Dysautonomia Complicated by DDAVP Use for Diabetes Insipidus

Abstract

This is the case of a normal, healthy 42-year old woman who sustained injuries in a spinning motor vehicle accident, leaving her with a series of diagnoses including Dysautonomia, that stumped all doctors. Because she was a Stanford-trained anesthesiologist and intensivist, she was able to self-diagnose several major illnesses, including traumatic brain injury (TBI), diabetes insipidus (DI), vertebral artery dissection (VAD) with aneurysm, oculo-vestibular dysfunction, and hypothyroidism. The DI required prescription DDAVP therapy to prevent dehydration and death. Here, we focus on the entities of the “see-saw” effect that DDAVP has on Total Body Water (TBW), hyponatremia, hypernatremia, and the complications arising from the chronic, underlying hypovolemic state of dysautonomia. Dysautonomia, an “Invisible Illness” causing dysfunction of the autonomic nervous system, is a frequently undiagnosed, hypovolemic state that can lead to syncope. The best-case scenario is to titrate the DDAVP clinically with a tendency to under rather than to over-dose, preventing hypervolemia and hyponatremia. One can also provide ambient temperature and avoid overheating, consider insensible water loss, allow the pituitary gland healing, prevent further falls or head injuries, and recognize when it is time to go off the DDAVP. There are a paucity of scientific data on DI, and its incidence is rare with coincidental dysautonomia. These entities need to be appreciated and diagnosed, especially when they occur simultaneously. The patient is in a precarious position of dying from either too much TBW (i.e., hyponatremia, brain swelling, orbital swelling) or from too little TBW (i.e., decreased glomerular filtration rate, need for dialysis, death, and the need for fluid resuscitation that can also lead to death). Let us learn in a step-by-step fashion.

“Brittle” Dysautonomia complicated by DDAVP after Post-Traumatic Diabetes Insipidus

Keywords: Dysautonomia; Traumatic brain injury; Diabetes Insipidus; Post-traumatic diabetes Insipidus; Total body water; DDAVP; Hypovolemia; Hyponatremia

Introduction

Traumatic brain injury has been associated with pituitary gland dysfunction and a ‘knock-out’ of the normal reflex arc from the hypothalamus (i.e., ‘higher’ center) to the posterior pituitary gland, such that it no longer “listens” to stimuli telling it to “stop urinating.” Hence, the net effect is that the patient presents with polyuria (i.e., production of too much urine) and polydipsia (i.e., extreme thirst and constant drinking of fluids) that can never “catch up” to the Total Body Water (TBW) losses. Electrolyte disturbances, mental fog, dehydration, renal failure, seizures, coma and death will ensue without intervention. Traditionally, this occurs in teenagers who do not recognize the symptoms as being a problem. They are simply found dead in their beds.

The state-of-the-art treatment is DDAVP prescription drug, taken both day and night, to prevent polyuria. This is one Case Report that illuminates the need for patient diagnosis, consideration of confounding variables, and forethought to treat “overtreatment,” should it occur. In probably the most common situation, the patient is given too much DDAVP, holds on to too much TBW, develops “water-balloon brain” and “water-balloon eyeballs,” may be hyponatremic down to 127 mcg/ml. On the opposite spectrum, a patient given too little DDAVP will continue to have polyuria, developing hypernatremia perhaps to the tune of 155 mcg/ml.

Either way, the brain undergoes massive microscopic and macroscopic intra- and extra-cellular water changes that greatly influence the quality of life of the patient. When all this occurs in a patient with Dysautonomia who is also taking prescribed Midodrine hydrochloride as a vasopressor to increase blood pressure, so (s) he can stand without syncope, confounding variables exist: the medication used to increase the blood pressure, salt intake, the use of Jobs® stockings, and an abdominal binder. Herein lies a comprehensive Case Report that builds layers upon layers of information, in the hopes that should the clinician encounter a patient with Dysautonomia, the treatments for traumatic brain injury (TBI) with diabetes insipidus (DI) are not worse than the syndrome, Post-Traumatic Diabetes Insipidus (PTDI) itself.
Case presentation

In retrospect, it was determined that the patient's SUV was T-boned and as she underwent a spinning torque injury of the brain and neck; her daughter in the back seat remained well. Initially, the patient presented with sun poisoning from waiting for all ambulances and fire trucks to clear the scene in Malibu, California. Then she began sleeping all day, for day after day. She saw her Primary Care Physician on Day #1 after injury, with normal head and neck x-rays. She was sent home.

