

# Non-Psychogenic Polydipsia With Hyponatremia

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## Abstract

Polydipsia with hyponatremia in patients is well-known in the literature. Descriptive terms for such patients include, "compulsive water drinking",<sup>1</sup> "self-induced water intoxication",<sup>2,3</sup> "self-induced water intoxication and psychosis",<sup>4</sup> "psychogenic polydipsia", "primary polydipsia", and "psychosis-intermittent hyponatremia-polydipsia (PIP) syndrome".<sup>5</sup> Non-psychogenic polydipsia with hyponatremia (individuals without identifiable psychiatric, brain, or endocrine disorder) is a rare entity. Reported herein is the course of a woman who had been drinking up to 8 liters of water a day, and was subsequently started on a diuretic for hypertension. Non-psychogenic polydipsia with hyponatremia is reviewed after a MEDLINE database search for articles, dates ranging from 1954-2004, under the terms "psychogenic and polydipsia", "polydipsia and hyponatremia", and "psychogenic hyponatremia". Articles describing polydipsic patients with endocrine, organic brain, personality or psychiatric disorders were excluded, and additional cases were evaluated after careful review of the relevant articles and their references. Non-psychogenic polydipsia with hyponatremia is categorized.

## BACKGROUND

Several categories of non-psychogenic polydipsia with hyponatremia have been reported including: individuals voluntarily drinking excessive fluid ± low solute intake, those iatrogenically infused with excessive hypotonic fluid or advised to "drink plenty", children that are fed excessive fluid, by intention or naïve caretakers, or any of the above with concomitant use of a substance impairing free water excretion. Table I. includes well-documented cases of non-psychogenic polydipsia with hyponatremia, excluding individuals iatrogenically infused with excessive fluid and children that were fed excessive fluid.<sup>6,7,8,9,10,11,12,13,14,15,16,17,18,19,20,21,22,23,24,25,26,27,28,29,30</sup> All cases in Table I. received medical evaluation. Acute water intoxication occurred within a few hours to days in all individuals but those with a long history of non-psychogenic polydipsia prescribed a medication associated with the syndrome of inappropriate anti-diuretic hormone release (SIADH, discussed later).

## Figure 1

Table I: Section 1. Cases of Non-psychogenic polydipsia with hyponatremia

Category:	Case	Reference	Age (yrs)	Sex	Medications
Acute:	1	6	44	M	X
Polydipsia	2	7	24	M	N
and low	3	7	45	M	N
solute	4	8	19	M	N
intake	5	9	34	F	Vitamin C, calcium carbonate, prenatal vitamins, selenium, chromium, tolmetin
	6	10	18	M	N
Acute:	7	11	28	M	Ampicillin and 4 liters daily for 1 week prescribed by doctor
Following	8	12	40	F	X
advice of	9	13	76	F	NPH insulin, hydrochlorothiazide
medical	10	14	70	M	Trimethaprim/sulfamethoxazole
staff	11	15	64	M	N
	12	15	59	M	Antibiotics for urinary tract infection
Acute:	13	16	21	F	N
Preparing	14	17	79	F	X
for					Carisoprodol/aspirin, ibuprofen, oxycodone/acetaminaphen, levo-thyroxine, decadron (1 dose on admission)
medical	15	18	64	F	N
test or	16	19	80	F	N
imaging	17	20	72	F	Indomethacin, propranolol
	18	21	72	M	N
	19	22	58	F	Nisoldipine, vitamin E, multivitamin, 1 square phenolphthalein as needed for constipation
Acute:	20	23	9.5	M	Topical aspirin and parestic for toothache pain
Self-					Cold tablet (phenylpropanolamine, chlorpheniramine, dextromethorphan), acetaminophen for 10 days
inflicted	21	24	40	F	N
	22	25	27	F	N
Less					
acute:	23	26	66	F	Amiloride, hydrochlorothiazide
Polydipsia	24	27	56	M	Chlorthalidone
and	25	28	61	M	Captopril, digoxin, prednisone, spironolactone, theophylline, calcium carbonate, dihydrotachysterol
medication	26	29	46	F	Chlorthalidone, bendroflumethiazide
	27	30	63	F	Lisinopril



signs and symptoms similar to water intoxication as a result of hypo-osmolar hyponatremia.

