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Case Report

Nonconvulsive status epilepticus and Creutzfeldt–Jakob-like EEG changes in a case of lithium toxicity

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ABSTRACT

We report a case of a 63-year-old lady with bipolar affective disorder on lithium who was brought to our emergency center in a comatose state.

Neurologically, the patient was comatose and had generalized hypotonia and hyporeflexia. Lithium toxicity was considered. Laboratory examinations revealed leukocytosis, normal blood sugar, blood level of lithium was 4.7 mEq/L and she had renal dysfunction. Cerebrospinal fluid examination and cranial computerized tomography were unremarkable. Blood lithium level was 4.7 mEq/L. Hemodialysis was initiated. However, in spite of dialysis and decreasing lithium levels, the patient remained unconscious. A possibility of nonconvulsive status epilepticus was considered; hence, EEG was advised. The EEG demonstrated bihemispheric slowing (4- to 5-Hz theta range) with bilateral periodic triphasic waves of 1- to 2-Hz frequency, similar to the EEG changes seen in Creutzfeldt–Jakob disease.

She was started on lorazepam. Her sensorium improved gradually, which correlated with the decline in blood lithium levels. A normal background alpha rhythm on EEG was ensured prior to discharge. At discharge, clinically, she had recovered completely, with no apparent neurological deficit or cognitive impairment.

This case highlights the importance of therapeutic drug-level monitoring of lithium, especially where toxicity is suspected, and the important role electroencephalography plays in diagnosing NCSE and its management.

1. Introduction

Lithium is considered the drug of first choice for treating bipolar affective disorders ever since John F.J. Cade's observation of its antimanic effects in 1949 [1,2].

Lithium toxicity causes electroencephalogram (EEG) abnormalities which can be correlated with the degree of neurotoxicity. Disorganization and slowing of background activity are the most common EEG changes, while periodic complexes of sharp waves may also appear in patients with severe neurotoxicity. Cases of lithium toxicity with clinical presentation and EEG findings suggestive of Creutzfeldt–Jakob disease have been reported, with complete clinical and EEG recovery [2].

We report a case of lithium toxicity in a patient with bipolar affective disorder who was in a comatose state and who presented with nonconvulsive status epilepticus and Creutzfeldt–Jakob-like EEG changes.

2. Case history

This 63-year-old lady diagnosed with bipolar affective disorder on lithium carbonate therapy for 25 years presented to our accident and emergency center in a state of altered sensorium of one day duration. There was no history of intake of other drugs or alcohol.

On examination, her vital signs were stable. Neurologically, the patient was comatose, her pupils were small and reactive, and her extraocular movements were restricted, with generalized hypotonia, hyporeflexia, and no other focal deficits. A clinical possibility of toxic/metabolic encephalopathy was considered.

Laboratory examinations revealed leukocytosis (13,450 cells/mm^3), normal blood sugar, hyponatremia (128), and elevated serum creatinine (2.3 mg/dL). Cranial computerized tomography, cerebrospinal fluid examination, and thyroid profile were unremarkable. Blood lithium level at admission was 4.7 mEq/L (normal: 0.8–1.2). Considering lithium toxicity, we initiated hemodialysis. However, in spite of 3 cycles of dialysis, decreasing lithium levels (1.2), and other parameters being normal, the patient remained unconscious. Considering the clinical possibility of nonconvulsive status epilepticus (NCSE), we obtained an EEG. The EEG demonstrated bihemispheric slowing (4- to 5-Hz theta range) with bilateral periodic triphasic waves of 1- to 2-Hz frequency, as shown in Figs. 1 and 2, similar to the EEG changes seen in Creutzfeldt–Jakob disease.

She was treated with i.v. lorazepam. She showed gradual improvement in her sensorium that correlated with the decline in blood lithium levels and the return of a normal background alpha rhythm on EEG

