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Refractory Hyponatremia on Therapy in Syndrome of Inappropriate Antidiuretic Hormone: A Case Report

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ABSTRACT

Hyponatremia is particularly common in neurosurgical patients, occurring in up to 50% of cases depending on the underlying diagnosis. One of the causes is the syndrome of inappropriate antidiuretic hormone (SIADH). Treatment for this condition mainly increases sodium intake and fluid restriction. Both of these therapies are potentially dangerous in some cases. Thus the therapy should be considered thoroughly. In this case we present, A 33-years-old male with refractory hyponatremia day-8 post craniotomy. Serum sodium was measured after every treatment. The patient was given hypertonic saline, fluid restriction advice, and a combination of those treatments. Yet every treatment gave various outcomes. Although the theory was put into practice, therapy in SIADH is harder than expected due to variations in the patient's condition.

INTRODUCTION

Hyponatremia is particularly common in neurosurgical patients, occurring in up to 50% of cases depending on the underlying diagnosis. One of the causes is the syndrome of inappropriate antidiuretic hormone (SIADH). Treatment for this condition mainly increases sodium intake and fluid restriction. Both of these therapies are potentially dangerous in some cases. Thus the therapy should be considered thoroughly.

CASE PRESENTATION

A 33-year-old male was admitted to the hospital in stable condition after craniotomy. Day-8 post-surgery sodium level decreased to 129 mmol/L and diagnosed with SIADH. Subsequent initial correction with 3% hypertonic saline infusion was administered 400cc/12 hours for 24 hours. On the following day, the sodium level has yet to increase.

Hypertonic saline correction stopped after 72 hours

and was switched to fluid restriction 0.8-1L/24 hours. After 24 hours of the restriction, the sodium level remained low at 127 mmol/L. After 72 hours of the restriction, the sodium level decreased to 114 mmol/L and Hypertonic-saline restarted with the rate of 46cc/hours for 40 hours combined with fluid restriction and the sodium level was re-evaluated showing 122mmol/L. 24 hours before the patient was discharged from the hospital, 1.5L of normal saline was administered and the sodium level was re-evaluated showing 122mmol/L. For take-home medication, the patient was given sodium tablets and maintained fluid restriction. The patient was scheduled for regular follow-up.

Table 1. Treatment'day

Treatment/day	2/9	3/9	4/9	5/9	6/9	7/9	8/9	9/9	10/9	11/9	12/9
NaCl 3% 400cc/12hours	X	X	X								
NaCl 3% 46cc/hours								X	X		
NaCl 0,9%					X	X	X				
500cc/24hours											
NaCl 0,9%				X						X	X
1500cc/24hours											
Fluid restiction 0.8-1.2L					X	X	X	X	X		
Diet plan											
Extra sodium meal (5g)	X	X	X	X	X	X	X	X	X	X	X
Laboratory Result											
Sodium level	129	127	122	126	122	N.E	115	114	122	N.E	122

N.E : Not evaluated

DISCUSSION

Hyponatremia is defined as a serum sodium <135 meq/L. Acute hyponatremia is characterized by onset of symptoms <48h. Patients with acute hyponatremia develop neurologic symptoms resulting from cerebral edema induced by water movement into the brain. These may include seizures, impaired mental status or coma and death. Chronic hyponatremia- Hyponatremia developing over >48 h should be considered "chronic". (Sahay & Sahay, 2014) The cause of hyponatremia in neurotrauma is multifactorial, but most cases are caused by the Syndrome of Inappropriate Anti-Diuretic Hormone (SIADH) (Cooke, Turin, & Walker, n.d.; Hannon & Thompson, 2014). Syndrome of Inappropriate Anti-Diuretic Hormone (SIADH) is a condition defined by excessive release of antidiuretic hormone (ADH) from the pituitary or non-pituitary glands at vasopressin receptors. SIADH is characterized by impaired water excretion leading to hyponatremia with hypervolemia or euvolemia (Cooke et al., n.d.). On day 8 after craniotomy, the sodium level of the patient was decreased to 129mmol/L but the patient was asymptomatic. The osmolality serum was not measured due to limitations. The patient was given 400cc/12 hours rate of 3% hypertonic saline infusion for 24 hours, according to Adrogué–Madias formula at 0.5mmol/L/hour, [infusate Na^+ - serum Na^+] / [total body water + 1], hence (513-129)/(0,6x80+1) = 30cc/hour. Recommended correcting no faster than 12 mmol/L/day (0,5mmol/L/hour) to avoid osmotic demyelination syndrome (ODS) (Jha, Behera, Jairam, & Baliga, 2014). If the patient is seriously symptomatic the rate can be increased to 1-2mmol/L/hour (Oracio, Drogué, & Adias, 2000). The goal of initial therapy is to achieve a 24-hour increase in serum sodium concentration by 4 to 6 mEq/L. Hypertonic saline seems failed to increase the sodium level. To prevent fluid overload, the therapy was switched to the fluid restriction (0.8-1L/24 hours). In SIADH. Fluid restriction to less than urine output is the primary therapy in hyponatremia. The usual recommended fluid intake is less than 800 mg/day (Pillai, Unnikrishnan, & Pavithran, 2011). After 72 hours of the restriction, the sodium level decreased to 114 mmol/L and Hypertonic-saline restarted with the rate of 46cc/hours for 40 hours based on Adrogué–Madias formula with the rate of 0.8 mmol/L/hour (513-114)/(0,6x80+1) = 46cc/hour, combined with fluid restriction and the sodium level was re-evaluated and increased to 122mmol/L.

CONCLUSION

Although the theory was put into practice, therapy in SIADH is harder than expected due to variations in the patient's condition. We believe further research is needed.

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