Расстройства баланса натрия у ребенка с тяжелой черепно-мозговой травмой

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Представлен случай синдрома церебральной потери соли у мальчика 12 лет с тяжелой черепно-мозговой травмой. С момента травмы у ребенка отмечалась рефрактерная внутричерепная гипертензия, что послужило причиной выполнения декомпрессивной трепанации черепа на 7-е сутки после травмы. На фоне инфузии гипертонических растворов хлорида натрия с 5-х суток пребывания в ОРИТ у пациента отмечалась гипернатриемия, с 11-х суток присоединилась полиурия и гиповолемия, что было расценено как проявления несахарного диабета центрального генеза. С 17-х суток после травмы была отмечена стойкая гипонатриемия, в связи с чем на 18-е сутки в терапию добавлен флудрокортизон в дозе 100 мкг/сут с повышением дозы до 150 мкг/сут, без значимого эффекта. С 30-х суток терапии флудрокортизон отменен, однако с 54-х суток он был назначен вновь в дозе 400 мкг/сут. На этом фоне полиурия постепенно уменьшилась до 4–5 л/сут, концентрация натрия в плазме оставалась в пределах референсных значений. С 66-х суток доза флудрокортизона увеличена до 600 мкг/сут. Ребенок был переведен в профильное отделение на 67-е сутки с момента травмы. С 94-х суток в условиях отделения нейрохирургии начато постепенное снижение дозы флудрокортизона с полной отменой на 122-е сутки от момента травмы. На 132-е сутки постравматического периода пациент был переведен в другой стационар для реабилитационной терапии.

Ключевые слова: черепно-мозговая травма, натрий, гипонатриемия, гипернатриемия, внутричерепная гипертензия, отек головного мозга, синдром церебральной потери соли, дети, клинический случай.

Sodium balance impairment in a child with severe traumatic brain injury

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We report a case of cerebral salt-wasting syndrome in a 12-year-old boy with severe traumatic brain injury. The child developed refractory intracranial hypertension at the time of injury, which required decompressive craniectomy on the 7th day after injury. Infusion of hypertonic sodium chloride solutions performed at the intensive care unit resulted in hypernatremia on the 5th day and polyuria and hypovolemia on the 11th day, which was regarded as manifestations of central diabetes insipidus. Persistent hyponatremia developed on the 17th day after injury; on the next day, the therapy was supplemented with Fludrocortisone at a dose of 100 μg/day, followed by an increase in the dose to 150 μg/day, which had no significant effect. Fludrocortisone was discontinued on the 30th day of therapy, but it was re-used at a dose of 400 μg/day from the 54th day. During this treatment, polyuria gradually decreased to 4 to 5 l/day, and the plasma sodium concentration remained within the reference values. The dose of Fludrocortisone was increased to 600 μg/day since the 66th day. The child was transferred to a specialized department on the 67th day after injury. At the Department of Neurosurgery, the dose of Cortineff was gradually reduced starting with the 94th day and completely discontinued on the 122nd day after injury. On day 132th of the post-traumatic period, the patient was transferred to another hospital for rehabilitation therapy.

Keywords: traumatic brain injury, sodium, hyponatremia, hypernatremia, intracranial hypertension, brain edema, cerebral salt-wasting syndrome, children, clinical case.

Background

Cerebral diseases and injuries are accompanied by water–electrolyte imbalance, with sodium haemostasis disorders being the most common among the imbalance disorders; both hypo- and hypernatraemia can directly threaten a patient's life and are associated with an increased risk of fatal outcomes [1].

Notably, hypernatraemia is often diagnosed in patients with central nervous system diseases and has long been known as a risk factor for adverse outcomes of severe central nervous system lesions. However, hyponatraemia is

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reported in individual cases, can be easily normalised and, in most cases, is not accompanied by severe neurological complications; therefore, it is not carefully considered and monitored in routine clinical practice, which has been described in this clinical case [2].