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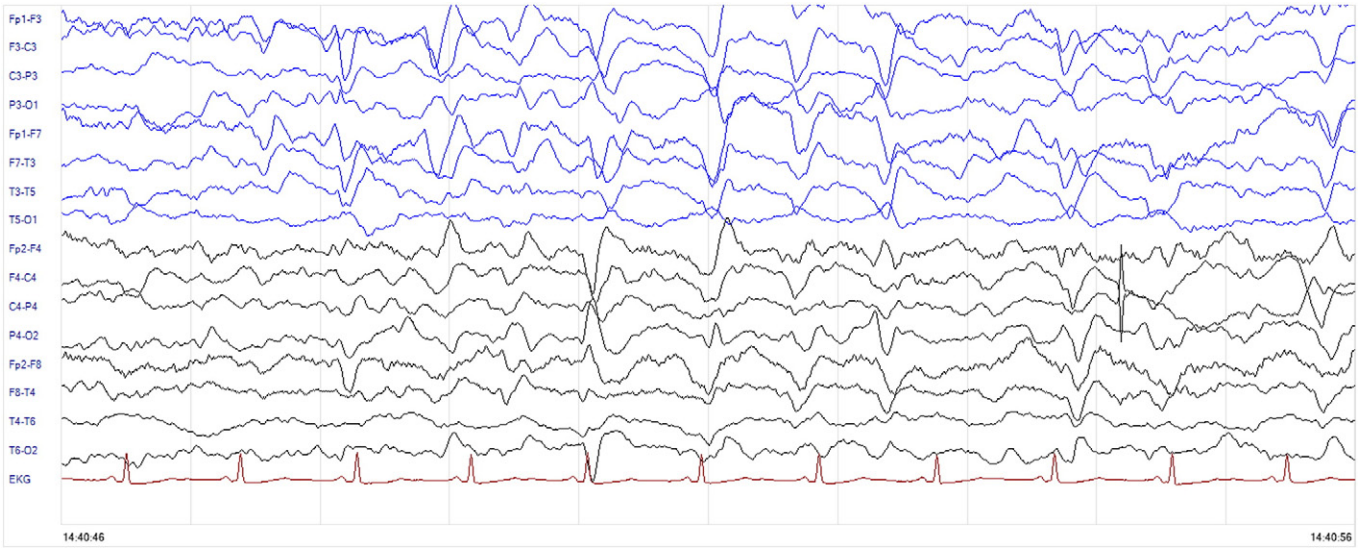

Fig. 1. EEG showing bihemispheric slowing (4- to 5-Hz theta range) with bilateral periodic triphasic waves of 1- to 2-Hz frequency.

(Fig. 3). At the time of discharge, she had recovered completely, with no apparent neurological deficit or cognitive impairment.

3. Discussion

Lithium is a commonly prescribed drug in bipolar affective disorders. Its therapeutic range is very narrow, which requires regular monitoring to prevent toxicity. It has been estimated that 75%–90% of patients maintained on lithium therapy are found to have signs and symptoms of toxicity at some point. Patients on long-term therapy are at greater risk of lithium toxicity, and among the various manifestations, severe neurotoxicity represents the most significant manifestation [1,2].

The most common signs of lithium neurotoxicity are generalized hypertonia and hyperreflexia, tremor, muscle fasciculations, myoclonus, ataxia, and dysarthria. Our patient had hypotonia and hyporeflexia which are not usual findings. A similar clinical presentation of lithium toxicity with general hypotonia, hyporeflexia, and convulsions was previously reported by Mouldi, Le Rhun, Gautier, Devemy, Destee and Defebvre [5].

Creutzfeldt–Jakob-like EEG changes associated with lithium toxicity were first described in two cases by Smith and Kocen, and since then, more cases have been reported in the literature [3].

Apart from causing a typical diffuse slowing on EEG, lithium neurotoxicity can also present as NCSE. Nonconvulsive status epilepticus is not an uncommon condition, especially in those who are critically ill. It carries a high morbidity and mortality and is often unrecognized as the cause of coma. The disease is confirmed when there are epileptic discharges on EEG and concomitant behavioral or cognitive changes lasting more than 30 min. A clinical and electroencephalographic response to intravenous benzodiazepine will also help in establishing the diagnosis. Nevertheless, recognition of NCSE is often delayed because its clinical manifestations mimic psychiatric disturbances, metabolic encephalopathies, and postictal confusional states.

Hemodialysis has been the mainstay of therapy for lithium overdose. It is generally considered when serum lithium levels are higher than 2.5 Eq/L. A forced alkaline diuresis can also be used in the presence of unimpaired renal function when hemodialysis expertise or facilities are not available [4].