Children whose parents improperly fed their children solute poor solutions may develop hyponatremia and its consequential manifestations.<sup>39,40,41,42,43,44</sup> Additionally, an infant was reported to have developed acute hyponatremia and seizures after swallowing excess water during a swimming lesson.<sup>45</sup>

Transurethral resection of the prostate or a bladder tumor may induce iatrogenic hyponatremia as a consequence of administration of up to 20 to 30 liters of nonconductive hypotonic flushing solutions, like glycine or sorbital during surgery.<sup>46,47,48,49</sup> Variable amounts of these solutions may enter the circulation, either by leakage into the retroperitoneal space through the perforated prostatic capsule or by direct entry into the prostatic veins. A similar sequence may follow hysteroscopy in women with the use of glycine solutions.<sup>50,51</sup>

Another iatrogenic case<sup>52</sup> involved a 23 year-old woman given caudal and subsequently epidural anesthesia with intravenous dextrose water for delivery that became hypotensive, and was resuscitated with infused with 5.5 liters over nine hours of dextrose and water. Her serum sodium fell to 115 mmol/L and her baby delivered immediately; developed transplacental hyponatremia with a sodium of 119 mmol/L, and improved without treatment. Successful treatment of the mother consisted of normal saline and furosemide.

In U.S. Army trainees, a number of cases hypo-osmolar  $\pm$  hyponatremia secondary to excess water drinking (up to 2 quarts an hour) have been reported.<sup>53</sup> Most trainees were in good health, however, these cases lack sufficient laboratory data to include in Table I. Relatively rare reports of the syndrome include: (a) dilutional hyponatremia following treatment of acute dehydration due to hyperemesis gravidarum<sup>54</sup> (b) iatrogenic induction of fatal water intoxication by gastric lavage with 7.8 liters of water within 2 hours, during mistreatment of a healthy 21 year-old in an apparent suicide attempt that was wrongly considered to have taken a poison containing phosphorus.<sup>55</sup>

In 1923, Rowntree<sup>56</sup> first described the signs and symptoms of water intoxication. Early symptoms include generalized malaise, nausea, vomiting, headache and blurred vision. Often, patients can have mental status changes, such as confusion, restlessness, irritability, and lethargy. Other signs

include muscle tremor and cramps, psychosis, seizures, increased salivation and diarrhea may be noted.

Hyperpyrexia and anhidrosis have been reported.<sup>3</sup> If severe, water intoxication can lead to coma and death, usually as a result of pulmonary or cerebral edema.<sup>57,58</sup> Initial presentation of water intoxication may be seizure and coma, if the sodium level is severely low or drops suddenly.<sup>59,60,61</sup> Whenever the patient's clinical status changes, the serum sodium level should be rechecked since it can drop precipitously during the course of a day.<sup>62</sup>

Premenopausal women apparently effect less efficient osmotic adaptation,<sup>63</sup> and as a result, appear to be at greater risk for severe hyponatremic symptoms with up to 25-times greater risk compared to men for residual neurologic injury following symptomatic hyponatremia.<sup>63</sup> Sex hormones may play a role in the susceptibility of adult women as prepubertal boys and girls had equal risk of symptomatic hyponatremia.<sup>64</sup>

Polydipsia and polyuria can also result in chronic changes or complications, such as bowel and bladder dilation,<sup>65</sup> hydronephrosis,<sup>66</sup> and renal failure.<sup>67</sup>

### CASE REPORT

A 71-year-old woman, originally from St. Lucia, recently diagnosed with hypertension, hyperlipidemia, and prior right eye cataract was evaluated for a syncopal episode after rising from a seated position during treatment with labetalol and nifedipine. The blood pressure was 178/76 mm Hg with a pulse rate of 76 per minute. Physical exam including a full neurological exam was otherwise unremarkable. At that time, the serum electrolytes were as follows: sodium 131 mmol/L, potassium 3.9 mmol/L, chloride 95 mmol/L, bicarbonate 19 mmol/L, the blood urea nitrogen 15 mg/dL (5.35mmol/L), the serum creatinine 0.9 mg/dL (79.6  $\mu$ mol/L), and the serum glucose 112 mg/dL (6.22 mmol/L). Liver screening tests and cardiac enzymes (creatinine phosphokinase and troponin I) were normal. An electrocardiogram showed normal sinus rhythm, normal axis, no hypertrophy, a Q wave in V1 with inverted T's in V1-3, no change compared with a previous EKG. Computer assisted tomography (CT) of the head revealed a small hypodense area in the left caudate region, interpreted as an infarct of indeterminate age. Syncope was attributed to orthostatic hypotension secondary to labetalol. The patient was discharged to her home with the additional medication of fosinopril and advised to discontinue labetalol. The patient did not fill the fosinopril prescription and returned to

clinic one week later when the blood pressure was still elevated at 180/96 mm Hg, and hydrochlorothiazide 12.5 mg daily was added to her drug regimen.

