Case Report

Hyponatremia-induced transient visual disturbances in acute spinal cord injury

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Study design: Case report and literature review.

Objective: To report an unusual case of prolonged hyponatremia in acute cervical spinal cord injury complicated by visual disturbances and to review the literature regarding the issue.

Settings: Spinal Cord Injury Unit in Göteborg, Sweden.

Methods: Retrospective analysis of clinical charts of an individual with traumatic spinal cord injury.

Results: A previously healthy 28-year-old man sustained a C7 injury in a motor vehicle accident. His injury was managed surgically and he was overhydrated during the acute management and postoperatively. When weaned off the respirator he was confused and anxious and showed a S-Na of 127 mmol/l and complained of visual disturbances. The hyponatremia was treated by extra sodium and fluid restriction but fell to a lowest value of 121 mmol/l (Day 14 post injury). Visual acuity was 0.2 on right eye and 0.06 on left eye and the eye examination revealed signs of fundus hypertonicus. CT scans and MRI revealed no signs of brain edema. The patient was further treated by mineral corticoids and fluid restriction for 4 months and his vision improved slightly, but on a final examination 125 days post injury he was found to have an afferent pupillary defect on his left eye, and bilateral atrophy of the visual nerves. The hyponatremia resolved and 6 months post injury he showed a normal S-Na.

Conclusions: SCI individuals are at increased risk of developing hyponatremia. The reported complication of visual disturbances further stresses the importance of prevention of overhydration and of timely management of hyponatremia in this group of patients.

Spinal Cord (2004) 42, 204–207. doi:10.1038/sj.sc.3101577

Keywords: spinal cord injury; acute management; hyponatremia; blindness

Hyponatremia is more frequently seen following acute spinal cord injury (SCI) than other serious conditions.^{1,2} Incidence rates of 20–35% have been reported, and the condition seems more common in complete lesions.² Hyponatremia commonly presents with nonspecific symptoms such as headache, nausea, emesis and confusion. Often these presentations are thought to be associated with trauma or postoperative state of the individual. There are a few reports in the literature of visual disturbance and transient blindness related to hyponatremia,^{3,4} but to our knowledge there are no reports of this serious complication in a SCI individual.

We are presenting a case of a previously healthy 28-year-old man who sustained a C7–T1 bilateral facet luxation as a result of a motor vehicle accident (MVA). He was first admitted to a local hospital and then

transferred to a regional hospital within a few hours after the injury. On admission, his neurological assessment revealed C7 quadriplegia, ASIA C. The rest of neurological assessment was normal with intact cranial nerves. Pupils were equal and reactive to light. His arterial blood pressure was 150/70 mmHg, heart rate 70 b.p.m. with serum sodium (S-Na) level within the normal range (140 mmol/l, Figure 1). On the following day, the patient underwent a surgical decompression with stabilization of the C7–T1 region. Preoperatively and during the surgery, the patient received 6000 ml NaCl, 1500 ml Haess 6%, 600 ml Sagman and 500 ml Cell-Saver intravenously. During operation, the patient had a blood loss of 2200 ml, a diuresis of 1150 ml, and an estimated loss of 2500 ml via perspiration. In total, he had an excess fluid intake of approximately 2500 ml. On Day 5 after the MVA, he was transferred to the Intensive Care Unit at Sahlgrenska University Hospital. The S-Na was within the normal range (135 mmol/l) with arterial blood pressure of 150/70 mmHg. The

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Figure 1 Changes in blood pressure (systolic blood pressure – diamonds, diastolic blood pressure – squares) and serum sodium concentration (triangles) from the admission to the hospital immediately after the motor vehicle accident (Day 1) and during the follow-up period (Day 104 post injury). Chronology of the major events: September 1 – Day 1 – MVA; September 2 – Day 2 – decompression of the spinal cord and stabilization of the spine; September 8 – Day 8 – first visual disturbances; September 14 – Day 14 – the lowest S-Na concentration 121

urinary production gradually increased and reached 4400 ml/day on Day 7 post injury. At that time, the patient received 1 mg Minirin[®] (desmopressin, Ferring). By the time when the patient was weaned off the respirator (Day 7 post injury) he was found to be anxious, confused and disoriented. His S-Na had declined and was 127 mmol/l. On the following day (Day 8 post injury) the patient complained that 'the room seemed to be dark' in spite of full daylight. The pupillary light reaction was normal at that time as well as during the previous period in the ICU (Days 5–7 post injury). S-Na at Day 8 was 126 mmol/l. Treatment of hyponatremia started with restriction of water intake to 1000 ml daily. He was also given NaCl tablets (0.5 g, dose: one tablet twice daily) and an infusion of 500 ml NaCl with addition of 80 mmol extra NaCl. Despite these measures, S-Na did not recover and reached the lowest level of 121 mmol/l (Day 14). The fluid intake was further restricted to 700 ml daily. At that time, the patient also complained of additional visual disturbances such as blurred vision, spots and lights.

