

INTRODUCTION

- Rhabdomyolysis results from breakdown of muscle fibers with release of toxic intracellular contents. Common causes of rhabdomyolysis include muscle injury, compression, infection, malignant hyperthermia, neuroleptic malignant syndrome, myopathies, hypophosphatemia, sepsis, burns, and drug-induced.

- Rhabdomyolysis may occur initially with the presentation of psychogenic-induced hyponatremia or in a delayed fashion after correction of hyponatremia. We discuss correction of hyponatremia and rhabdomyolysis that may develop during correction of hyponatremia.

- In this case report a 24 year old male with a history of schizophrenia who was found to have marked hyponatremia secondary to psychogenic polydipsia and developed a delayed onset of rhabdomyolysis during the correction of this electrolyte disorder.

CASE SUMMARY

- A 24 year old schizophrenic male was found down at his home incontinent of urine with altered sensorium. Further history revealed the patient was consuming large amounts of water for several weeks prior to admission and possibly had a seizure. He has had numerous recent hospitalizations for schizophrenia. His medications included fluoxetine, olanzapine and omeprazole.

- On physical exam his temperature was 100.9 F and pulse of 110. He was intubated for airway protection. He did not open his eyes or follow commands. The rest of his physical exam was normal.

- Initial laboratory values showed serum sodium value of 111mEq/L. Serum sodium concentration was reassessed and found to be 112 mEq/L. Hypertonic saline was initiated at 40ml/hr. The rate was decreased to 20 ml/hr when sodium rose to 118 mEq/L. Subsequent sodium was 128 mEq/L and thus the hypertonic saline was discontinued. (table 1).

- Further labs including ammonia, salicylate, acetaminophen, urine toxicology, ethanol, and liver function panel were within normal limits.

- His admission CK was 3,825 U/L and peaked at 26,667 U/L 48 hours later (fig 1). An extensive search for sources of ongoing muscle injury was unremarkable and included careful physical examination, radiographs of lower and upper extremities for occult fracture, EEG for ongoing seizure activity, and CT scan of the chest, abdomen and pelvis.

DATA/IMAGES

Androgué-Madias Formula¹

$$\text{Change in serum Na}^+ = \frac{(\text{infusate Na}^+ + \text{infusate K}^+) - \text{serum Na}^+ \text{ total}}{\text{body water} + 1}$$

Laboratory Trends

Time (h)	Sodium (mEq/L)	Chloride (mEq/L)	Potassium (mEq/L)	Serum Osmolality (mOsm/kg of water)	Urine Osmolality (mOsm/kg of water)
0	111	75	4.4		
3	112	78	4.1	243	351
6	114	84	4		
10	112	86	4.8		
15	117	85	4.8		
19	128	96	4.6	267	502
24	127	98	4.6		
33	129	100	5.3 (SH)	139	139
36				278	244
41	130	97	3.8	275	415
44	130	97	3.8	274	639
48	129	99	3.7	273	562
60	131	396	3.7	277	516
75					292
90					451

