Transient blindness as the primary symptom of acute hyponatremia post surgery

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Abstract. Hyponatremia is a common electrolyte abnormality among children and adults. Visual disturbance associated with hyponatremia has been described in the past; however, all of these cases presented primarily with other classical signs and symptoms of hyponatremia, such as seizures, gastrointestinal upset, lethargy, or headache. We present a child who developed blindness on post-operative day #6 after resection of a brain tumour. Computed tomography of the head showed no new bleed, mass lesion, edema, or occipital lobe pathology. Laboratory evaluation revealed acute hyponatremia (115 mmol/L). No other obvious symptoms of hyponatremia were present prior to its identification. As her hyponatremia was gradually corrected, her blindness completely resolved. This is the first reported patient with acute hyponatremia to present primarily with blindness; a “precondition” of chronically increased intracranial pressure (first hit) compounded by acute hyponatremia (second hit) may have induced reversible visual loss. Checking electrolytes should be the standard of care for neurosurgical patients with acute visual disturbance or other unexplained symptoms. It is a simple measure that quickly identifies a dangerous but reversible condition.

Keywords: Sodium, hyponatremia, blindness, child, neurosurgery, vision disorders

1. Introduction

Hyponatremia is the most common electrolyte disorder seen in hospitalized patients [1,2]. The classical signs and symptoms of acute hyponatremia are predominantly neurological (lethargy, headache, seizures, and coma), gastrointestinal (nausea, vomiting, and abdominal upset) and musculoskeletal (muscle cramps and weakness) [3]. There are a few reports in the literature of transient blindness associated with hyponatremia, involving primarily adult patients [4–10] and one pediatric patient [11].

In all reported patients, the hyponatremia presented with several other classical signs and/or symptoms.

We report a child with acute hyponatremia after brain surgery (Na^+ of 115 mmol/L) who had no significant or readily identifiable symptoms other than blindness. The blindness resolved as the serum Na^+ was gradually corrected. To our knowledge, this is the first reported patient with acute hyponatremia presenting primarily with blindness. Clinicians should be aware of this unusual presentation of a common electrolyte abnormality.

2. Materials and methods

A 7-year-old previously healthy girl presented with a progressive 6 week history of nausea and vomiting,

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headache, anorexia, weight loss, and new-onset diplopia. Initial ophthalmologic assessment revealed bilateral grade 4 papilledema with symmetrically sluggish and equal pupils, though pupil size was not recorded. Her visual acuity was 6/48 in the right eye and 6/12 in the left eye. She had a right eye abduction deficit and was noted to have right homonymous hemianopia. Computed tomography and magnetic resonance imaging of her head showed massive hydrocephalus with a suprasellar mass blocking the third ventricle. She underwent partial resection of a sellar/suprasellar cystic mass ($4.0 \times 3.1 \times 2.7$ cm) that was pathologically consistent with a pilocystic astrocytoma. She was transferred to the pediatric critical care unit post-operatively, where she had an uneventful course. When discharged to the general pediatrics ward after 24 hours, she was awake, alert, and oriented, with no visual complaints and equally reactive pupils. Neurosurgical assessment on post-operative days (POD) #3 and #4 demonstrated 3 mm pupils that were equal and reactive to light. She had a mild limitation of right eye abduction and normal finger counting and reported "good vision" in both eyes. Visual fields were not examined. Electrolytes were monitored daily for the first 4 days postoperatively and were normal. The patient only received isotonic intravenous solution (0.9% sodium chloride with or without 5% dextrose). Her only regular medication was dexamethasone, and she received no other *pro re nata* medications after POD #4.