The development of hyponatraemia in patients with acute brain damage is usually associated with inadequate infusion therapy; drug side effects; inadequate secretion of antidiuretic hormone syndrome and salt-wasting syndromes, particularly, cerebral salt-wasting syndrome (CSWS) [1-4].

CSWS was first described by Peters et al. in 1950 as natriuresis and hypovolaemia in three patients with different types of brain lesions, namely, encephalitis, intracerebral haemorrhage and bulbar poliomyelitis [3]. Unfortunately, the aetiopathogenesis of CSWS has not been fully investigated. It is considered to develop due to increased natriuretic peptide levels and alterations in sympathetic nervous system, renin–angiotensin–aldosterone system and adrenomedullin levels [5]. However, no exact data are available on the frequency of its development.

In their literature review, Leonard et al. showed that the incidence of CSWS in adult patients ranges from 0.8% to 36.4%, with the largest number of cases reported in a study including patients with a score of <9 on the Glasgow coma scale [6, 7].

CSWS has been observed in both adult and paediatric patients. In adult patients, it is more likely to occur in cases of subarachnoid haemorrhages, whereas in majority of paediatric patients, it develops in those with craniocerebral injury, brain tumours at various locations, tuberculous meningoencephalitis, Wernicke's encephalopathy and seizures and after craniofacial surgeries [8-15].

CSWS is a rare condition and has mostly been reported in literature reviews, small cohort studies or individual clinical cases. The reviews mainly contain data on epidemiology, aetiopathogenesis and biochemical changes in CSWS, and its normalisation methods are scarcely described, especially in paediatric practice, because the relevant studies in paediatric patients are primarily reports of individual cases [16].

CSWS is mainly diagnosed on the basis of the presence of hyponatraemia, hypernatriuria and polyuria, followed by hypovolaemia. The clinical manifestations of the syndrome are generally noted in the first week post-injury (postoperatively), and they persist for about 2–4 weeks and regress spontaneously; however, a longer course is also possible [4].

Currently, most treatment recommendations for CSWS include symptomatic therapy in the form of fluid replacement with isotonic and/or hypertonic sodium solution (depending on hyponatraemia severity); however, cases of successful treatment with fludrocortisone drugs have been described [13, 16, 18-20]. We present the case of a 12-year-old paediatric patient who developed CSWS after a severe craniocerebral injury an in whom positive therapeutic effects of these drugs were observed.

Case description

a 12-year-old boy was admitted to the intensive care unit (ICU) of the K.A. Rauchfus Children's Municipal Hospital No. 19; the patient had sustained a motor vehicular injury from a road traffic accident and was brought by an ambulance with a diagnosis of open craniocerebral injury, basal skull and calvarial bone fractures, cerebrogenic coma and aspiration syndrome on 29th November 2015.

At the pre-hospital stage, peripheral vein catheterisation was performed; 0.5 mg of 0.1% atropine sulphate solution, 20 mg of 0.5% Relanium solution and 4 mg of Arduan were intravenously administered; tracheal intubation was performed and artificial pulmonary ventilation was started. With upper respiratory tract and tracheobronchial tree sanitation, a haemorrhagic discharge was obtained. According to the ambulance physician, the patient's consciousness level was registered as coma before the initiation of therapeutic measures.

Upon admission, the patient's condition was extremely critical. His consciousness level was depressed to deep coma due to medications; laryngeal reflex and photoreaction were absent and anisocoria (OD>OS) was present. X-ray examination and computed tomography showed multiple skull fractures and contusion–haemorrhagic foci, as well as signs of diffuse axonal injury and massive subarachnoid haemorrhage. In the cardiovascular system, distinct microcirculatory disorders and signs of hypovolaemic shock (heart rate, 130/min; BP, 103/55 mm Hg) were observed. Gas composition and blood acid–base balance analyses indicated decompensated mixed acidosis, hyperlactataemia and hyperglycaemia **(Table 1)**.