Table 1

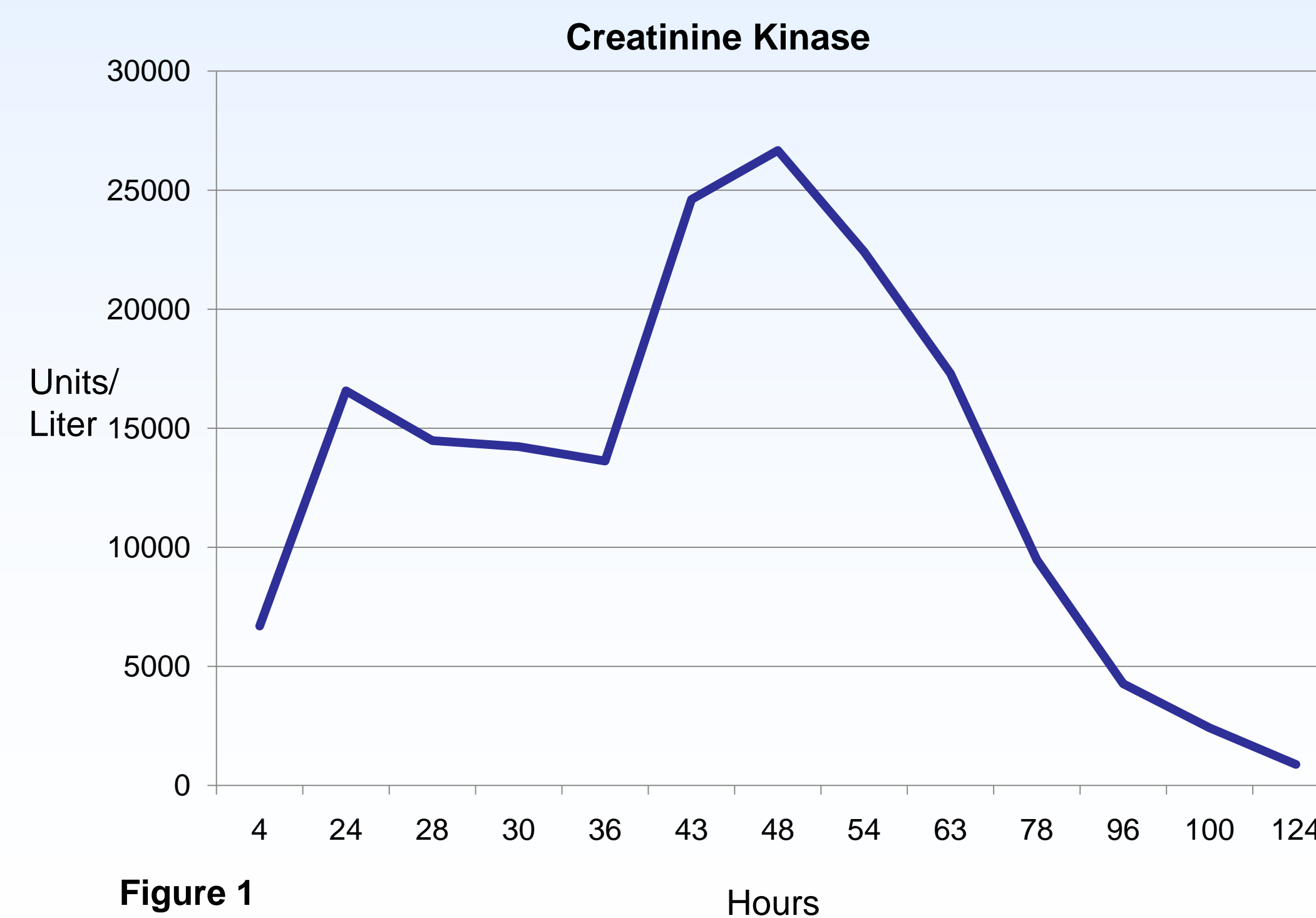


Figure 1

DISCUSSION

- Psychogenic polydipsia is seen in up to 17.5% of psychiatric patients². This behavior was noted in the psychiatric literature prior to the introduction of psychotropic agents.

- The management of these patients is challenging due to the need to correct the serum sodium slowly and the difficulty in predicting the rise in serum sodium with correction. The acute management of hyponatremia may require the use of hypertonic saline. Clinical predictors such as the Androgué-Madias formula are often used to safely guide sodium correction. Mohamed et al retrospectively reviewed use of hypertonic saline via the Androgué-Madias formula over a 5 year period and found over 20% of patients were overcorrected³. Overcorrection should be avoided as it can lead to central pontine myelinosis.

- Additionally, our patient developed rhabdomyolysis. His admission CK of 3,825 U/L may be due to his initial seizure versus his psychotropic medications. However, the extent and continued rise of CK values in the absence of an obvious muscle insult made it likely due to the correction of his hyponatremia.

- Three case reports have described rhabdomyolysis after correction of hyponatremia in the setting of psychogenic polydipsia⁴⁻⁶. All three cases describe delayed onset of rhabdomyolysis with a peak serum CK occurring 48 to 62 hours after admission. The calcium-sodium exchange mechanism of the skeletal muscle myocyte, or extracellular hypoosmolality leading to failure of cell volume regulation may provide an explanation for this process.

- The chemical gradient for sodium/calcium exchange may be altered leading to increased intracellular calcium. This leads to activation of lipases and proteases causing cell death.

- The treatment of severe symptomatic hyponatremia poses unique challenges for the clinician. Overcorrection of serum sodium may occur with hypertonic saline thus it should be used with caution. Formulas used to guide sodium correction poorly predict correction.

- Finally, rhabdomyolysis may occur initially with the presentation of psychogenic induced hyponatremia or in a delayed fashion after correction of hyponatremia. As a result, total CK should be checked initially and with correction of the hyponatremia

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