She continued to sleep profusely, and then lost her concentration. For example, she went north on the 405 freeway to pick up someone at the airport, when she knew she had to go south. She stopped her husband and made him sit down to listen to her worries. He agreed to accompany her to all future doctor visits. In the interim, she became severely ataxic, was tripping on the dogs, tripping over home flooring leftovers, and almost lunged into the 11-foot emptied pool that was being remade. This is when she sought help from a neurologist.

After about two months, a head and neck MRI with contrast was negative. She continued to present to the Emergency Room for headaches, vomiting, ataxia, and confusion. During one admission, the ER doctor ordered a spiral CT scan that showed a vertebral artery dissection (VAD). She was taken by ambulance to a major medical center, where an angiogram confirmed a VAD from C3-5, Extracranial, with a 2 cm aneurysm at the base. Treatment can include stent placement [1] and the cause of death for most patients is cerebral clotted stroke, so she was placed on a platelet binder or Spanx® wear; Jobst® stockings, thigh-highs, and physical therapy sessions as well as nurse visits for her 3-year long stint with a life-saving PICC line. She remained wheel-chair bound for three years, and has been nearly non-ambulatory for almost 10 years.

Eventually, she graduated to a walker and then to a cane. When she almost tripped on the cane, she threw it away. She was recuperating over time until approximately March of 2014, when a neurologist thought that she was “malingering” and let her fall to the hard wood floor unobstructed, during Clinical Examination, while her eyes were closed. She has no proprioception, so she fell, once again suffering the 2nd PTDI. After using a walker for two more years, she began to develop more stamina, becoming a Patient Advocate and author.

Both bouts of DI presented with dry mouth, polyuria, and polydipsia despite attempts to keep fluid intake up. When it was clear to the physician: patient that “something was wrong” she presented to her Cardiologist and both times, was admitted to the hospital.

On the first PTDI diagnosis, she was not dehydrated, so she was given the Water Deprivation Test for over 4 hours, during which time she had almost 5 liters urine output. Urine output slowed to a screeching halt with a dose of IM DDAVP. So she went home with SQ DDAVP, and took it twice/day. The patient diagnosed DI resolution when she had 500 cc dark yellow urine output in the morning, with decreased frequency of urination around-the-clock. Her doctors confirmed this.

On the second bout of PTDI after the fall, the onset was insidious, perhaps 2 weeks after the event. It was a hot summer, and she had much insensible water loss. She first used the nasal spray DDAVP at night, and a po dosage by day. As time progressed, she weaned herself to small inhaled doses, the last one at bedtime. Eventually, she used inhaled DDAVP by night and po DDAVP by day. She weaned off of DDAVP by following weight gain + 2 lbs/day, volume of ‘light colored’ vs. ‘dark colored’ urine, and clinical onset of thirst. Both times, she was able to wean off DDAVP in 6 months time.

Discussion

Dysautonomia

This is a hypovolemic illness whose cause is generally unknown or hereditary, and whose symptoms range from gradual to severe onset. Other names for Dysautonomia include Familial Dysautonomia, Autonomic Dysfunction, Orthostatic Intolerance, and Riley-Day Syndrome. There is no cure for Dysautonomia, but supportive measures (i.e., medications, fluids, salt, abdominal binder, Jobst® stocking thigh-highs, Spanx®) can temper clinical symptoms for improved quality of life. Diagnosis is variable, dependent on etiology, with underlying nervous system degeneration often leading to a poor prognosis. In this case, death is often due to acute respiratory failure, aspiration pneumonia, or cardiopulmonary arrest. Helpful resource organizations include the NIH Recruitment for Dysautonomia Clinical Trials, Supportive Organizations, and free publications [2].