Respiratory support was provided to assist breathing; artificial pulmonary ventilation was in the volume-control ventilation mode with the following parameters: FiO_2 = 0.3; Vt = 250 mL; f = 18/min; PEEP = 4 cm and H_2O, I:E = 1:2.

CT showed an abundant haemorrhagic discharge from the tracheobronchial tree, but lung contusion was not detected on fibrobronchoscopy; therefore, the symptom was regarded as aspiration syndrome. No pathological changes were detected in the internal organs. Hypocoagulation

Таблица 1. **Arterial blood gases and acid-base balance at the time of admission to the hospital**

Parameter	Value
pH	7,24
pCO_2, mmHg.	46,6
pO_2, mmHg.	99
HCO_3, mmol/L	18,4
BE, mmol/L	–6,9
Na^+, mmol/L	136
K^+, mmol/L	2,9
Glucose, mmol/L	15,3
Lactate, mmol/L	3,7
Saturation, %	96,7

Таблица 2. **Coagulation profile at the time of admission to the hospital**

Parameter	Value
Prothrombin index, %	64
Fibrinogen, g/L	2,0
Activated partial thromboplastin time, sec	35,7
International normalised ratio	1,33

was detected in a study on haemostasis indicators **(Table 2)**.

Immediately after admission, a comprehensive examination was performed; the examination findings helped establish a diagnosis of motor vehicle injury with open craniocerebral injury; severe brain contusion; contusion foci in the right temporal lobe pole, basal fields of the frontal lobes and basal ganglia on the left; cranial vault multiple linear fractures; basal skull fracture in the anterior and middle cranial fossa; ethmoid bone fracture; right temporal bone pyramid fracture; haematosinus of the ethmoid labyrinth cells; primary injury to the brain stem; diffuse axonal damage; massive subarachnoid haemorrhage; pneumocephalus; cerebral oedema; cerebrospinal rhinorrhoea and aspiration syndrome.

Infusion therapy along with haemodynamic support (dopamine, 10 μg/kg/min), respiratory support (artificial pulmonary ventilation), sedation and analgesia (thiopental sodium, 2.0–2.5 mg/kg/h; 0.005% fentanyl solution, 3.3–2.5 μg/kg/h) was initiated. Fresh-frozen plasma was repeatedly infused to normalise hypocoagulation.

Signs of dislocation syndrome and shock phenomena were observed due to the presence of anisocoria; therefore, an intracranial pressure (ICP) sensor was installed, with 4 mmHg being the initial measurement that later increased to ⩾20 mmHg.

Despite the aggressive therapy aimed at ICP normalisation, signs of cerebral oedema persisted during the first 7 days post-injury, with ICP increasing to 40 mmHg periodically **(Fig. 1)**. Due to the ineffectiveness of the conservative therapy for intracranial hypertension, decompressive craniotomy was performed on day 7, which normalised ICP indices.

On day 11 post-injury, sedation was withdrawn, which triggered the development of a pronounced pattern of diencephalic–catabolic syndrome; therefore, antipsychotics (droperidol), benzodiazepines (Relanium) and Finlepsin were administered. Despite the withdrawal of sedative therapy, the patient remained in coma, which later progressed into a vegetative condition with tetraparesis.

Since day 5 post-injury, a tendency for the development of hypernatraemia up to 175 mmol/L was noted **(Fig. 3)**.

From day 11 to 12, polyuria (up to 4.5–5 mL/kg/h) was noted, with normal or high sodium concentration. Urea and creatinine levels remained within the normal range, with no signs of kidney damage; therefore, diabetes insipidus of central genesis was considered. The patient was occasionally administered with 25 μg of desmopressin for the management of polyuria and persistent hypernatraemia, and the therapy was found to be effective.

