Atypical Presentation of Hypothermia Induced Diabetes Insipidus: A Case Report

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BACKGROUND

Central diabetes insipidus (DI) is a well-known complication of central nervous system trauma or tumors but is a rare complication of hypothermia. Review of the literature reveals scant case reports of DI as a complication of therapeutic hypothermia after cardiopulmonary resuscitation or head injury, but to date there has been no mention of DI resulting from hypothermia alone.

CASE

A previously neurologically intact 13 year old Caucasian male with ADHD and ODD developed severe hypernatremia after developing profound hypothermia during treatment for antipsychotic induced toxic epidermal necrolysis. Despite aggressive internal and external warming, his core temperature reached a nadir of 85.1F (29.5C) while his sodium concentration simultaneously increased. He required ECMO for temperature regulation and while his hypothermia improved, his hypernatremia worsened to 164 mEq/L despite fluid resuscitation. Initial septic work up for hypothermia was negative. Interestingly, within 48 hours of starting IV aqueous vasopressin for mild hypotension he became eunatremic. However, when vasopressin was discontinued, his hypernatremia returned to 152 mEq/L with a serum osmolality of 332 mOsm/kg, an inappropriate urine osmolality of 400 mOsm/kg, and an inappropriately normal antidiuretic hormone level of 8.8 pg/ml (1.0-13.3). Efforts to discontinue vasopressin or DDAVP were unsuccessful as he would experience intermittent polyuria and hypernatremia. Pharmacologically, there were no medications that were reported to induce diabetes insipidus. He was subsequently presumed to have partial central diabetes insipidus and further pituitary workup revealed a normal Cortrosyn stimulation test and normal thyroid function tests. After he became more clinically stable, an MRI of the brain (c/s contrast) was obtained which showed an anatomically normal anterior pituitary and an appropriate location and presence of the posterior pituitary "bright spot." Thereafter, trial off DDAVP was successful and he remained eunatremic and euvolemic.

Temperature Dysregulation

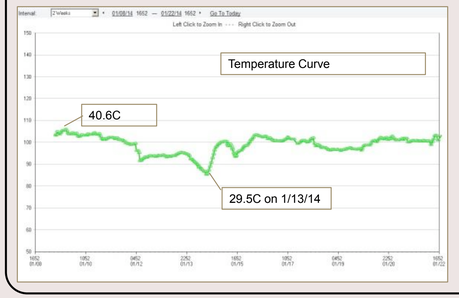


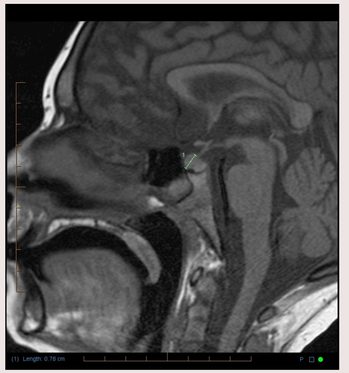
FIGURE 3: On 1/10 patient developed hyperthermia and was treated for neuroleptic malignant syndrome.

On 1/12 he then developed spontaneous hypothermia that progressed despite invasive re-warming. He reached a nadir of 29.5C before he was placed on ECMO for re-warming.

Sodium Curve

FIGURE 4: Patient's sodium was normal on admission, but on 1/13 when his temperature reached its nadir, his sodium level rapidly increased to 145, and peaked in the 150s. Even after re-warming with ECMO and fluid adjustments, his Na concentration was difficult to correct. It wasn't until he was placed on therapeutic DDAVP that his sodium corrected.

MRI



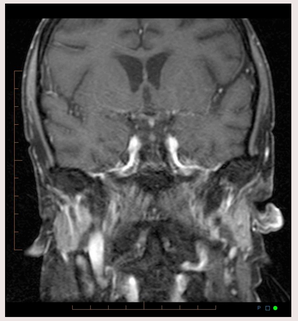


FIGURE 4. Pituitary gland is normal in size, morphology, and enhancement.

Conditions Shown to Benefit from Therapeutic Hypothermia
Stroke
Traumatic brain or spinal cord injury
Neonatal encephalopathy
Cardiac arrest
Neurogenic fever following brain trauma

Intake and Output

FIGURE 1:

Interval: 24 hour | Feb 3-Feb 9

Date		02/03/14 - 02/03/14	02/04/14 - 02/04/14	02/05/14 - 02/05/14	02/06/14 - 02/06/14	02/07/14 - 02/07/14	02/08/14 - 02/08/14	02/09/14 - 02/09/14
Time		0000 - 2359	0000 - 2359	0000 - 2359	0000 - 2359	0000 - 2359	0000 - 2359	0000 - 2359
IN	IV	4,133	4,033	2,719.6	3,124.4	1,950.2	2,194.6	2,070
	Blood	360	300		300	425		
	Other	1,050						
	NG/OG/GT/JT	2,810	2,902	2,960	3,100	2,835	2,240	1,815
	Irrigation Fluid				10			20
	Total Intake	8,353	7,235	5,679.6	6,534.4	5,210.2	4,434.6	3,905
OUT	Urine	749	864	4,385	2,973	1,779	1,557	965
	Stool	0		0	0			0
	NG/OG/GT/JT	0	90	0	0	420	820	0
	Total Output	749	954	4,385	2,973	2,199	2,377	965
Net I/O		+7,604	+6,281	+1,294.6	+3,561.4	+3,011.2	+2,057.6	+2,940
Net Since Admission (01/09/14)		+107,848.8	+114,129.8	+115,424.4	+118,985.8	+121,997	+124,054.6	+126,994.6

FIGURE 1: On 2/5/14 patient noted to have spontaneous diuresis when briefly off DDAVP. This was associated with rebound hypernatremia to 157 and inappropriately low urine osmolality of 360 given serum osmolality of 327 at that time. Vasopressin was restarted for blood pressure control on 2/7/14 with normalization of sodium by 2/10/14.

FIGURE 2: A planned trial off DDAVP was undertaken on the evening of 2/10/14 with subsequent serial evaluations of sodium, urine osmolality and serum osmolality. Due to a marked rise in sodium with this intervention, DDAVP was restarted on 2/12 and patient return to eunatremia.

Osmolality

FIGURE 2:

Date	2/6	2/10		2/11	2/11	2/12	2/12	2/12	2/12		2/13	2/13	2/13	2/14	2/15	2/16
TIME	0400	0400		1600	2100	0300	0900	1600	2100		0900	1430	1600	0900	1600	0400
OSMOLALITY, URINE (300-1000 MOSM/K)	360		DDAVP Stopped	755	391	420	400		528	DDAVP Re-Started		533			468	
OSMOLALITY (280-305 MOSM/K)	327			313	314	314	323	332	344		348	335				
SODIUM (136-144 MM/L)	157	143			150	153	152		159		159		155	147	145	139
SPECIFIC GRAVITY (1.003-1.035)	1.009				1.025		1.018				1.022			1.013	1.016	

CONCLUSIONS

1. Hypothermia, either done therapeutically or a consequence an acute medical condition/ exposure, can be potentially complicated by diabetes insipidus which can then lead to increased morbidity and mortality if not promptly recognized.
2. Clinicians should consider and be aware of this potential sequelae when performing therapeutic hypothermia and in patients who have undergone hypothermia and/or extreme temperature dysregulation from other conditions.
3. Unlike previous case reports citing hypothermia related DI, our case is not confounded by an inciting anoxic event. This unusual case encourages investigation into the role hypothermia may play in disrupting neuroendocrine functioning in pediatric patients.