Lifestyle: Typically, the lifestyle scenario for a patient with Dysautonomia involves:

a. Aggressive fluid intake: this patient required a PICC line for over three years to sustain life;
b. Dietary changes: increased salt, “immunosuppression” meals and fish oil [3];
c. Compression wear: Jobst® stockings, thigh high; abdominal binder or Spanx® wear;
d. Pharmacologic treatment with vasopressors and/or steroids, and/or salt supplementation; includes flucortisone, midodrine, salt tablets, Medrol dose pack, DDAVP, and oral desmopressin;
e. Lifestyle modifications and decreased insensible TBW loss: divert the patient away from an overheated, sunny climate that would lead to either sun-poisoning (i.e., sun allergy including anaphylaxis, tongue and throat swelling) heatstroke (i.e., fever, unconsciousness, failure of central, brain-mediated temperature control so that overheating occurs), or over breathing hot air (the lungs have a surface area the size of half of a tennis court, between 80-100 m²).

Patient complaints: The average patient with dysautonomia suffers the following:

i. Dehydration: frequent trips to the ER requiring fluid boluses of 0.5% Normal Saline over 1-2;
ii. Physician incompetence: as put by many patients, “Don't tell me how to live my life when you can't even spell the name of my illness.”

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It is most likely that the economic costs of TBI will never be fully measured, given the undiagnosed TBIs, lost wages, lost pension plan contributions and Social Security adjustments, and the value of the family or paid Caregivers for the patient with TBI. The 2008 annual estimate of both acute and chronic rehabilitation in the USA was $9-10 billion. The average lifetime costs per patient may be from $600,000 - $1,875,000 [6].

Diabetes insipidus (DI) and post-traumatic DI (PTDI)

D1 is caused when a TBI causes impact to the brainstem, decreasing antidiuretic hormone (ADH), or vasopressin secretion from the supraoptic nuclei of the hypothalamus. When caused by Post-Traumatic Brain Injury, the DI is currently reclassified as "Post-Traumatic DI," or PTDI. While TBI alone is associated with a high mortality, the addition of PTDI adds additional mortality, especially when occurring very soon after the TBI.

The decreased ADH production can be either from decreased secretion (i.e., central, or neurogenic, or psychogenic DI) or decreased action (i.e., nephrogenic DI). Causes of DI that affect the hypothalamus and the posterior pituitary axis include: brain surgery, TBI, brain tumors, pituitary gland tumors, infections such as meningitis, aneurysms, and more [7]. As with this patient, the most common presentation is a poor general status, severe dehydration (BUN/Cr = 108/54), hypotonic polyuria, and hypermeteria (Na = 155 mEq/ml).

In surviving patients, the PTDI is only there for days or weeks. Only in a small number of cases is it permanent. If not treated with fluid resuscitation, then dehydration and renal failure are imminent. This can be seen by dilute polyuria, hypernatremia, hypovolemia, hypotension, low cerebral perfusion pressure, low platelet quantity (n = 30,000) and quality (patient is on Plavix®), with a glomerular filtration rate (GFR) of 30% and a platelet count of 30,000.

Severe water and electrolyte imbalances occur if a timely diagnosis is not made [8]. If (acute) hypervolemic hyponatremia occurs over 24-48 hrs, this is more dangerous than (chronic) hyponatremia that develops over days or weeks. This is an emergency medical situation that needs to be swiftly treated. If the patient’s PTDI is severe, one may simply suspend all IV drips, allowing the PTDI to “declare itself” gradually, over time, with increased urine output and increased TBW loss. Although painful to the patient, seemingly with prolonged agony, this author does not believe that 5% NS is a viable solution for the patient, who is in a hypervolemic state. The patient has the clear sensation that

**Traumatic brain injury**

The patient’s TBI was first diagnosed after memory loss, headaches, and fatigue that left her sleeping for a good portion of the day. On a finer level, she was found to have difficulty expressing specific nouns, often-times replacing them with another noun (i.e., expressive aphasia) and losing her train of thought when not allowed to complete a sentence (i.e., derailing). She also had cognitive difficulties. These are common indicators of a head injury [5].