The visual disturbances in our patient coincided with the development of fever of 39.1°C (Day 9). The blood collected for culturing at that time showed growth of *Candida albicans*. Initially, the visual disturbances were suspected to be caused by the systemic infection. However, an ophthalmologic consult was also scheduled in order to rule out other causes of visual symptoms. An ophthalmologic examination (Day 17) revealed a visual acuity on his right eye of 0.2 and the left 0.06 with a normal pupillary reaction in both eyes. The veins were enlarged, the arteries were thin and the demarcation of the papilla was unsharp at the nasal border in both eyes. These findings were interpreted as fundus hypertonicus. The patient denied any previous history of hypertension or renal disease. Moreover, the patient had been on regular health investigations before the accident and had never revealed any signs of hypertension.

Two consecutive CT scans (Days 14 and 15) and one MRI (Day 19) of the brain were taken and showed no signs of brain edema or other pathological findings. The patient was seen by an endocrinologist, who ordered measurements of tU-cortisol, s-cortisol, s-prolactin and assessment of thyroid function. The excretion of cortisol in urine was enhanced and S-prolactin was slightly above reference value whereas the thyroid function was normal (Table 1). By restriction of fluid intake, the S-Na slowly was corrected to normal values within the following 2 weeks.

The patient started his mobilization with tilt training and wheel-chair training as soon as his fever resolved. In the middle of October (approximately 6 weeks post injury), the patient tried to increase his fluid intake to 1000 ml, but then S-Na showed a tendency to decrease again to 129 mmol/l. The case was presented to Dr Gary Robertson, Professor of Medicine at the Northwestern University Medical School, Chicago. He suggested that the sustained fall in S-Na could be due to an ongoing stimulation of antidiuretic hormone (ADH) release caused by a fall in the blood pressure by more than 10–15% during tilting. Dr Robertson recommended mineral corticoids as treatment. Blood pressure investigations during tilting in our patient showed a decrease of systolic blood pressure by more than 10% and the tilt training was interrupted. Moreover, following the recommendation, Florinef[®] (fludrocortison, Bristol-Myers Squibb) 0.1 mg, half a tablet every second day was initiated at that point of rehabilitation (61 days post injury). This dose was then increased to 0.5 tablet daily after 3 weeks. The fluid intake was still restricted. The S-Na was held just at the lower margin by this regimen. At approximately 75 days post injury, the patient again tried to increase his fluid intake to 1000 ml/day, but the S-Na then tended to drop and reached its lowest value of 126 mmol/l. The restriction of fluid intake was again restored to 700 ml. In the middle of January (approx. 135 days post injury), the Florinef[®] treatment was

Table 1 Biochemical tests during the period of hyponatremia, Day 18 post injury

Biochemistry	Value	Ref. value
tU-cortisol (nmol/period)	3610	75–350
U-cortisol/creatinine	168	
tU-creatinine (mmol/period)	21.5	11.0–22.0
U-creatinine (mmol/l)	12	
U-cortisol (nmol/l)	2050	
S-prolactine (mIU/l)	348	<260
S-tyroxin (nmol/l)	110	56–147
S-free T4 (pmol/l)	20	10–22.0
S-TSH (mIU/l)	1.5	0.2–4.0
S-cortisol (nmol/l)	650	200–800
U-osmolality (mosmol/kg)	916	800–1200

changed to half a tablet every second day and the patient increased his fluid intake to 1000–1500 ml without any significant drop in S-Na concentration. The mineral corticoid treatment was ceased on day 199 post injury, after $4\frac{1}{2}$ months of treatment. In the following 3-month period, S-Na was within the normal range.