Twenty-three days later, the patient reported to ambulatory surgery clinic for pre-operative evaluation for cataract extraction. Her regimen consisted of nifedipine, hydrochlorothiazide, and pravastatin. Pre-operative evaluation detected a serum sodium of 116 mmol/L, confirmed by a repeat specimen. Electrolytes were as follows: potassium 3.2 mmol/L, chloride 73 mmol/L, bicarbonate 30 mmol/L, the blood urea nitrogen was 10 mg/dL (3.57 mmol/L), the serum creatinine 0.7 mg/dL (61.9  $\mu$ mol/L), and the serum glucose 101 mg/dL (5.61 mmol/L). Surgery was cancelled and the patient was referred to the emergency room where further testing disclosed a serum uric acid was 4.5 mg/dL (267  $\mu$ mol/L), serum osmolality of 251 mosm/kg, a urine osmolality of 216 mosm/kg, a urine sodium of 8 mmol/L, with a urine specific gravity of less than 1.005. Calcium, liver function tests, cortisol, and thyroid function tests were normal. The electrocardiogram was unchanged.

The patient reported feeling weak and dizzy recounting a history of drinking up to 8 liters of water a day, for "years" prior to starting any medication. The patient reported that she "felt good drinking cold water", would drink if her throat was dry, and sometimes would drink when not thirsty. She denied being advised by any medical doctor to drink in those quantities as well as a history of psychiatric, endocrine, or neurologic disease; and was unaware of the prior finding of a cerebral infarct. Besides the prescribed medications, she had used acetaminophen and ibuprofen in the past for headaches secondary to uncontrolled hypertension. Since starting antihypertensive medication, she did not take analgesics. She denied nausea, vomiting, diarrhea, constipation, chest pain, dyspnea, orthopnea, pedal edema, difficulty sleeping, or seizures.

The patient had no known allergies, previous surgery, or family history of renal, cardiac, lung disease or cancer. She denied a history of smoking, alcohol, or illicit drug use.

Physical examination, noted the blood pressure as 161/80 mm Hg, the pulse 88 per minute, respirations 18 per minute and an oral temperature of 37.1°C. The height was 1.70m and the weight was 57.7 kg. Pulse oxygenation was 100% on room air. Physical exam noted a right eye cataract, fine rales auscultated bilaterally at the lung bases, a 2/6 systolic murmur heard at the mitral station. There was neither jugular

venous distention nor pedal edema. Treatment was initiated with normal saline at a rate of 83 ml/hr for 12 hours, while oral fluids were restricted to 1 liter a day, hydrochlorothiazide was discontinued, and fosinopril 20 mg daily was started.

The following day, fluid balance was negative 600 ml and the patient was distinctly improved with a blood pressure of 129/61 mm Hg with a pulse of 82 per minute. The serum sodium was 127 mmol/L, and the remainder of routine blood chemical tests was normal. Seventy-two hours later the sodium rose to 133 mmol/L. The patient who was free of complaints was discharged to her home on fosinopril 40 mg daily, as well as aspirin, pravastatin, and nifedipine.

Two weeks later uneventful cataract surgery was performed without complication. At that time, the serum sodium was 136 mmol/L and the serum potassium was 4.5 mmol/L. Over the ensuing month, the patient has remained compliant with fluid restriction and has not had further problems with her medications.

### MECHANISMS OF POLYDIPSIA AND HYPONATREMIA

The hypothalamus is most closely linked to fluid and electrolyte balance.<sup>36</sup> The internal osmostat controlling serum osmolarity is presumed to be the lamina terminalis at the anterior border of the third ventricle. The lateral hypothalamus appears to be largely responsible for drinking behavior. Synthesis of anti-diuretic hormone (ADH), takes place in the supra-optic nucleus of the hypothalamus, also located anterior to the third ventricle.<sup>36</sup> Hobson and English,<sup>3</sup> in 1963, hypothesized that self-induced water intoxication may be a result of a hypothalamic control disorder.

