

Lithium toxicity and sepsis: Time for a rethink?

Sir,

We read the article by Meneguetti *et al.*^[1] with great interest. In spite of discovery of newer antipsychotic drugs still, lithium has not lost his glory and commonly used drug in the treatment of manic depressive and bipolar disorders. This report was reviewed from the point of underlying disease status, management of arrhythmias and contributory factors for lithium intoxication. Furthermore, in the ensuing paragraphs the importance of newer methods of renal replacement therapy in the removal of lithium has been highlighted.

This 61-year-old male known case of bipolar disorder with diabetes mellitus type 2, hypothyroidism and hypertension has developed progressive deterioration of conscious level of 5 days duration, and probably might have received his

regular medication including Lithium carbonate (600 mg/day). On admission he had features suggestive sepsis (rales bibasilar, tachypnoea, and elevated absolute neutrophils count of 7790/mm³, hypotension, elevated creatinine and encephalopathy). All these indicate that the patient might have suffered from an infection which has led to sepsis, toxic tubular necrosis, and toxic encephalopathy. Furthermore, authors too mentioned the contribution of sepsis for elevated troponin in their case. Encephalopathy portends a poor prognosis. Finally, he developed hemorrhagic complications possibly disseminated intravascular coagulation secondary to sepsis.

The cardiac toxicity due to lithium is ascribed to various mechanisms and most precisely competition with sodium, potassium, calcium, and magnesium ions, each of which plays an important role in cellular membrane physiology. These disturbances seem to induce a variety of electrophysiological changes.^[2] In addition, there is one reported case of ventricular tachycardia during lithium intoxication which is refractory to conventional therapy, but treated effectively with IV magnesium sulfate.^[3]

The contributory factors for elevated lithium were, continued administration of lithium while the patient has developed sepsis and renal dysfunction, coupled with administration of angiotensin-converting-enzyme inhibitors (enalapril) and hypotension. Furthermore, patient was given atenolol, which decreases the glomerular blood flow. In other words, the presumed accidental ingestion of overdose of lithium is less likely, but it could be cumulative lithium toxicity in a person with compromised renal function. Details of antimicrobial therapy were not mentioned despite his chest findings, elevated neutrophils, and features of sepsis from the beginning.

Furthermore, this patient was subjected to conventional hemodialysis and the serum level was brought down to 0.6 mmol/L from 2.9 mmol/L (reference value 0.6-1.2 mmol/L). Interestingly, intracellular lithium diffuses slowly from cellular compartment with increased rebound phenomenon after conventional short lasting hemodialysis, may explain recurrent cardiac arrhythmia in this case. It appears that repeated or continuous hemodialysis is required in order to prevent the 'rebound' of lithium concentrations which needs more logistic support. To overcome this snag, a new technique sustained low-efficiency dialysis (SLED) can be employed. It combines the advantages of both intermittent and continuous modalities, which prevents the rebound phenomenon. Fiaccadori *et al.*^[4] had successfully treated patient with SLED and showed initial rapid clearance of lithium with resolution of symptoms, followed by a sustained clearance minimizing the rebound. The optimum

role for SLED in the management of toxicities merits further study.

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