The patient was seen by the ophthalmologist on Day 40 post injury. His S-Na had been within the normal range for the previous 2 weeks. His pupils were of equal size and reactive to light bilaterally. The vision on the right eye was now 0.8 but on the left only 0.13. His fundus had recovered and showed no signs of fundus hypertonicus. He was seen three times further during the winter and the eye bottom was photographed as well. On the third investigation on day 69, a visual field examination was conducted. There was no hemianopsia, but both visual fields showed decreased vision centrally, with a low neural capacity. The left pupil seemed wider than the right, but the pupillary reaction was normal. However, approximately 2 weeks later (Day 80 post injury), a defined pupillary asymmetry was noticed with dilated left pupil. However, there was no noticeable change in pupillary reaction to light in both eyes. During the last ophthalmologic investigation (Day 125 post injury), the left pupil was noticeably larger and an afferent defect was confirmed. The investigation also showed bilateral atrophy of the visual nerves interpreted as probably due to ischemia.

Discussion

We present here a case of a previously healthy young man who developed a prolonged period of hyponatremia, as well as severe visual disturbances in the acute period of a cervical SCI. Previously, temporary blindness has been reported as an unusual presentation of postoperative hyponatremia in nontraumatic cases.^{3,4} Rapid absorption of large amount of fluid into systemic circulation followed by metabolic disturbances were indicated as possible causes of low S-Na concentration in these cases.⁴ However, the electrolyte abnormalities in individuals with SCI have a much higher occurrence rate

as compared with the general hospital population.¹ It has been shown that in individuals with SCI additional elements could contribute to the development of hyponatremia: depletion of sodium stores due to abnormal sodium loss; decreased glomerular filtration rate; and either an absolute increase in circulating ADH level or renal hypersensitivity to ADH; or a combination of these factors.^{1,5}

One of the obvious reasons for hyponatremia observed in our patient was initial overhydration in the acute postoperative phase. The fall in mean arterial pressure following cervical SCI is sometimes treated by volume expansion. It has been suggested that maintenance of an MAP within normal range may play a neuroprotective role in the acute phase.⁶ However, this can be done with other means that will not put the patient at risk of overhydration. Moreover, it was previously reported that the most significant predictor of hyponatremia in individuals with SCI is the level and the severity of the injury.¹ Our patient suffered an incomplete (ASIA C) C7-T1 tetraplegia, which put him into the high-risk group. When he began to correct the overhydration by increasing his urine production 3–4 days later, he was given a small dose of desmopressin in spite of hyponatremia. Whether this had any influence on his further decline in sodium concentration and his visual disturbances is not known. However, brain edema associated with severe hyponatremia as well as with desmopressin-treatment in hyponatremic conditions could contribute to neurological deterioration.^{7,8} Hyponatremia in our case was treated with mineral corticoids and restriction of fluid intake for a total period of 6 months. Due to restriction of fluid intake, the patient showed an increased number of episodes of bacteriuria. During the hospitalization (in total 212 days) the urine was cultured twice weekly and, from a total of 61 cultures, 32 were positive for bacteria. On six occasions bacteriuria required antibiotic therapy.

It is hard to pin down the initial site of dysfunction within the visual pathways that resulted in visual deficit in our patient. The eye investigation in the acute phase showed a fundus hypertonicus, but the patient had no previous history of hypertension. Moreover, there was only one episode of increase in blood pressure during the first day after arrival to Sahlgrenska University Hospital. However, it is unlikely that this rather modest increase would cause atrophic changes in the eye fundus. There was also the possibility that the eyes were injured during the accident. However, a normal pupillary reaction initially and a delay of visual symptoms that coincide with severe hyponatremia suggests that hyponatremia was a possible cause of the visual disturbance.

Conclusions

The individuals with SCI are at increased risk of developing hyponatremia. The presented case suggests that blindness can occur in this group of patients as a rare complication of severe hyponatremia. Clinicians should be particularly aware of susceptibility of

individuals with high cervical SCI to the risk of fluid overload and decrease in S-Na.

Acknowledgements

We are thankful to Dr E McLachlan, and Dr W Jänig, organizers of the Satellite symposium 'Autonomic dysfunction in spinal cord injury', Sydney, Australia for the possibility to start fruitful collaborations between our research teams. Ann-Katrin Karlsson is supported by grant from the Swedish Medical Society (2001-671) and Andrei Krassioukov is supported by Christopher Reeve Paralysis Foundation Grant (KB2-0003-1) and a grant from the Heart and Stroke Foundation of Ontario (NA4951).

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