Neuroendocrinology Letters Volume 38 No. 5 2017
ISSN: 0172-780X; ISSN-L: 0172-780X; Electronic/Online ISSN: 2354-4716
Web of Knowledge / Web of Science: Neuroendocrinol Lett
Pub Med / Medline: Neuro Endocrinol Lett

CASE REPORT

N-terminal pro-B-type Natriuretic Peptide in three different mechanisms of dysnatremia onset after a child's craniopharyngioma surgery

Vera Spatenkova [1], Jan Hradil [2], Petr Suchomel [2]

1 Neurocenter, Neurointensive Care Unit, Regional Hospital, Liberec, Czech Republic
2 Neurocenter, Department of Neurosurgery, Regional Hospital, Liberec, Czech Republic

Correspondence to: Vera Spatenkova, MD., PhD.
Neurocenter, Neurointensive care unit
Husova 10, Regional Hospital, 46063 Liberec, Czech Republic.
tel/fax: +420 48 5101078; e-mail: vera.spatenkova@nemlib.cz

Submitted: 2016-11-25 Accepted: 2017-09-03 Published online: 2017-10-18

Key words: **central diabetes insipidus; CSW; polydypsia; craniopharyngioma; NT-proBNP**

Neuroendocrinol Lett 2017; **38**(5):321–324 **PMID:** 29106785 NEL380517C03

Abstract

Craniopharyngioma, due to its sellar location, can be perioperatively complicated by different types of dysnatremia. We present a rare postoperative onset of a combination of three different mechanisms of dysnatremia with N-terminal pro-B-type Natriuretic Peptide (NT-proBNP) and renal function parameters in a boy with a good outcome after craniopharyngioma surgery: 1/ Central diabetes insipidus (CDI) onset immediately after the operation, hypernatremia with peak serum sodium (SNa) 158 mmol/l) caused by free water polyuria (electrolyte-free water clearance, EWC 0.104 ml/s), NT-proBNP 350 pg/ml; 2/ cerebral salt wasting (CSW) onset on day 7, hyponatremia (SNa 128 mmol/l) with hypoosmolality (measured serum osmolality, SOsm 265 mmol/kg) caused by natriuresis (sodium – daily output 605 mmol/day, fractional excretion 0.035), NT-proBNP 191 pg/ml; 3/ Polydypsia onset on day 11 caused hyponatremia (SNa 132 mmol/l), EWC 0.015, NT-proBNP 68 pg/ml.

In conclusion, a rare combination of three different dysnatremia mechanisms (CDI, CSW, polydipsia) in a boy with a good outcome after craniopharyngioma surgery showed sodium management where NT-proBNP did not provide the anticipated values in comparison to renal function parameters.

Abbreviations:

ADH	- Antidiuretic hormone
CDI	- Central diabetes insipidus
CNa	- sodium clearance (ml/s)
CSW	- Cerebral salt wasting
DDAVP	- desmopressine acetate
dUNa	- daily output of sodium (mmol/l)
ECF	- extracellular fluid
EWC	- electrolyte-free water clearance (ml/s)
FEH_2O	- fractional excretion of water
FENa	- fractional excretion of sodium
GCS	- Glasgow Coma Scale
ICF	- intracellular fluid
NICU	- neurointensive care unit
NT-proBNP	- N-terminal pro-B-type Natriuretic Peptide (pg/ml)
SIADH	- Syndrome of inappropriate secretion of antidiuretic hormone
SNa	- serum sodium (mmol/l)
SOsm	- measured serum osmolality (mmol/kg)

INTRODUCTION

Craniopharyngioma is a benign tumor originating from the embryonic tissue of adenohypophysis (Rathke's pouch), which is typically localized in the sellar region, often involving the pituitary gland, structures of the third ventricle and optic chiasm. Surgery and radical removal of the tumor is usually the first solution, however, significant morbidities from pressure from the tumor can occur, some of which can be life threatening themselves – namely dysnatremias.

