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Use of sodium polystyrene sulfonate in an acute-on-chronic lithium poisoned patient: A case report

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ABSTRACT

A 35-year-old woman with an acute-on-chronic lithium overdose received multiple oral doses of sodium polystyrene sulfonate totaling 120 g over a 24-h period. During the 72 h after the institution of therapy, the serum lithium level decreased from 3.80 to 0.42 mEq/L. Multiple doses of sodium polystyrene sulfonate may be useful in lowering the serum lithium level in severely ill patients with acute renal failure, and can substitute hemodialysis.

1. Introduction

Lithium salts are prescribed routinely for prophylactic control of manic-depressive episodes (bipolar affective disorders) and a treatment of acute mania. Lithium pharmacokinetics is subject to considerable interindividual differences that, given a narrow therapeutic window, dictate close monitoring of serum concentrations[1].

Therapeutic concentrations are 0.6–1.2 mmol/L for prophylactic control of mania and 1.0–1.5 mmol/L for treatment of acute mania. Symptoms of toxicity may present at 1.5 mmol/L, but it can vary with the individual and the time course of exposure[2,3].

Lithium toxicity can cause death and severe morbidity, and may have renal, gastrointestinal, endocrine and predominantly neurologic manifestations[3–5]. Therefore, management of lithium intoxication includes fluid resuscitation, electrolyte and acid/base management, and methods to decrease absorption and increase lithium elimination[6].

Here, we reported a case in which sodium polystyrene sulfonate (SPS) was used in the treatment of a patient with a severe acute-on-chronic lithium poisoning.

2. Case presentation

The medications of a 35-year-old woman with a history of manic depression included a twice-daily regimen of 500 mg lithium. She was referred to the emergency room because of agitation, confusion and hypovolemic shock due to a diarrhea.

The patient's vital signs were as follows on arrival: blood pressure 77/60 mmHg, pulse rate 160 beats/min and regular, respiratory rate 30 breaths/min, rectal temperature 38.4 °C, room air oxygen saturation 99%, finger stick glucose level 132 mg/dL and her level of consciousness was 10 (eyes 4; voice 2; motor 4) on the Glasgow coma score.

The laboratory data revealed hemoglobin of 10.6 g/dL, hematocrit of 21%, platelet count of 223 000, sodium of 133 mEq/L, potassium of 3.4 mEq/L, chloride of 105 mEq/L, glucose of 120 mg/dL, blood urea nitrogen of 306.3 mg/dL, creatinine of 4.52 mg/dL and serum lithium of 3.82 mEq/L. In addition, her liver function was normal.

An arterial blood gas analysis showed perturbed findings as follows: pH, 7.66; arterial partial pressure of carbon dioxide, 27 mmHg; arterial partial pressure of oxygen, 87.8 mmHg; base excess, 8.1 mmol/L.

Her electrocardiogram was normal. Besides, a CT scan of the brain was performed and showed a chronic hydrocephaly (Figure 1).

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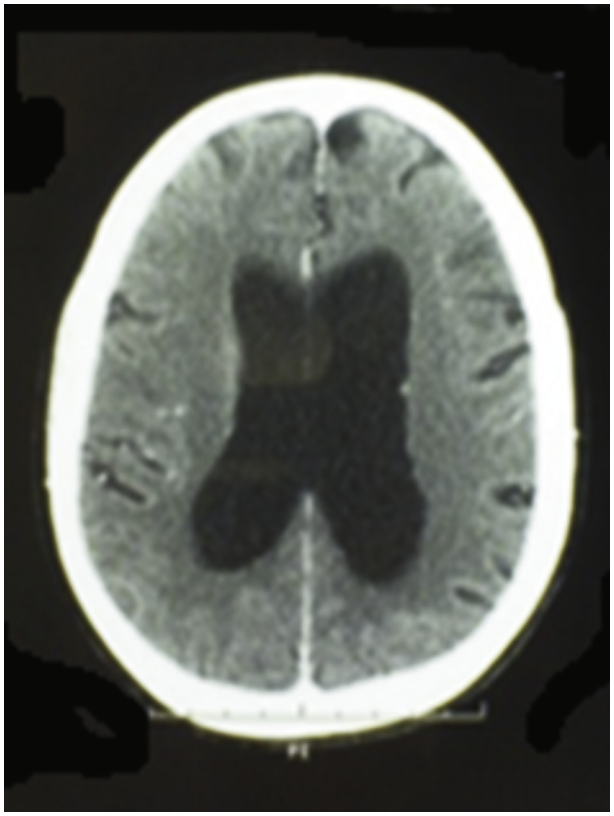

Figure 1. CT scan showing findings of chronic compensated hydrocephalus leading to normal pressure hydrocephalus.

The patient was immediately requiring invasive mechanical ventilation, fluid resuscitation and norepinephrine infusion.

Empirically, intravenous ciprofloxacin and amoxicillin/clavulanic acid were initiated. The complementary tests performed to find the focus of infection (urine culture, tracheal aspirate) were negative.

An oral administration of 30 g SPS every 6 h was prescribed, for a total of nine doses and the patient received 3 000 mL of 5% dextrose in water with 0.45% saline solution (300 mEq sodium) and 52 mEq of supplemental potassium intravenous perfusion during the first 48 h of her hospital stay.