**Incidence and prevalence:** TBI is primarily due to car accidents (50%), violent head trauma (20%), and 10% are due to sports injuries. Head violence is highest in people ages 15 - 24. In the very young, TBI can be due to Shaken Baby Syndrome, child abuse, social isolation, loneliness, anxiety, depression, social cruelty and more.

### iii. Self-education: just as with the Stanford-X Medicine (Stanford Medicine-X, 2016) program, patients learn from other patients, who then become ‘teachers’ of newly diagnosed patients; and proudly brand themselves as a “health care rebel;”

### iv. Accusations of Malingering: Just because the doctor never heard of it, the patient is treated as if (s) he is faking an illness for drug-seeking behavior, illicit pursuits or malingering because she is a “drug addict;” this can have devastating psychological sequelae on the patient, and is a frequent patient experience; hence Dr. Aranda drew up a Change.org Petition for more physician education for all “Invisible Illnesses” as well.

### v. Anxiety, Depression, Suicide: Some patients reach “the end of the rope” when no validation of their disease can be found. Usually, the patient’s family is making fun of the patient, who seeks doctor after doctor consult, only to be told, “It’s all in your head.” This is the crux of the failure of the medical systems worldwide, and it can be remedied with increased medical school and physician knowledge that includes other “Invisible Illnesses” as well.

### vi. Bed-ridden status: Since ‘gravity is the enemy,’ most patients feel comfortable and safe simply living from bed or a chair, without driving or walking into stores or family gatherings. The fear or the realistic occurrence of nausea and then vomiting is an ever-present threat, leading to social isolation, loneliness, anxiety, depression, social cruelty and more.

### Costs:** It is most likely that the economic costs of TBI will never be able to be measured, given the undiagnosed TBIs, lost wages, lost pension plan contributions and Social Security adjustments, and the value of the family or paid Caregivers for the patient with TBI. The 2008 annual estimate of both acute and chronic rehabilitation in the USA was $9-10 billion. The average lifetime costs per patient may be from $600,000 - $1,875,000 [6].

**Diabetes insipidus (DI) and post-traumatic DI (PTDI)**

PTDI is a term used to describe the syndrome of diabetes insipidus (DI) that occurs after traumatic brain injury (TBI). PTDI is characterized by the development of hypernatremia, polyuria, and polydipsia in patients with a history of TBI. PTDI is a type of DI that is caused by damage to the hypothalamus or posterior pituitary gland, leading to decreased production of antidiuretic hormone (ADH). PTDI is often seen in patients who have sustained a TBI, especially those with a history of severe head trauma. The development of PTDI after TBI is thought to be due to damage to the hypothalamus or posterior pituitary gland, which results in decreased production of ADH.

PTDI is a serious condition that requires prompt and appropriate treatment. Treatment typically involves the use of desmopressin acetate (a synthetic form of ADH), which helps to increase water reabsorption in the kidney and decrease the frequency and volume of urine. In severe cases, a catheter may be inserted into the bladder to measure urine output and monitor hydration status.

In addition to medical treatment, patients with PTDI may benefit from psychological support and rehabilitation services. These services can help patients to manage the emotional and functional challenges associated with PTDI, such as social isolation, anxiety, depression, and decreased quality of life.

Prevalence and Incidence:

PTDI is relatively rare, with an estimated prevalence of 1% to 3% in patients with TBI. The incidence of PTDI is difficult to determine due to the lack of standardized diagnostic criteria and the varying severity of TBI.

Risk Factors:

Risk factors for the development of PTDI include severe head trauma, loss of consciousness, and a history of seizures. In addition, patients with a history of alcohol or substance abuse may be at increased risk for PTDI due to the associated brain damage.

Management:

The management of PTDI involves a multidisciplinary approach that includes medical, psychological, and rehabilitative interventions. Medical management typically involves the use of desmopressin acetate, which helps to increase water reabsorption in the kidney and decrease the frequency and volume of urine.