The risk of dysnatremia is associated with influencing the effective osmolality of extracellular fluid (ECF), which changes the amount of water in intracellular fluid (ICF). Hyponatremia can lead to cell edema, whereas hypernatremia causes cell dehydration.

Dysnatremias can be caused by various mechanisms which lead to a dysbalance between sodium and water in ECF. The best known hypernatremia in craniopharyngioma is central diabetes insipidus (CDI) (Pratheesh *et al.* 2013), where a lack of antidiuretic hormone (ADH) causes free water diuresis. Diagnosis can be confirmed using a specific biochemical parameter from collected urine, electrolyte-free water clearance (EWC), which evaluates the ADH-kidneys axis (Shoker 1994). Management of CDI is also quite straightforward thanks to the availability of a causal medicament, desmopressine acetate (DDAVP).

Hyponatremias, on the other hand, are most often related to two syndromes which have entirely different causes. Depletion hypoosmolal hyponatremia due to natriuresis in cerebral salt wasting (CSW) (Peters *et al.* 1950) is caused by elevated natriuretic peptides. In CSW, daily output of sodium (dUNa >260 mmol/l) as well as fractional excretion of sodium (FENa >0.012), free water (FEH_2O >0.02) and natriuretic peptides are elevated. Another syndrome is dilutional hypoosmolal hyponatremia, which is caused by free water retention in syndrome of inappropriate secretion of antidiuretic hormone (SIADH) (Schwartz *et al.* 1957), with antidiuresis and low EWC (<0.006 ml/s) (Shoker 1994; Lolin & Jackowski 1992).

Assessment of NT-proBNP is routinely used in clinical practice, especially in cardiology (Hobbs *et al.* 2005). It is a non-active part of the proBNP prohormone (108 aminoacids), which results from the detachment of the active hormone, natriuretic peptide type B (32 aminoacids). Although easy to test, this peptide has not reached the same significance in neurocritical care yet.

Dysnatremias after a child's craniopharyngioma surgery are an important issue in neurocritical care. We present a rare postoperative onset of a combination of three different mechanisms of dysnatremia (CDI, CSW, polydipsia) in a boy with a good outcome after craniopharyngioma surgery, with sodium management including NT-proBNP and renal function parameters.

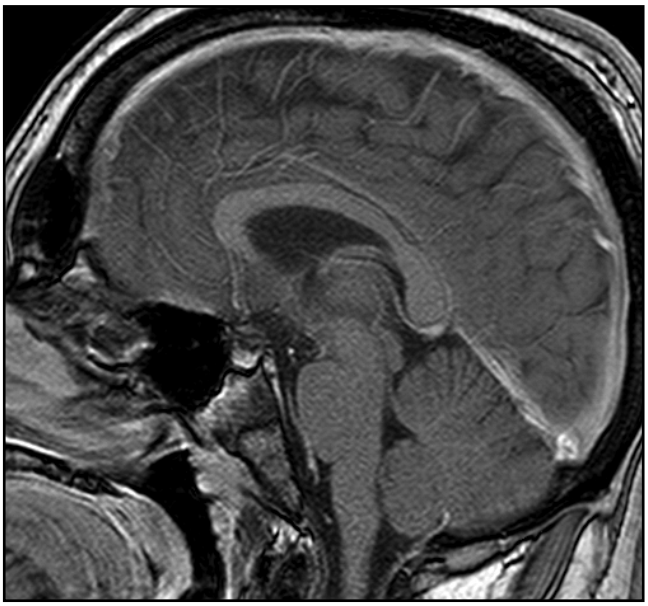

Fig. 1. Magnetic resonance (T1W FFE) after operation.

