Lithium A Gold Standard Mood Stabilizer - Renal Failure A Hindrance in Use

Keywords: Lithium, Alkali metal, Mood stabilization, Renal function

ABSTRACT

Introduction: Lithium, lightest of all Alkali metals used in Medical field since 19th century. In Psychiatry, its use started from 1949 for mood stabilization in people having mood disorders. There are reports of side effects with reference to renal function impairment occurring sporadically. The current study is to document the side effects of Lithium at our center (Mc Gann District and Teaching Hospital, Shivamogga, Karnataka, India.) and compare with other centers.

Objectives: To compare the side effects of Lithium, particularly of renal function with other centers.

Methods: The published articles of Lithium induced toxicity at different centers are collected and compared with our data.

Results: There were no differences in the renal side effect of lithium in our data and the side effect published at other centers.

Conclusion: Most of the side effects of Lithium are reversible. The Lithium levels are to be monitored constantly if used for long periods of time. Further multicentric data collection and comparison need to be continued.

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INTRODUCTION

Lithium, first used in the treatment of Gout in 1940’s\(^1\). It was used in the treatment of hypertension and withdrawn because of side effects. It has a narrow therapeutic index. In 1949 Cade\(^4\) found its use in controlling the fluctuations of mood in patients suffering from mood disorders. Mood disorders involves presence of depression or mania which are the two ends of mood spectrum. Around 50-65% of them have either one or the other end of the spectrum in their life time. To avoid this, mood stabilizers like lithium is used. Lithium has a good membrane stabilization of neurons by modulating energy metabolism. It provides neuroprotection and neuroplasticity\(^2\). There are different schools of thought to use mood stabilizers along with antidepressants or antipsychotics in the first episode or further episodes. The first incidence of lithium toxicity was reported three decades ago\(^3\). Renal diseases in the form of tubular acidosis, chronic tubulointerstitial nephritis, glomerular nephritis and nephrotic syndrome are seen with chronic use of lithium\(^4\). Histopathologically the presence of tubular cysts is highly characteristic of lithium toxicity\(^5\). It results in decrease in urine concentrating ability\(^6\). Lithium modulates phosphoinositol pathway in the pathogenesis of minimal change glomerulonephritis disease.\(^7\) Neuropsychiatric symptoms like irregular coarse tremors, fascicular twitchings, rigid motor agitation, muscular weakness, ataxia, sluggishness, delirium are the features of lithium toxicity. Nausea, vomiting, diarrhea, sinus bradycardia and hypotension are other side effects\(^9\). Severe lithium intoxication results in seizures, stupor, coma and 10% risk of permanent neurological damage. Important renal manifestations include puffiness of face, polyuria and nephropathy\(^10\). Lithium induced reversible polyuria and impairment of urine concentrating ability is due to inhibition of ADH in distal nephron\(^11\). Therapeutic blood level of lithium has to be maintained in range of 0.6-1.2 mEq/l. Lithium toxicity does not cause progressive renal insufficiency\(^12\). It also induces hypothyroidism on chronic therapy. A regular monitoring of serum lithium and serum Creatinine levels is to be done once in every six months to one year\(^13\). At our hospital a patient came with a history of treatment with Lithium 900mg/day in divided doses for 15 years, for which he developed puffiness of face, decreased urine output and laboratory investigations showing serum lithium level of 5meq/l and serum creatinine level of 3.56meq/l. Lithium was stopped and hemodialysis was done with other supportive measures, it resulted in improvement of renal function and serum creatinine level was reduced to 1.1meq/l, were as serum lithium level was reduced to 2meq/l.

MATERIAL AND METHODS
The data was collected from the available published articles and compared with our center data

INCLUSION CRITERIA
I) People suffering with Mood disorder on Lithium therapy
II) Both sex
III) All age
IV) Comorbid Medical illness not considered except renal failure

EXCLUSION CRITERIA
Patients who are in renal failure.

RESULTS
As the lithium was stopped and patient treated with haemodialysis and other supportive measures there was significant improvement in the patient condition and the serum creatinine was reduced to 1.1.meq/l were as serum lithium level was reduced to 2meq/l.

DISCUSSION
Lithium though a gold standard in mood stabilization is to be used cautiously as it has a narrow therapeutic index. toxic effects are on kidney, thyroid, nervous system. The Lithium toxicity usually wanes off with stoppage of drug. This was also seen in our patient as he responded well with stoppage of drug and initiation of treatment with hemodialysis and other supportive measures. The same is seen in other literatures from different centers. Lithium is a widely used mood stabilizer with a dose of 600-1200mg/day in the therapeutic blood levels of 0.6-1.2meq/l. The mortality rate is reported to be approximately 25% in acute over dose and 9% during chronic therapy. A constant monitoring is needed for 1) Serum Lithium, 2)T3, T4, TSH , 3) Serum Creatinine. The above tests are needed to ensure the compliance of patients. Lithium toxicity is more when serum Lithium levels are >4meq/l. Acute lithium toxicity is managed with stoppage of drug, hemodialysis, forced diuresis and other supportive measures.
CONCLUSION

Lithium, an effective mood stabilizer has a narrow therapeutic index, hence mandates constant supervision over its blood levels and side effects. The monitoring of serum lithium, renal function test, complete hemogram, thyroid profile and observing for neuropsychiatric symptoms is important in lithium therapy. Educating the patients about adverse effects of lithium therapy and further multi center data collection and comparison is needed.
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