Psychological and rehabilitative interventions may also be necessary to address the emotional and functional challenges associated with PTDI. These interventions can include counseling, therapy, and support groups.

Conclusion:

PTDI is a serious condition that requires prompt and appropriate treatment. Early recognition and intervention are critical to prevent complications and improve outcomes. Further research is needed to better understand the pathophysiology of PTDI and to develop more effective treatment strategies.
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Hypernatremia: confusion, loss of orientation, hyperreflexia, lethargy, seizures, and then coma, and death [9].

Hyponatremia: confusion, restlessness, muscle cramps or weakness, fatigue, irritability, headache, bloated eyes, loss of appetite, nausea, vomiting, and then convulsions, hallucinations, decreased consciousness, coma, brain herniation, and death [10].

Brittle dysautonomia: All of this hyper/hyponatremia and hypo/hyervolemia is exacerbated in the patient who also has an underlying diagnosis of hypovolemic dysautonomia, with small changes in water intake/output resulting in large changes in the “brittle” nature of the dysautonomia. The patient may have more syncope, palpable tachycardia, a sensation of cardiothoracic autonomic stimuli, or creased shortness of breath even at rest, and a general feeling that the dysautonomia is now “Brittle,” with more severe or frequent symptoms. Small changes in DDAVP dosage result in both large clinical symptoms that are uncomfortable, painful, or put the patient in great fear of death, and result in electrolyte variability that is not conducive to a steady and slow progress in the right direction [11].

“Which drug to take next?” becomes the endless question of each of the multiple days, weeks, and months ahead.

Pharmacology, physiology and real life

First, we will lay out all the drugs/pills before you, and then we will decide which one(s) to take at different times of the day, depending on present urine output, body weight + 2 lbs, the sensation of thirst, the last dosages of drug(s) taken, amount and color of urine, and the way that the patient’s ring on her right ring finger fits her right hand [12].

Fludrocortisone (Florinef®): is on the list of the World Health Organization’s List of Essential Medicines [13] have a molecular mass of 380.45 g/mol and a formula of C21H29FO5. The plasma t ½ is 3.5 hr, and the biological t ½ is 18 - 36 hr. It is absorbed from the GI tract with a Tmax of 1.7 hr; metabolism is in the liver [14].

The first line of therapy for dysautonomia is usually fludrocortisone acetate, tablets 0.1 mg po/day. Interestingly, many patients with dysautonomia are prescribed this drug on a long-term basis for orthostatic hypotension; hence, one side effect is hypertension. Other side effects include bruising, sweating, hives, and rash.

Many patients are placed on steroids not only for dysautonomia, but also for brain swelling after TBI, and for PTDI itself.

Midodrine hydrochloride (Orvaten® and ProAmatine®): Midodrine has a chemical formula of C12H18N2O4 and a molecular mass of 254.282 g/mol. It is a prodrug, whose active metabolite is desglymidodrine, the α1-agonist that activates α-adrenergic receptors on both arteries and veins, causing constriction and higher blood pressure. The cardiac β-adrenergic receptors do not participate in this response and diffusion across the blood barrier is poor. Midodrine lasts about 4 hr; adults may take it up to 3 times/day. The prodrug peaks at about 30 minutes; the metabolite reaches peak concentration at 1-2 hr with a half-life of about 3-4 hours. The metabolite is 93% bioavailable, and neither form binds to proteins [15].

Midodrine tablets come in scored pills, in these doses: 2.5 mg, 5.0 mg, and 10.0 mg.

With renal insufficiency, decrease dose to 2.5 mg if CrCl is < 80 ml/min; adjust from there. With liver dysfunction (causes deglycination of midodrine to desglymidodrine, and partially metabolizes both compounds): “use with caution.” Which may really say, “Don’t use.”