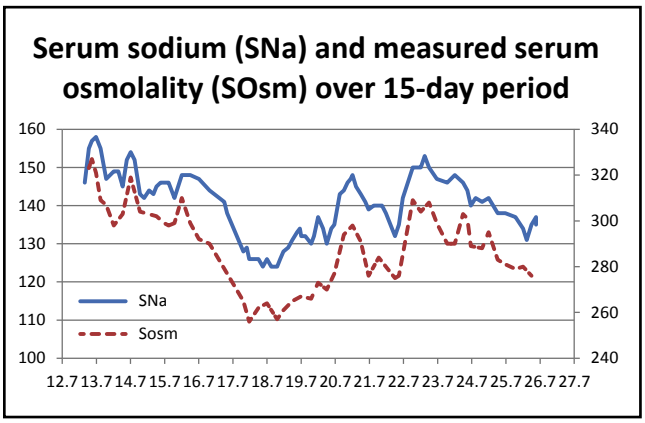

Fig. 2. Serum sodium and measured serum osmolality after operation.

CASE REPORT

An 11-year boy (weight 46 kg, height 138 cm, body mass index, BMI 24.2) without preoperative suffering of dysnatremias underwent 305-minute surgery for a craniopharyngioma in its typical localization (size 3×3 cm). The surgery was performed without any complications and led to the complete removal of the tumor (Figure 1). Following this operation the boy spent 29 days in our neurointensive care unit (NICU) due to three different dysnatremia mechanisms. He had an arterial line, which allows the return of blood as a prevention of anemia due to frequent sampling. He was conscious for his entire stay, with Glasgow Coma Scale (GCS) 15 and without any motor or sensory neurological deficit, but he had a severe problem in keeping sodium homeostasis.

His first dysnatremia occurred immediately after the surgery (Figure 2), CDI with hypernatremia peak

SNa of 158 mmol/l, free water polyuria with EWC 0.104 (reference range –0.000 ± 0.006 ml/s) and high NT-proBNP (350 pg/ml, reference range <317 pg/ml). This CDI lasted for 6 days and was meticulously balanced by the precise administration of DDAVP according diuresis and EWC.

His second dysnatremia, CSW, occurred abruptly on day 7, showing values of hyponatremia (SNa 128 mmol/l), serum hypoosmolality (measured serum osmolality SOsm 265 mmol/kg, refence range 275–295 mmol/kg), natriuresis with elevated daily output of sodium (dUNa 605 mmol/day, reference range 100 – 260 mmol/day), sodium clearance (CNa 0.055 ml/s, reference range 0.008 – 0.016 ml/s), fractional excretion of sodium (FENa 0.035, reference range 0.004 – 0.012) and NT-proBNP (191 pg/ml). We administered hypertonic saline. CSW lasted for 3 days.

On day 11, he had hyponatremia (SNa 132 mmol/l, SOsm 275 mmol/kg EWC 0.015) caused by polydypsia with fluid intake of 3070 ml/day, predominantly during the afternoon, when the boy was with his parents. His NT-proBNP was 68 pg/ml.

From day 12, CDI returned and lasted throughout the rest of his stay in NICU. The unstable levels of natremia slowly settled and the boy was discharged with permanent CDI. ADH substitution was continually administered. The boy continued school with the same marks as prior to the operation, and leads a normal life without harm to mental functions and personality patterns. This case report is presented with the approval of the family.

DISCUSSION

In craniopharyngioma, due to its sellar loacation, dysnatremias are some of the most frequent and serious complications. They can occur preoperatively, in some cases postoperatively, and can remain permanently. Several mechanisms can cause these dysnatremias, both hypo and hypernatremia. Literature describes many combinations, almost always including central diabetes insipidus, which we can see in our case report. However, we present a previously unreported combination of CDI with two different mechanisms of hyponatremia, CSW and polydipsia. Furthermore, in our case we see that this combination did not occur until post operation. CSWS and polydipsia were only present for a short time, but CDI returned during his stay in the NICU, and remained permanent following his discharge.