On the morning of POD #6, she awoke and complained that she was unable to see with either eye. She did not blink to confrontation. On closer examination, she was oriented but was intermittently slow to converse. An urgent computed tomography scan of her head showed no evidence of a new bleed, mass lesion, cerebral edema, or occipital lobe pathology. Re-examination by ophthalmology demonstrated ongoing bilateral grade 4 papilledema (unchanged from her preoperative examination) with no evidence of hemorrhage, retinal detachment, or optic nerve atrophy; her pupils were sluggish and asymmetric. Serum electrolytes were drawn after her loss of vision was identified, revealing a serum Na^+ of 115 mmol/L. Other pertinent laboratory findings included a serum potassium of 3.7 mmol/L, chloride of 84 mmol/L, and bicarbonate of 22 mmol/L with creatinine of 22 $\mu\text{mol/L}$ and blood urea nitrogen of 4.1 $\mu\text{mol/L}$. She was transferred to the pediatric critical care unit for further treatment and observation.

She was hemodynamically stable on admission. She had a serum osmolality of 238 mOsm/kg, a urine

osmolality was 960 mOsm/kg, and a urine Na^+ of 273 mmol/L. She received several boluses of 3 mL/kg of 3% NaCl to correct her serum Na^+ . Within 12 hours (serum Na^+ 126 mmol/L) her vision improved and she could identify objects directly in front of her, although the peripheries of her visual fields remained compromised. Concomitantly, her clinical presentation became consistent with cerebral salt wasting: her urine output was 8 mL/kg/hr, and her urine Na^+ was 237 mmol/L. She received ongoing sodium replacement with hypertonic saline (3% NaCl) and intravenous fluids based on her urinary volume and Na^+ losses. As the hyponatremia persisted despite ongoing sodium and volume replacement, fludrocortisone was administered with good response. After 24 hours of treatment, when her serum Na^+ had been corrected to 130 mmol/L, her visual field examination was completely normal with confrontational testing. Within 48 hours of admission, her urine output normalized, serum Na^+ was 137 mmol/L, and she was completely asymptomatic with normal pupillary and neurological examinations. Serum sodium values throughout her course have been summarized in Fig. 1. The patient's ophthalmologic examination remained unchanged from the day after the sodium was corrected to the time of a follow-up examination six weeks later. Her pupils were 3 mm and briskly reactive to light, extra-ocular movements were full and conjugate, visual acuity was 6/12 in both eyes, and she had a right homonymous hemianopia. Four months after the resolution of the blindness, her visual acuity had improved to 6/6 in both eyes.

3. Discussion

We describe a child recovering from brain surgery who presented with blindness as the primary symptom of acute hyponatremia (documented duration of less than 48 hours). Her vision normalized completely with electrolyte correction. The etiology of the hyponatremia was never confirmed. It may have been secondary to cerebral salt wasting, as the urine output was as high as 8 mL/kg/hr. However, the timing of the hyponatremia (POD #6) may suggest a different explanation: an isolated second phase of the triphasic response following pituitary stalk damage that may have occurred during resection of the suprasellar mass [12].

The most common manifestations of acute hyponatremia reflect brain edema and include headache, nausea, vomiting, lethargy and seizures [3]. Hyponatremia presenting with visual disturbance has been described

Fig. 1. Serum sodium levels in our patient over time, where hour 0 references the time of onset of her blindness.

in adults (usually following transurethral resection of the prostate, which has been associated with the use of glycine irrigation fluid [4–7,9,10]) and in one child [11]. However, in previously reported cases, the visual disturbance was accompanied by more typical signs and symptoms of hyponatremia such as gastrointestinal upset [4,5,7,10], significant lethargy and/or altered level of consciousness [8,9,11], headache [5,11], or seizures [6–8,11]. Although this patient's intermittent inattention may have been evidence of encephalopathy, it was subtle enough that it was not identified until the blindness alerted us to an underlying problem. This contrasts with the other child in the literature with hyponatremia-associated blindness [11], who had obvious neurologic changes (including seizures) that would have prompted a diagnosis of hyponatremia prior to the development of blindness.