In health, water balance is maintained at approximately  $\pm$  0.22 percent of body weight.<sup>69</sup> When water loss exceeds 0.5 percent of total body weight, an individual becomes thirsty and seeks water.<sup>70</sup> Fluid compensation in tissues takes place hours after fluid ingestion.<sup>70</sup> Under normal conditions, when appropriate fluid is consumed to correct for dehydration, an individual will stop drinking.<sup>71</sup> A sensation of bloating, nausea, or dizziness is induced, as a regulatory mechanism to control overdrinking.<sup>70</sup> Severe symptomatic hyponatremia is rare in the absence of an organic brain disorder, medical conditions (such as diabetes), polydipsia, polyuria, or excessive fluid retention.<sup>72,73</sup>

Still unclear is why only a subgroup of polydipsic patients become hyponatremic.<sup>36</sup> Hyponatremia does not usually

develop from over ingestion of water, since the renal capacity for free water excretion (about 28 liters a day) is usually more than adequate to handle large fluid volume loads.<sup>74,75</sup> With rare exceptions, additional compounding factor(s) compromising free water excretion coupled with high fluid intake are present when water intoxication develops.<sup>36</sup>

Commonly, SIADH is found in patients demonstrating impairment of free water excretion.<sup>36</sup> Normally, hyponatremia results in an endocrine response leading to the conservation of sodium and excretion of free water.<sup>36</sup> Schwartz et al.<sup>76</sup> in 1957, reported that some hyponatremic patients, in whom ADH was not appropriately suppressed continued to lose sodium in the urine manifesting SIADH. The diagnosis of SIADH requires the combination of hyponatremia plus hypo-osmolality of the serum and extracellular fluid with continued renal excretion of sodium.<sup>36</sup> This combination results in a higher than appropriate urine osmolality relative to the serum, such that the urine is less than maximally dilute.<sup>36</sup> Only in the presence of normal renal, cardiac, and adrenal function and the absence of fluid volume depletion, can SIADH can be diagnosed.

SIADH has many causes. Tumors including carcinoma of the lung, duodenum, or pancreas may secrete ectopic ADH.<sup>36</sup> Neurologic conditions<sup>36</sup> can also lead to secretion of ADH including encephalitis, hemorrhage, stroke, tumor, trauma, Guillan-Barre syndrome,<sup>77</sup> and amyotrophic lateral sclerosis.<sup>78</sup> Pulmonic disorders that can lead to SIADH include pneumonia, tuberculosis, and pulmonary abscess.<sup>36</sup> Other causes include endocrine disorders,<sup>79</sup> HIV encephalopathy,<sup>80</sup> smoking,<sup>81</sup> and idiopathic.<sup>82</sup> A wide-array of medication types have been implicated with SIADH, including analgesics,<sup>83</sup> antiarrhythmics,<sup>84</sup> anticonvulsants,<sup>85</sup> antidepressants,<sup>86</sup> antiglycemics,<sup>87</sup> antilipemics,<sup>85</sup> antineoplastics,<sup>88</sup> antipsychotics,<sup>87</sup> and diuretics.<sup>87,89</sup> In addition, 3,4 methylenedioxy-methamphetamine (ecstasy) has also been reported.<sup>90</sup>

There may be a lowered threshold for ADH release in patients with polydipsia and hyponatremia.<sup>91</sup> Hariprasad et al.<sup>92</sup> termed this "reset osmostat" after reporting on a group of patients with low serum sodium levels and dilute urine. Surprisingly, during fluid deprivation tests, the urine became concentrated while the serum remained hypo-osmolar. This may be a protective response in some patients with chronic hyponatremia, mitigating variation of electrolyte levels at the

expense of continued hyponatremia.<sup>92</sup> There is disagreement in the literature whether or not this is distinct entity<sup>92</sup> or a variant of SIADH.<sup>93</sup>

The kidney alone can influence development of a hyponatremia and hypo-osmolar state. High fluid intake decreases renal concentrating ability to some extent in normal volunteers.<sup>94,95,96</sup>

Diuretics are often implicated in hyponatremia by interfering with sodium resorption at the proximal tubule, exacerbating sodium excretion, and impairing free water excretion.<sup>87</sup> Their use may lead to acute water intoxication. In one study,<sup>59</sup> diuretic use was thought to play a role in 12% of patients with water intoxication. The use of diuretics can make maintenance of electrolyte balance difficult, and should probably be avoided in polydipsic patients.

In the presence of a normal thirst mechanism, an individual can maintain adequate fluid balance, despite taking a thiazide which can decrease the capacity to excrete dilute urine.<sup>87</sup> However, our patient presented the unusual combination of primary polydipsia and a thiazide-induced limitation of urinary diluting capacity.

When the offending agent is discontinued, medication-related SIADH usually reverses. Although SIADH can recur with rechallenge, it may be temporally limited, and possible to reinstitute the medication at a later date in the future.<sup>97</sup> Drugs alone can cause SIADH with water intoxication in non-polydipsic individuals.<sup>33, 98</sup> Non-psychogenic polydipsia with hyponatremia can be quite striking when such a medication is prescribed (as described in the presented patient).

