i.d. and thioridazine 75 mg q.h.s., and a course of ECT was initiated.

The course of ECT was 10 modified, right unilateral, monitored treatments with an average seizure duration of 24 seconds, administered by a Mecta stimulation device. Following each treatment, she recovered from anaesthesia uneventfully within 60 minutes. After eight treatments a decrease in agitation and slight improvement in affect was noted. Twenty-four hours after the 10th ECT treatment (at 2030 hours), the patient became more agitated, vomited, and developed a generalized seizure. She had received thioridazine 50 mg p.o., chlorpromazine 50 mg p.o., and diphenhydramine 50 mg i.m. within 12 hours of the seizure.

DRUGS

Transient positive Babinski reflexes followed the seizure. Otherwise, examination was unremarkable. Serum sodium was 106 mmol/l, 50 minutes after the seizure. The urine volume was 5.4 litres during the next eight hours. Repeat electrocardiogram, chest and skull x-rays, intravenous pyelogram, brain scan, and toxic drug screen were normal. She was treated with intravenous saline.

Following recovery, she readily admitted to drinking approximately 5-10 litres of cold water per day during the hospitalization because of dry mouth and anxiety. After 15 hours without food or fluids (and after serum sodium returned to normal), the urinary specific gravity increased from 1.010 to 1.020, urine osmolality was 400 mmol/l, and serum osmolality was 283 mmol/l. At the ending of the fast, 10 units of vasopressin was given subcutaneously resulting in less than a nine percent rise in urinary osmolality. Antidiuretic hormone level was < 1.0 pmol/l (normal < 7.5 pmol/l) and neurohypophyseal function was considered normal.

The patient remained in the hospital five weeks after the spontaneous seizure. Treatment with lithium, isocarboxazid, and L-tryptophan produced some improvement in her depression, and the patient agreed to limit her water intake. Biweekly morning serum sodium levels were normal. The diagnoses at discharge were major depressive episode

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(recurrent) and hyponatremic convulsion due to polydipsia.

Discussion

Our case stimulates a discussion of the differential diagnosis of generalized seizures occurring among patients after receiving ECT. Spontaneous seizures occur following ECT in approximately 0.5 percent of cases (1). It should be noted that this is not higher than the incidence in the general population, and that improved anaesthesia and oxygenation during ECT have reduced this complication (2).

Seizures are well-known complications of water intoxication as shown by Rowntree (3) in controlled animal experiments. The prevalence of polydipsia among hospitalized psychiatric patients has been estimated to range between 6.6 and 17.5 percent with half of such patients experiencing water intoxication. Roughly 80 percent of patients with water intoxication have schizophrenia and the mortality from severe water intoxication may approach 10 percent over a two-year period (4).

Vieweg et al (5) provide a recent review of the morbidity and mortality of water intoxication. Their review suggests that symptoms of water intoxication improve with treatment of the underlying psychiatric conduction including treatment with antipsychotic drugs. However, Jones (6) has argued that antipsychotic drugs may make polydipsia worse since he believes it to be a dopaminergic supersensitivity disorder. Koczapski et al (7) report that water intoxication symptoms appear more frequently in the afternoon than in the morning. This is due to diurnal variation in sodium levels. Koczapski has also observed abnormal diurnal weight gain among chronically psychotic patients subject to water intoxication. Interestingly, our patient's seizure was during the second half of the day.

Goldman et al (8) and Illowski and Kirch (9) provide a recent review of the pathophysiology of water intoxication and hyponatremia.

Conclusion

Had we not measured serum sodium following the patient's generalized seizure, we probably would have implicated the combination of ECT and psychotropic drugs known to lower the seizure threshold. Even without a history of polydipsia and polyuria, it is incumbent upon the clinician to obtain a serum sodium measurement following a generalized seizure in a psychiatric patient.

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Résumé

Le présent rapport porte sur l'étude d'un cas de crise spontanée de grand mal chez une patiente de 55 ans traitée aux médicaments et aux électrochocs pour une dépression. La crise était due à une hyponatrémie causée par l'intoxication volontaire à l'eau. Les médicaments psychotropes ont cependant peut-être joué un rôle en abaissant le seuil du déclenchement de l'état de crise. La question du diagnostic de l'hyponatrémie est également abordée.