Common side effects: Initially, scalp burning to the point where some patients pick at scabs on their head (may last for months or 1-2 years); urinary retention or frequency, so beware if you already are incontinent; dry mouth which can contribute to stroke and cardiac disease leading to death (so watch dental hygiene); anxiety; confusion; syncope; dizziness; bradycardia (Drugs, 2016). Because desglymidodrine does not cross the blood: brain barrier, there are no central nervous system effects.

Since midodrine results in supine hypertension, it is recommended that the last dose not be taken after 1800, nor if the patient is going to take a nap. To increase patient safety, it is wise to ask this question before taking Midodrine Rx: “Is this patient going to get up and walk?” If the answer is “Yes,” then proceed. If one asks the question, “Is this patient going to take a nap?” and the answer is “Yes,” then do not give that dose of Midodrine, or you risk giving the patient a hypertensive stroke during sleep. The patient was already taking life-long midodrine vasopressor for syncope caused by Dysautonomia.

This patient was taking 15.0 mg q AM. She always took a 3-4 hr nap in the early afternoon, then awakening at 1600 and taking the second dose of 10 mg in the afternoon. She then stayed awake until 2300 or 0300.

Desmopressin acetate (DDAVP® injection): This is a man-made formulation of a pituitary hormone that comes in an injectable liquid at 4 mcg/ml, synthetic 8-arginine desmopressin. Desmopressin oral pills can come in 0.1 mg or 0.2 mg doses. Its molecular weight is 1183.34 with an empirical formula of C46H64N14O12S2.C2H4O2.3H2O and 1 mg contains 4.0 mcg desmopressin acetate, 9.0 mg sodium chloride, and hydrochloric acid to adjust the pH to 4.0 for injection. Chlorobutanol preservative 5.0 mg/ml is used per 10 ml vial. IV DDAVP has 10X the antidiuretic effect of an equal dose administered by nose. DDAVP undergoes renal excretion, and comes in IV, SQ, oral po, and oral lyophilisate forms (which melt in the mouth) [16].

Nasal DDAVP is subject to a variety of absorption factors that make the net dosage delivered of some question. The absorption is poor, nasal congestion can block absorption, nasal mucoa can atrophy, and severe atrophic rhinitis may result. For patients on concomitant blood thinners, epistaxis can also result from nasal DDAVP [17].

DDAVP injection is used for patients with mild-to-moderate von Willibrand’s disease (Type I), with Factor VIII levels > 5%. It can be administered 30 minutes prior to a surgical procedure.
DDAVP is contraindicated with a CrCl<50 ml/min, and in patients with hyponatremia or a history of hyponatremia, as rare cases of potentially-fatal water intoxication have been reported. DDAVP may lead to a transient fall in blood pressure and a compensatory increase in heart rate, aggravating heart disease. Use with caution in any patient with electrolyte disorders (i.e., cystic fibrosis, renal disorders, cardiac failure), as these patients have a propensity toward hyponatremia [18].

Table 1: History of Vasopressin in Cardiopulmonary Resuscitation.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Authors</th>
<th>Year</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Combination Vasopressin-Epinephrine for Patients with Cardiac Arrest (Out–of-Hospital)</td>
<td>Ghafoorian, et al. [23]</td>
<td>2015</td>
<td>May be useful.</td>
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ADH: Anti-Diuretic Hormone; CPR: Cardiopulmonary Resuscitation.

Patient outcome: The patient had been on fludrocortisone long-term before, but did not like the skin thinning/bruising. Due to her dire circumstances, though, she was discharged on fludrocortisone tablets from both admissions for PTDI. She stopped taking fludrocortisone after one month.

The patient was given SQ DDAVP Rx during and after her first hospitalization for PTDI. Upon discharge, she was instructed to dose herself in the abdomen. After months went by, her entire abdomen was bruised and purple, and each SQ injection left a welt, like a bee sting. It took about five years for these welts to disappear, and the patient opted for nasal or po vasopressin therapy thereafter, to treat the second episode of PTDI. She was taken off the SQ DDAVP only after presenting after the PTDI had resolved, unbeknownst to her.