The pathophysiology of hyponatremia-associated visual disturbance is not well established. While it is plausible in this case that an intra-operative complication or post-operative localized edema led to transient blindness, the late onset of blindness (POD#6), the normal computed tomography scan, and the very close temporal relationship of the course of the blindness with sodium levels make this less likely. Hyponatremia may directly induce cortical blindness through localized occipital lobe edema [9,11] caused by failure of the occipital lobes cells to restore osmotic equilibrium following an osmotic disturbance [8]. This assumes

a selective vulnerability of the occipital cortex to hyponatremia in affected patients. However, in cortically-mediated visual dysfunction, pupillary reflexes and fundoscopic examination should be normal, which was not the case for this patient. The blindness in the child presented may be attributed to a cortical process if the abnormal pupillary reflexes were due to concurrent slowing in peripheral nerve conduction velocity (i.e. in the oculomotor nerve). This phenomenon has been documented during hyponatremia and is reversible with electrolyte correction [13], though this would not explain the asymmetric nature of our patient's pupillary responses. Since acute hyponatremia is a common disorder that only rarely presents with blindness, we speculate that in our patient a "precondition" of chronically increased intracranial pressure (first hit) compounded by acute hyponatremia (second hit) induced the transient visual loss experienced. In summary, we report a neurosurgical patient with acute hyponatremia presenting primarily with blindness. The underlying mechanism is unclear; however, the strong temporal association of the visual disturbance with the hyponatremia and the complete resolution of the visual disturbance with serum sodium correction imply a causal relationship. This uncommon presentation of hyponatremia may be the result of a "second hit" to a high-risk patient with increased intracranial pressure or brain edema. Checking electrolytes should be the standard of care in a neurosurgical patient with acute visual disturbance or other unexplained symptoms.

References

- [1] Upadhyay A, Jaber BL, Madias NE. Incidence and prevalence of hyponatremia. *Am J Med* 2006;119:30–5.
- [2] Anderson RJ, Chung HM, Kluge R, Schrier RW. Hyponatremia: A prospective analysis of its epidemiology and the pathogenetic role of vasopressin. *Ann Intern Med* 1985;102:164–8.
- [3] Jospe N, Forbes G. Fluids and electrolytes-clinical aspects. *Pediatr Rev* 1996;17:395–403.
- [4] Radziwill AJ, Vuadens P, Borruat FX, Bogousslavsky J. Visual disturbances and transurethral resection of the prostate: the TURP syndrome. *Eur Neurol* 1997;38:7–9.
- [5] Ovassapian A, Joshi CW, Brunner EA. Visual disturbances: An unusual symptom of transurethral prostatic resection reaction. *Anesthesiology* 1982;57:332–4.
- [6] Agarwal R, Emmett M. The post-transurethral resection of prostate syndrome: therapeutic proposals. *Am J Kidney Dis* 1994;24:108–11.
- [7] Barletta JP, Fanous MM, Hamed LM. Temporary blindness in the TUR syndrome. *J Neuroophthalmol* 1994;14:6–8.
- [8] Hagerty C, Licho R, Recht L. Cortical blindness after correction of symptomatic hyponatremia: dynamic cerebral dysfunction visualized using serial SPECT scanning. *J Nucl Med* 1995;36:1272–4.
- [9] Henderson DJ, Middleton RG. Coma from hyponatremia following transurethral resection of prostate. *Urology* 1980;15: 267–71.
- [10] Kaiser R, Adragna MG, Weis FR Jr, Williams D. Transient blindness following transurethral resection of the prostate in an achondroplastic dwarf. *J Urol* 1985;133:685–6.
- [11] Giordano M, Seminara G, Infantone L, de Pascale E, Giordano C. A case of transient blindness in a postoperative hyponatremic child. *Clin Nephrol* 2000;53:222–5.
- [12] Uhlmann MC, Hoffman GE, Nelson PB, Robinson AG. Transient hyponatremia after damage to the neurohypophyseal tracts. *Neuroendocrinology* 1992;56:803–11.
- [13] Arányi Z, Kovács T, Szirmai I, Vastagh I. Reversible nerve conduction slowing in hyponatremia. *J Neurol* 2004;251: 1532–3.