The mean values of repeat serum sodium and potassium levels at the end of SPS therapy were 140.00 mEq/L and 3.63 mEq/L, respectively.

Serial serum lithium determinations were carried out and the level was noted to return to the therapeutic range at 72 h after SPS administration (Figure 2) and the renal function returned to the normal (Figures 3 and 4). Unfortunately, the patient was complicated by multiple organ failure syndrome and died eleven days after disease onset.

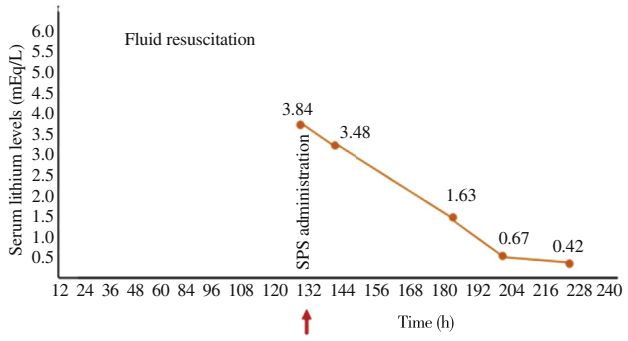

Figure 2. Serum lithium levels during SPS administration.

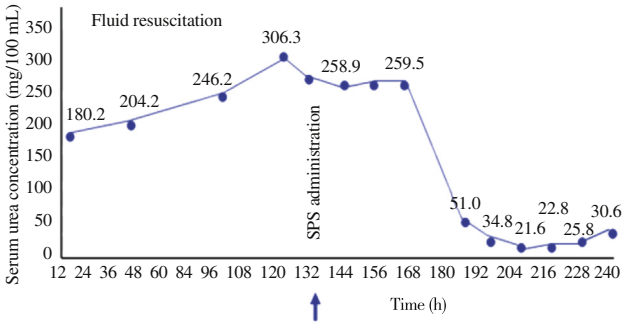

Figure 3. Serum urea concentration evolution.

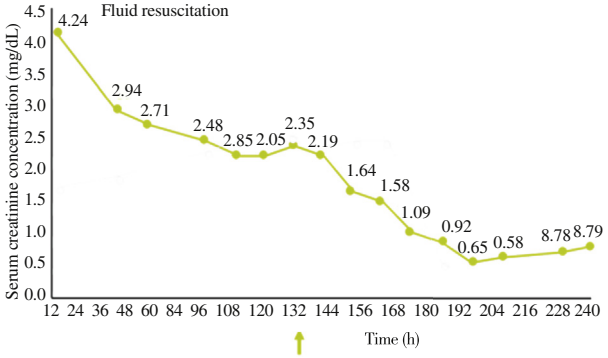

Figure 4. Serum creatinine concentration evolution.

3. Discussion

Lithium carbonate has been used as an invaluable drug in the treatment of manic-depressive illness. Its main disadvantage is a narrow therapeutic window: therapeutic concentrations of 0.6–1.2 mmol/L for prophylactic control of mania and 1.0–1.5 mmol/L for treatment of acute mania. In fact, lithium toxicity can cause death and severe morbidity, and may have renal, gastrointestinal, endocrine and predominantly neurologic manifestations[4,5,7]. It is known that lithium competes with sodium and potassium at the renal tubular level, therefore, sodium and water balance cause not only the serum level of lithium but also its toxicity. The most important in lithium toxicity is dehydration, which will produce sodium and water imbalance[6,7]. Concerning lithium toxicity management, the hemodialysis is indicated if the lithium concentration in serum is: (a) > 4.0 mmol/L, regardless of patients' presentation; (b) > 2.5 mmol/L in markedly symptomatic patients within 36 h[7,8]. Theoretically, hemodialysis is the most effective when it is instituted early, before equilibration with extravascular and intracellular compartments and thereby the lithium removing is more rapidly. Early extensive hemodialysis may also reduce toxicity by minimizing tissue concentrations and by limiting the postdialysis rebound inherent with re-equilibration[8–10].

Our patient was presented with a severe degree of intoxication, based on the amount of drug ingested, the initial serum lithium level, the impairment of renal function, the severity of neurologic symptoms, and systemic manifestations. She developed acute renal failure probably as result of volume depletion since it was rapidly reversible by infusion therapy and SPS administration without the necessity of hemodialysis.

As evident from our case, saline diuresis was found to be an ineffective method for lithium elimination[11]. An effective oral

treatment adjunct is the administration of sodium polystyrene sulfate, which can prevent lithium absorption when it is used early after ingestion[12,13]. In fact, SPS has been shown in an animal model to increase lithium elimination even when lithium was given parenterally and recently[14]. A retrospective cohort study suggested that SPS was capable of promoting lithium elimination in chronic intoxications[15].

We suggest that SPS may be useful in treating a subset of clinically sever patients with a toxic serum lithium level. It can be also considered as an alternative to hemodialysis in seriously lithium-intoxicated individuals. Further evaluation of SPS in a randomized, blinded clinical trial(s) will be required.

Conflict of interest statement

The authors report no conflict of interest.

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