On day 17, polyuria with hyponatraemia (approximately 126–128 mmol/L) was observed, which required correction with a hypertonic sodium solution. Urine density remained within the physiological range or increased

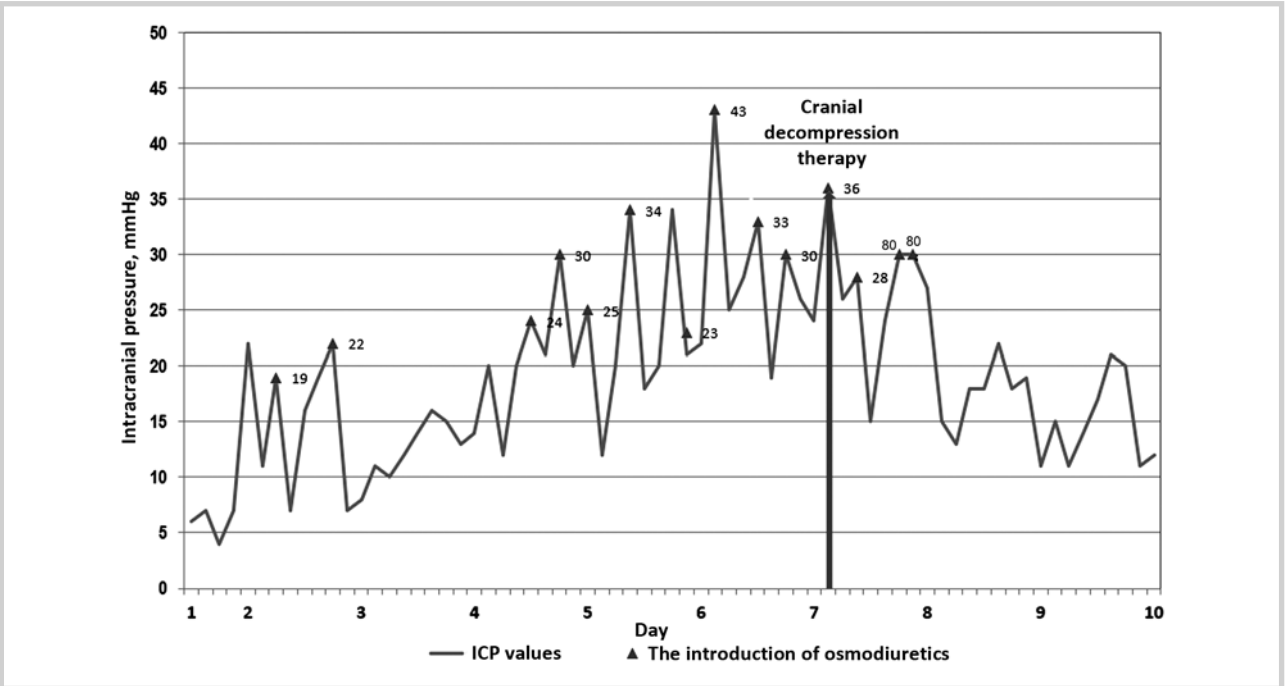

Fig. 1. **Dynamics of intracranial pressure within 10 days after injury.**

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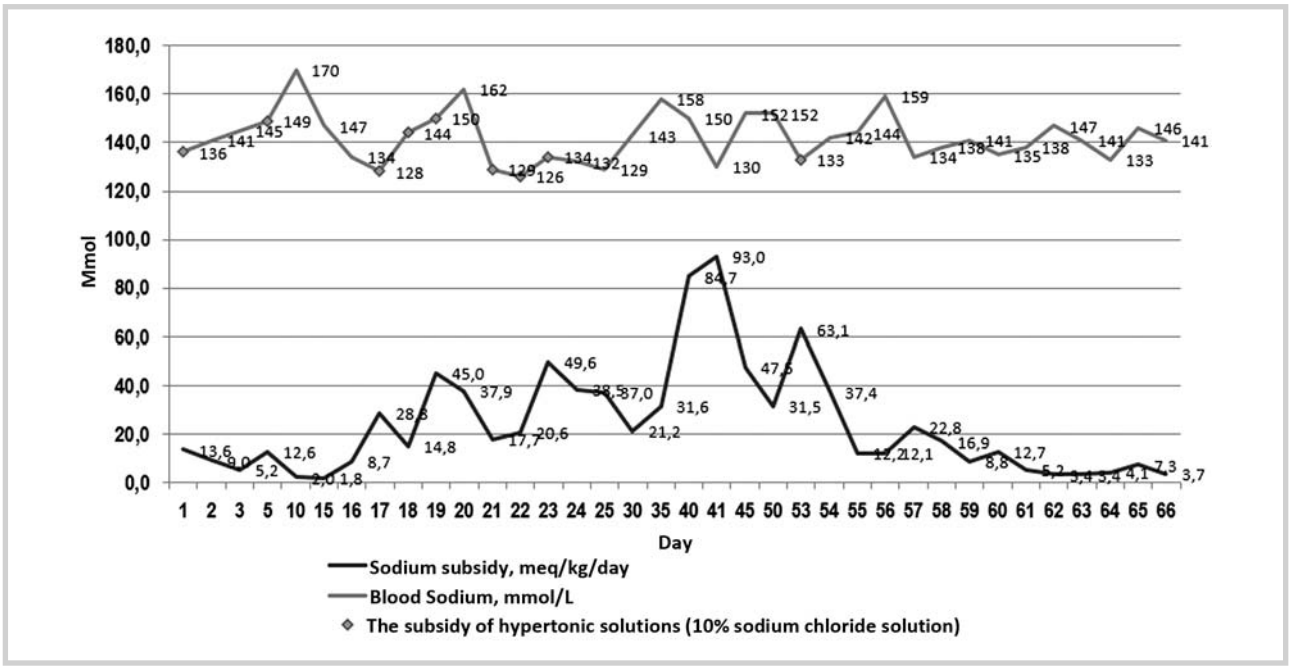