On the second case of PTDI, the patient was given intranasal DDAVP for the evenings, so as not to awaken during the night to urinate. During the day, she was given po desmopressin. She suffered multiple bouts of either hypovolemic hypernatremia and near-renal failure, or hypervolemic hyponatremia and the feeling of impending uncal herniation. The “Brittle Dysautonomia” had been severely affected by the PTDI, and fluid management was the priority of the day. The following clinical signs far outweighed the need to go in for serum Na checks over days, weeks, and the impending months:

(a) Daily body weights in the morning;
(b) How the same ring fit on the same finger each day;
(c) Urine color (i.e., “Lasix® pee” that was clear, vs. dark yellow urine);
(d) Urine volume;

(e) Increased salt and electrolyte intake, especially Ca, K, and Mg.

The patient was successfully able to wean herself off the nasal spray first by developing a tolerance to it and substituting it for a po pill dose instead. She watched and investigated her clinical symptoms, allowing the PTDI to declare it daily, before taking the remedy. This is of utmost importance, and one can liken it to the sedation and paralization of a NeurolCU patient who needs a daily neurological exam. This neurologic patient needs to be [1] easily arousable and not under deep sedation and [2] not overdosed on paralytic drugs, so that a physical exam can be elicited almost immediately.

In this manner, the management of these patients is to first avoid hypervolemic hyponatremia. If it is caused iatrogenically by overdosing with DDAVP, and the patient has severe clinical complaints, then do not ignore them. These are the shifting water molecules going into the extracellular fluid to cause edema of the brain and eyes, and it is very, very painful. This author does not believe that any in-house patient would have this occur as a "long-term" or "chronic" event. It would only be iatrogenic and "quick," for example, by both dosing the patient with DDAVP and leaving her IV going at 1 ml/kg/hr.

This author’s opinion is to treat in-house hyponatremia as an “acute” event, avoid seizures, uncal herniation and death by avoiding one drop of even 5% saline, and just being patient (it is supposed to be a ‘slow’ correction, anyway) by withdrawing all IVs and PTDI medications, so that the DI can “declare itself” again, self-correcting the hyponatremia in its own time. Be prepared to immediately avoid reinitiation of hypervolemia again, because the patient’s brain is also damaged by the TBI and the less harm, the fewer changes, the more you assess the patient, the better.

ICU admission is a reason for observation and knowing when to check electrolytes and respond to cardiopulmonary arrest or other iatrogenic sequelae. Few endocrinologists have seen PTDI occur in a patient who also has dysautonomia. It is also a situation where the ER doctors need to test the patient’s serum Na upon arrival, then give fluid resuscitation if the patient has heat stroke, and test the patient’s serum Na for hypovolemic, ring slides on same finger, around in a circle. This author’s opinion is to treat in-house hyponatremia as an “acute” event, avoid seizures, uncal herniation and death by avoiding one drop of even 5% saline, and just being patient (it is supposed to be a ‘slow’ correction, anyway) by withdrawing all IVs and PTDI medications, so that the DI can “declare itself” again, self-correcting the hyponatremia in its own time. Be prepared to immediately avoid reinitiation of hypervolemia again, because the patient’s brain is also damaged by the TBI and the less harm, the fewer changes, the more you assess the patient, the better.

So few doctors know what either dysautonomia or PTDI is, that the patient in pain and agony, with brain fog and expressive dysphagia can hardly be relied upon to be her best advocate. To boot, some doctors view this as all being so foreign that they “chalk the patient up” as being a mental health patient instead of a physical health patient, thus putting the patient through more anguish, duress, and contributing to additional PTSD from not being “believed.” Let us avoid that at all costs. Give the patient and the physician: patient to be her/his own “rebel,” as this is the only way that this patient has survived [23].

References


12. Aranda Margaret (2016) Self-reporting of finger bloating and inability to take off medical school class ring while hypervolmic; then when hypovolemic, ring slides on same finger, around in a circle.


17. Aranda Margaret Self-reporting of unilateral epistaxis after use of DDAVP nasal spray and subsequent nare inflammation, while on clopidogrel therapy.