### **IDENTIFYING THE PATIENT**

Initial evaluation of a polydipsic polyuric patient should focus on excluding known causes of polydipsia ± polyuria such as diabetes mellitus, diabetes insipidus, chronic renal failure, hypocalcemia, hypokalemia, lithium, diuretics, alcohol.<sup>36</sup> The differential diagnosis of hyponatremia includes: dehydration with salt depletion, renal dysfunction, heart failure, hepatic failure, endocrine disorders.<sup>36</sup> Also worth noting is pseudo-hyponatremia (a low serum sodium level with normal serum osmolality usually a result of lab error, hyperglycemia, or hyperlipidemia).

Attention should be devoted to uncovering a history of surreptitious fluid drinking, noting the actual amount of fluid consumed and excreted, and the signs, symptoms, and

frequency of hyponatremic episodes.<sup>36</sup> Polydipsia or polyuria may be related to the use of medications. The physical examination should focus on signs of dehydration, edema, congestive heart failure, organomegaly, encephalopathy, cachexia, or focal neurologic deficit.<sup>36</sup> Laboratory testing should include a complete blood count, electrolyte panel, liver function panel, serum osmolality, uric acid, urine sodium, and specific gravity. Endocrine function should be screened with thyroid function panel and morning cortisol level. In any patient presenting with seizure, an EEG can give useful information.<sup>36</sup> A CT scan of the head can be helpful in evaluating for structural pathology such as pituitary, hypothalamic tumor, or stroke.<sup>36</sup> A chest X-ray can help screen for lung cancer, a well-known cause of SIADH.<sup>36</sup> If the patient is admitted, observation alone may suffice to detect polydipsia ± polyuria. Otherwise, a 24-hour urine collection to evaluate the extent of polyuria, can provide fairly accurate estimates of 24-hour urine volumes based on the measurement of urinary creatinine excretion.<sup>99</sup>

### MANAGEMENT

Hyponatremic seizures can be managed with intravenous anticonvulsant medication.<sup>36</sup> Rapid correction of hyponatremia can induce central pontine myelinosis, a demyelination of the pons and other areas of subcortical white matter.<sup>100</sup> Hyponatremia may be associated with the development of bulbar palsy, quadriplegia, coma, and death within days of the correction of hyponatremia.<sup>36</sup> Normal saline can be used, along with oral fluid restriction and frequent electrolyte monitoring. Serum sodium should be corrected a rate no more than 0.5 mmol/L an hour or elevation of 10-12 mmol/L in a 24 hour period. Long term management includes fluid restriction, when appropriate, and removal of exacerbating factors. Cigarette smoking can cause ADH stimulation, therefore, should be kept to a minimum.<sup>36</sup>

### DISCUSSION

Numerous literature reports document hyponatremia in geriatric individuals as a result of medication-related SIADH<sup>83\*84\*85\*86\*87\*88\*89\*90</sup> demonstrating the need for caution in prescribing diuretics to elderly patients, as they are prone to medication-related hyponatremia.<sup>10,15</sup> It is not surprising that the presented patient developed water intoxication following prescription of a diuretic. As evidenced by the scarcity of well-documented cases,<sup>6\*7\*8\*9\*10\*11\*12\*13\*14\*15\*16\*17\*18\*19\*20\*21\*22\*23\*24\*25\*26\*27\*28\*29\*30</sup> non-psychogenic polydipsia with hyponatremia is a rare entity.

Before stating a patient has non-psychogenic polydipsia; endocrine, organic brain, psychological and personality disorders must be excluded. In the current patient presented, a CT of the head revealed a small hypodense area in the left caudate region, most probably an infarct of indeterminate age. The patient was neither aware of having a stroke nor demonstrated neurologic impairment on physical exam. She did not recall symptoms suggestive of neurological impairment, besides the one time episode of syncope where labetalol was thought to be implicated. Interestingly, during that emergency room evaluation, her sodium was 131 mmol/L, perhaps already showing impairment of her renal system to accommodate for her excessive fluid intake. The patient believed that her excessive water intake was by personal choice over a period of years. After discussing water restriction with her, it was demonstrated that this was not a compulsive behavior, evidenced by improvement of the sodium level to 133 mmol/L and higher. Acute water intoxication occurred within a few hours to days in nearly all cases detailed in Table I., except those using SIADH-associated medication concurrently. Most patients reported in the literature made a full recovery, with little or no complications. Unfortunately, a few died as a result of cerebral edema, herniation, or other hospital complications.

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