***Fig. 2*. Dynamics of sodium concentration in plasma.**

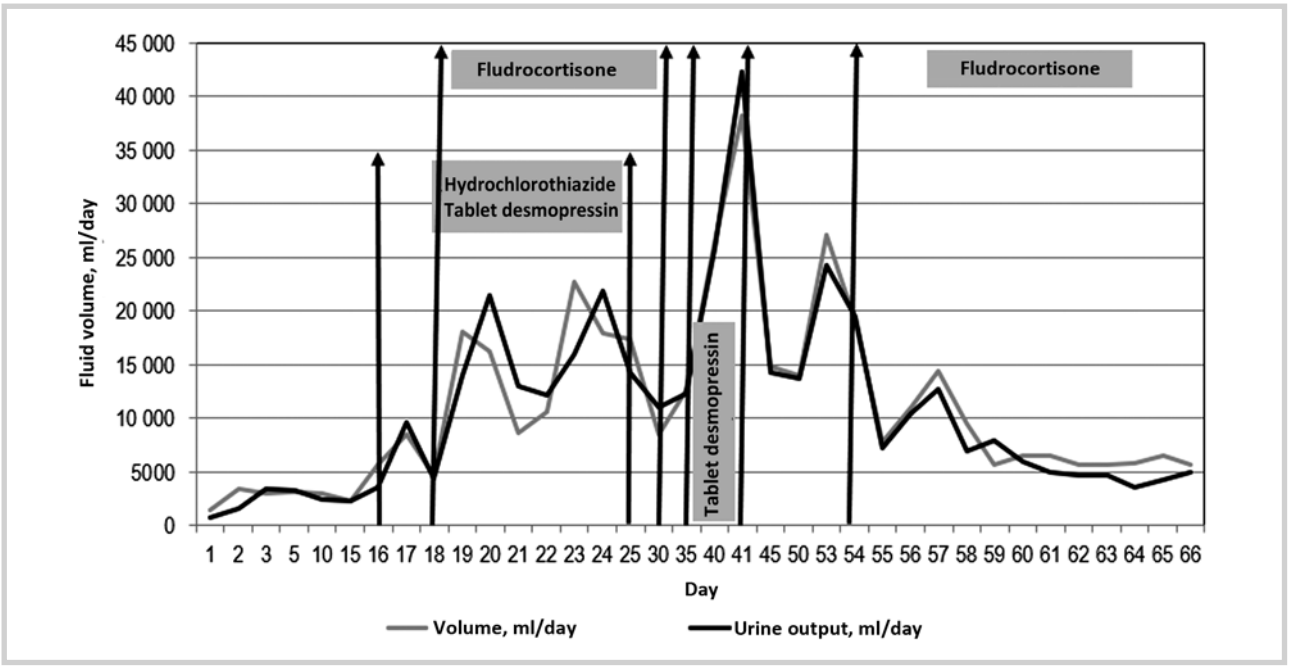

***Fig. 3*. Daily diuresis and the volume of the filled fluid during therapy with desmopressin tablets, hydrochlorothiazide and fludrocortisone.**

(from 1,010 to 1,025). Urine sodium loss on day 22 reached 127 mmol/L, which accumulated to 1,625 mmol/day with a urine output of 12.8 L/day **(Fig. 2)**.

High rates of hourly urine output (up to 30 mL/kg/h) were maintained, whereas the daily urine volume reached 22 L/day, resulting in the requirement of large volumes of infusion therapy. Attempts to limit the infusion therapy led to the rapid emergence of hypovolaemia and dehydration. Therefore, fluid was replaced with crystalloid and gelatin-based solutions. In addition, enteral rehydration was initiated (up to 3 L/day). The water balance was calculated per day, with an increased urine output rate of >5 mL/kg/h; hourly urine output was recorded and infusion therapy corrected. Periodically, hypernatraemia of up to 150 mmol/L was noted with the increased infusion of saline solutions.

On day 18 of ICU stay, in agreement with the endocrinologist, 100 μg/day of fludrocortisone (Cortineff) was administered, which was subsequently increased to 150 μg/day; however, no significant effect was observed. Therefore, *ex juvantibus* treatment with 6 mg/kg/day of hydrochlorothiazide was initiated, given its positive effect in nephrogenic diabetes insipidus. Subsequently, hydrochlorothiazide and fludrocortisone were withdrawn due to the lack of effect **(Fig. 3)**.

After the withdrawal of the therapy, pronounced polyuria persisted, and the patient excreted an average of 18–23 L of urine per day, with the maximum urine output being 42.5 L/day on day 41 (with a body weight of approximately 30–32 kg). With extremely high rates of urine output, episodes of hypokalaemia (up to 2.9 mmol/L) were noted, which were corrected by the administration of 4% potassium chloride solution at 0.5 mmol/kg/h **(Fig. 3)**.

Since day 54 of ICU stay, Cortineff at 400 μg/day was re-administered, which gradually decreased polyuria to 4–5 L/day, and the blood plasma sodium concentration remained within the reference range **(Table 3)**. On day 66, the dose of Cortineff was increased to 600 μg/day to further reduce the urine output to 2–2.5 L/day. At this stage, rehydration was predominantly enteral (up to 5 L/day). Under Cortineff treatment, sodium levels stabilised to normal values.

On day 67 post-injury, the patient was transferred to the specialised department, with re-admission to the Department of Anesthesia and reanimation on day 84 due to respiratory failure with tracheostomy cannula obstruction. CSWS persisted in the form of moderate polyuria without electrolyte disturbances along with the planned Cortineff therapy at the previous dose (600 μg/day = 20 μg/kg/day). The patient was transferred to the specialised department on day 87.

On day 94, Cortineff dose was gradually tapered, and it was completely withdrawn on day 122 in the neurosurgery department, while the urine output rate (about 2.5 mL/kg/h) and sodium concentration remained within the normal range. On day 132, the patient was transferred to another hospital for rehabilitation therapy.