Dysnatremias are an issue in neurocritical care for their risk of secondary brain damage caused by shifts in the amount of water in intracellular fluid. Therefore careful attention should be paid to them. An important task in sodium diagnostic and therapeutic management is to avoid iatrogenic damage. This can occur in DDVAP overdoses and the onset of iatrogenic drug-associated SIADH. For this reason, therefore, administration of DDVAP must be balanced, as we see in our case report, where administration of DDVAP according to diuresis and EWC avoided this type of hypoosmolal hyponatremia. However, we had other non-iatrogenic hypoosmolal hyponatremias instead, CSW and polydypsia.

This case was highly complicated, the boy spent two months in intensive care, but in the end he had a good outcome and could return to school and playing his favorite sport, football. In this case, we presented our sodium management, which has an important role for a good outcome, because dysnatremias have a risk of serious secondary brain damage and pontine or extrapontine myelinolysis. In our sodium management we have a very active approach towards monitoring all the crucial serum and urine biochemical parameters. We use measured/calculated renal function parameters from 24-hour urine collection every day for proper treatment and to avoid negative iatrogenic damage. EWC is the most important of all these parameters. It enables the assessment of the ADH-kidney axis (Shoker 1992), and determines whether we are facing a compensatory response to higher fluid intake (polydypsia, overinfusions) or an abnormal response to brain damage (SIADH, CDI). In our report, EWC proved its benefit as it led to the timely diagnosis of polydipsia. On the other hand, frequent blood sampling has a risk of anemia, especially in a child, we therefore used an arterial line allowing the return of blood into the system in order to prevent anemia.

NT-proBNP is a quick routine serum biochemical parameter, which is still searching for its place in neurocritical care. It seems it does not provide such useful data as in cardiology (Hobbs *et al.* 2005). We found an elevated level in CDI, for which we have no explanation. Increased volemia does not seem to be the cause as there were decreased values during polydypsia. We did not find an elevated level in CSW.

We present this rare combination of three different mechanisms of dysnatremia (CDI, CSW, polydipsia) occurring after the craniopharyngioma surgery requiring a long period of NICU stay due balance sodium management with renal function parameters for avoiding iatrogenic damage.

CONCLUSION

In craniopharyngioma, due to its sellar location, dysnatremias are some of the most frequent and serious complications. They can be caused by various mechanisms.

In our report we demonstrate a rare case with a combination of three different mechanisms of dysnatremias onset in a boy after craniopharyngioma surgery, one hypernatremia due to CDI and two hyponatremia due to CSW and polydipsia. We present a sodium management using renal function parameters and NT-proBNP, where NT-proBNP did not provide the anticipated values in comparison to renal function parameters.

Conflicts of interest

The authors declare that they have no conflicts of interest.

REFERENCE

1 Hobbs FD, Korewicki J, Cleland JG, Eastaugh J (2005). Freemantle N. The diagnosis of heart failure in European primary care: The Improvement Programme survey of perception and practice. Eur J Heart Fail. **7**: 768–79.

2 Lolin Y, Jackowski A (1992). Hyponatraemia in neurosurgical patients: diagnosis using derived parameters of sodium and water homeostasis. Br J Neurosurg. **6**: 457–466.

3 Peters JP, Welt LG, Sims EA et al (1950). A salt-wasting syndrome associated with cerebral disease. Trans Assoc Am Physicians. **63**: 57–64.

4 Pratheesh R, Swallow DM, Rajaratnam S at al (2013). Incidence, predictors and early post-operative course of diabetes insipidus in paediatric craniopharygioma: a comparison with adults. Childs Nerv Syst. **29**: 941–949.

5 Schwartz WB, Bennett W, Curelop S et al (1957). A syndrome of renal sodium loss and hyponatremia probably resulting from inappropriate secretion of antidiuretic hormone. Am J Med. **23**: 529–542.

6 Shoker AS (1994). Application of the clearance concept to hyponatremic and hypernatremic disorders: a phenomenological analysis. Clin Chem. **40**: 1220–1227.