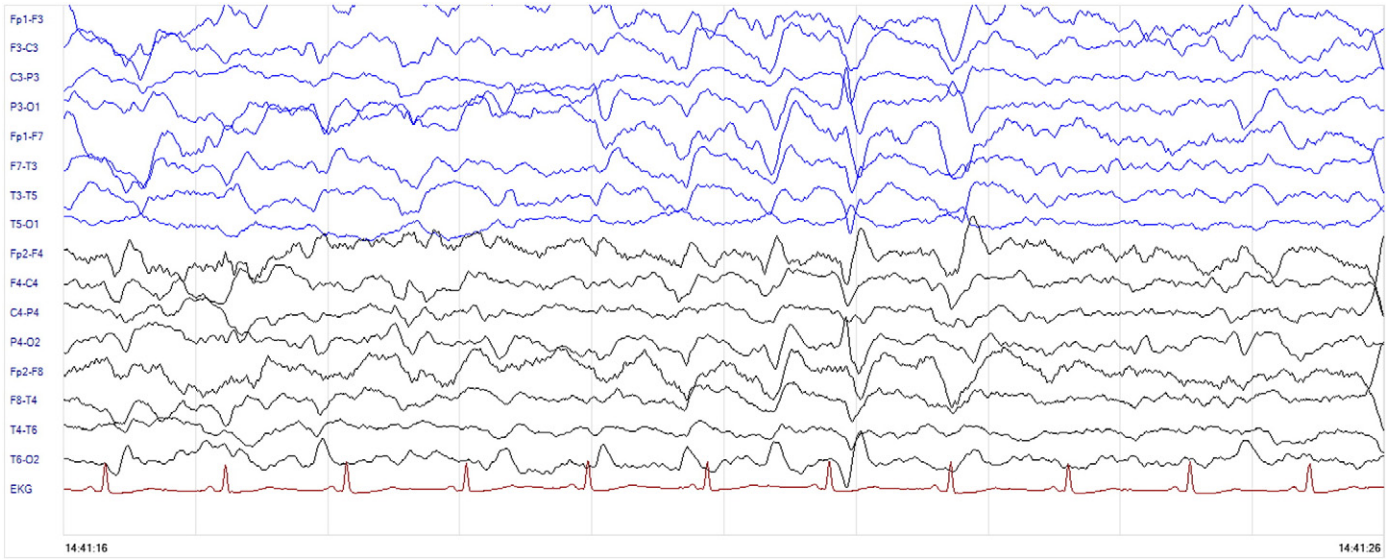

Fig. 2. Bihemispheric slowing (4- to 5-Hz theta range) with bilateral periodic triphasic waves of 1- to 2-Hz frequency similar to EEG Changes seen in Creutzfeldt–Jakob disease.

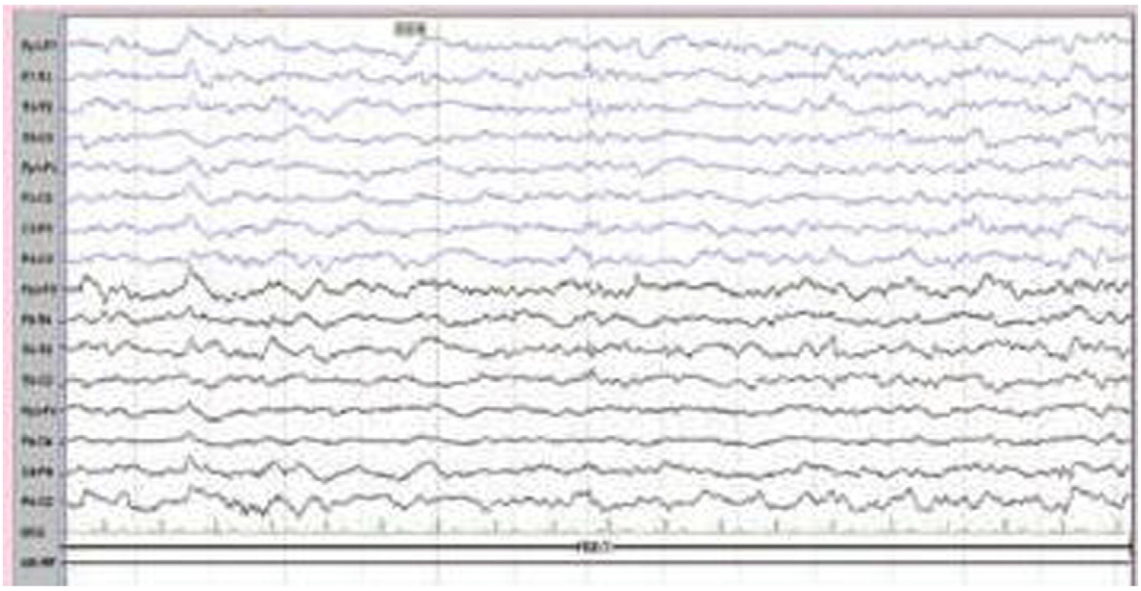

Fig. 3. Normal background alpha rhythm on the EEG.

4. Conclusion

This case suggests the importance of therapeutic drug-level monitoring of lithium, especially where toxicity is suspected, and the important role electroencephalography plays in diagnosing NCSE and its management.

Conflict of interest

No conflict of interest.

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