Discussion

To date, there is no standardised approach to the interpretation and treatment of sodium imbalance in patients with severe craniocerebral injury. This is especially true for CSWS, which is an extremely rare condition with limited information in the literature.

This case was unique because of the extremely severe course of CSWS in a paediatric patient with craniocerebral injury, and all the widely known intensive care measures appeared to be ineffective; therefore, systemic glucocorticoids were administered to increase sodium reabsorption [17].

Despite the fact that fluid replacement with a 1:1 ratio of isotonic sodium can sufficiently normalise the sodium concentration and volume status in some cases of CSWS, this treatment remained ineffective in our patient, leading to the prescription of mineralocorticoids.

Cortineff (fludrocortisone) is a synthetic adrenal cortex hormone and is a fluorinated derivative of hydrocortisone with high mineralocorticoid activity. Fludrocortisone enhances sodium and water reabsorption in the renal tubules and increases potassium and hydrogen ion excretion. Therefore, it is the drug of choice for hormone replacement therapy in primary and secondary adrenal cortex insufficiency, such as in patients with adrenogenital syndrome. The recommended saturation dose in paediatric patients can reach 300 μg/day, with the maintenance dose being approximately 50–100 μg/day [17]. The first data on the use of fludrocortisone for the management of CSWS in adults were published in the 1980s, with recommended doses of 0.1–0.4 mg/day [18].

In paediatric practice, the use of mineralocorticoids is recommended in cases of severe forms of CSWS, when polyuria and hyponatraemia persist for a long time even with fluid and sodium loss replacement [16, 18-20].

In particular, Taplin et al. (2006) described four paediatric patients in whom fludrocortisone (0.2–0.4 mg/day) was administered, leading to the immediate normalisation of electrolyte levels. The therapy lasted for 4–125 days. However, hypokalaemia was observed in three cases and arterial hypertension in one case; thus, fludrocortisone was withdrawn [19].

Choi et al. (2012) described a clinical case of CSWS in a paediatric patient with a shunted porencephalic cyst. They showed that hyponatraemia and polyuria persisted even after large volumes of infusion therapy. After the administration of fludrocortisone at 5 μg/kg/day, no effect was noted; thus, the dose was increased to 7.5 μg/kg/day, which normalised the sodium level and rate of urine output [16].

In our patient, a starting dose of 5 μg/kg/day fludrocortisone was also used, which is equivalent to 0.15 mg of Cortineff. The lack of positive changes was mistakenly regarded as drug ineffectiveness; therefore, the therapy was interrupted. However, due to the long-term manifestations of CSWS and the extremely high rate of urine output, Cortineff was re-administered with an increased dose of 13 μg/kg/day (0.4 mg/day), and a steady decrease in the rate of urine output was achieved only at 20 μg/kg/day (0.6 mg/day).

Generally, the sodium level remained within the normal range or even increased when CSWS symptoms were observed. This was more likely due to the fact that the sodium content in solutions used for rehydration exceeded the sodium lost in the urine, which, along with extremely large volumes of infusion (>20 l/day), led to hypernatraemia.

Decreased blood plasma sodium concentration was mainly observed when positive hydrobalance was not achieved. After decreasing the rate of urine output with fludrocortisone, fluctuations in blood plasma sodium con-

centration became less pronounced. Individual episodes of hypokalaemia were seen only at the top of polyuria and were not associated with the use of fludrocortisone. Furthermore, no marked side effects of mineralocorticoid therapy were observed. The duration of CSWS in our patient (from the time of the initial manifestation of symptoms to the complete withdrawal of fludrocortisone) was 106 days.

Conclusion

This clinical case is an example of a severe and rather long (compared with the available literature data) course of CSWS in a paediatric patient with severe craniocerebral injury. Given the lack of recommendations on the use of fludrocortisone preparations, the therapy was prescribed empirically, and an individual dosing was required, which successfully eliminated the signs of CSWS.

Additional information

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