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LETTER TO THE EDITOR

Cerebral salt wasting syndrome in traumatic brain injury following therapeutic barbiturate coma

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Dear Editor

We present a 22-year-old male with traumatic brain injury (TBI) treated with therapeutic barbiturate coma, who developed cerebral salt wasting syndrome (CSWS) within 24 h of stopping the pentobarbital infusion.

A brain computed tomography (CT) scan revealed subarachnoid haemorrhage and cerebral oedema. He was transferred to an intensive care unit (ICU), where his intracranial pressure (ICP) was found increased (ICP: 30 mmHg).

Despite the applied measures [2], ICP continued to increase and the patient was treated with pentobarbital. The patient remained for 3 days under barbiturate coma and the laboratory findings were unremarkable. After the ICP value was stabilized to 18 mmHg for 24 h, the cessation of pentobarbital infusion was decided. Within a few hours, a sudden steep rise of potassium levels (from 4 to 6.0 mEq/l) accompanied by hyponatremia (serum sodium 138 mEq/l falling to 125 mEq/l in 5 h) was observed. The biochemistry results and the values of right heart catheterization were in favour of the diagnosis of CSWS. Serum potassium concentration returned to normal within 7 h after administration of glucose-insulin infusion and calcium. Despite fluid volume correction with normal saline, the serum sodium dropped to 120 mEq/l. Hypertonic solutions were also administered in the effort to correct the hyponatraemia with a gradual response within 7 days. Finally, his general condition was improved, and he was transferred to the neurosurgery ward.

The early and correct differential diagnosis of hyponatremia in critically ill neurological patients is of major importance and includes the (CSWS) and the syndrome of inappropriate secretion of antidiuretic hormone (SIADH).

CSWS and SIADH share several diagnostic criteria (Table 1) with the two most important differences being extracellular volume and salt balance. CSWS is recognized as a distinct entity from SIADH, a transient phenomenon usually resolving within 3–4 weeks and a therapeutic goal in patients with acute craniocerebral injury.

Smith et al. [6] first proposed the role of atrial natriuretic factor (ANP) in the development of the CSWS. The biological effects of ANP include natriuresis, diuresis, vasodilation and suppression of renin and aldosterone secretion. The administration of pentobarbital induces effects on ANP synthesis gene expression and secretion. A single dose (30 mg/kg, i.p.) of pentobarbital sodium resulted in a suppression in the plasma levels of immunoreactive ANP for up to 1 week of administration and stimulates ANP gene expression and ANP synthesis of the atrium for up to 6 weeks [4]. After cessation of barbiturate

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Table 1 Differential diagnosis of CSWS and SIADH

	CSWS	SIADH
Plasma volume	Decreased	Increased
Salt balance	Negative	Variable
Signs and symptoms of dehydratation	Present	Absent
Weight	Decreased	Increased or not change
Pulmonary capillary wedge pressure	Decreased	Increased or not normal
Central venus pressure	Decreased	Increased or normal
Hematocrit	Increased	Decreased or no change
Osmolality	increased or normal	Decreased
Blood urea nitrogen	Increased	Normal
Serum protein concentration	Increased	Normal
Urine sodium concentration	Increased	Increased
Serum potassium concentration	increased or no change	Decreased or no change
Serum uric acid concentration	Normal	Decreased

coma, an increased plasma ANP concentration is observed. In this case, the ANP levels could not be documented due to lack of facilities.

The hyperadrenergic status after the cessation of barbiturates might have contributed by altering arterial pressure to natriuresis [5].

According to the literature, some cases of patients with TBI presented a biphasic course of treated hypokalaemia, followed by rebound hyperkalaemia after cessation of barbiturate coma [1, 3]. In this case, hyponatraemia, hyperkalaemia and high natriuresis occurred also after cessation of pentothal without presenting hypokalaemia during pentobarbital infusion. We consider that the release of ANP in response to cessation of barbiturate coma mediated inhibition of aldosterone, followed by natriuresis and hyperkalaemia.

The broadly accepted treatment for patients with CSW is, generally, salt supplementation and water replacement.

The prevention and/or treatment of CSWS in patients with TBI appear to be a readily available therapeutic strategy with crucial significance for the neurological outcome. This case emphasizes to development of CSWS after cessation of barbiturate coma to patient with TBI. As possible mechanisms, we consider the realease of ANP and adrenergic surge after the cessation of pentothal. More studies must be completed in order to secure more save results.

Conflicts of interest None.

References

1. Cairns CJS, Thomas B, Fletcher S, Parr MJA, Finfer SR (2002) Life-threatening hyperkalaemia following therapeutic barbiturate coma. Intensive Care Med 28(9):1357–1360
2. Guidelines for the management of severe traumatic brain injury, 3rd edn (2007) J Neurotraum 24(Suppl 1)
3. Schaefer M, Link J, Hannemann L, Rudolph KH (1995) Excessive hypokalemia and hyperkalemia following head injury. Intensive Care Med 21:235–237
4. Seul KH, Cho KW, Kim SH, Hwang YA, Park CU, Koh GY (1993) Single injection of pentobarbital induces long–lasting effects on ANP synthesis and gene expression in the rat atria. Life Sci 52 (16):1351–1359
5. Singh S, Bohn D, Carlotti APCP, Cusimano M, Rutka JT, Halperin ML (2002) Cerebral salt wasting: truths, fallacies, theories, and challenges. Crit Care Med 30:2575–2579
6. Smith HW (1957) Salt and water volume receptors: an exercise in physiologic apologetics. Am J Med 23:623–652