CLINICAL VIGNETTE

Urinary Retention as a Cause of Acute Hyponatremia

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Introduction

Urinary retention has been hypothesized to be a cause of hyponatremia. Prior been case reports suggested a link between urinary retention and hypotonic hyponatremia.[1-6] The mechanism by which urinary retention causes hyponatremia is not established but bladder distension and pain associated with urinary retention can cause vasopressin release from the posterior pituitary gland.[1] Clinically, this can appear as the syndrome of inappropriate antidiuretic hormone secretion (SIADH).[1] Hyponatremia may be further exacerbated by significantly increased water intake as patients attempt to relieve their urinary retention.[2,3] Urinary retention should be considered in patients with euvolemic hypotonic hyponatremia. Resolution of urinary retention may require close monitoring of sodium levels. This patient developed acute urinary retention in the setting of hematuria, which was associated with acute symptomatic hyponatremia. This case had clear correlation between the onset of urinary retention and onset of hyponatremia.

Case Presentation

A 50-year-old man with past medical history notable for aortic valve replacement and atrial fibrillation on metoprolol and apixaban presented to the emergency room with confusion and lethargy. One week prior to presentation, he had ablation for atrial fibrillation. Immediately following his procedure, he developed hematuria. He was observed overnight in the hospital and discharged with a Foley catheter.

Two days after discharge, urology removed his catheter with resolution of hematuria. He restarted anticoagulation 48 hours after catheter removal. The day after restarting anticoagulation he noted a recurrence of his hematuria in addition to severe suprapubic pain and urinary retention. He was again seen by urology and the Foley catheter was replaced. The patient reported drinking increased fluids following Foley insertion to improve hematuria. However, after few hours, he developed severe lethargy and confusion and was taken to the emergency department.

In the emergency department, he was hemodynamically stable and afebrile. On physical examination, he was lethargic, confused, and oriented only to self. He was euvolemic and Foley catheter was draining cranberry-colored urine. He had a National Institutes of Health Stroke Scale (NIHSS) score of 8, and a stroke and Neurology evaluation were initiated. Evaluation for stroke was negative, including computed tomography (CT) of brain without contrast, CT angiography of head and neck with contrast, and magnetic resonance imaging (MRI) of brain. Comprenhensive metabolic panel and complete blood count were remarkable for sodium of 121 mEq/L. Sodium three days prior was 139 mEq/L. Plasma osmolality was 242 mOsm/kg. N-terminal Pro-BNP was 926. Urine osmolality was 184 mOsm/kg with urine sodium of 32 mEq/L. Chest radiograph was negative.

Given his symptoms, 100mL of 3% hypertonic saline was administered in the emergency department with rapid improvement. On reexamination, he was fully oriented and answered questions appropriately. He had approximately five liters of urine output during his time in the emergency department. He was followed closely in the intensive care unit with improvement and stabilization of his sodium levels. Subsequent testing was negative for hypothyroidism and cortisol deficiency. On discharge, his sodium level was 134 mEq/L with complete resolution of symptoms. The Foley catheter was removed shortly after discharge, and he was restarted on his anticoagulation with no recurrence of hematuria and negative urologic evaluation.

Discussion

This patient presented with acute confusion and lethargy consistent with symptomatic acute hyponatremia suspected to be secondary to urinary retention. The patient had recent labs three days prior to presentation which showed normal sodium. Therefore, symptomatic hyponatremia developed after urinary retention in setting of hematuria. Testing was consistent with SIADH with urine osmolality greater than 100 mOsm/kg and urine sodium was greater than 30mEq/L. Common causes of SIADH were not present. He did not have clinical history or laboratory evidence of hypothyroidism, cortisol deficiency, kidney or liver dysfunction, or heart failure. Although he reported drinking more water after Foley placement, he was not drinking quantities of fluid typically associated with primary polydipsia and developed confusion very soon after Foley placement.

With respect to his SIADH, common causes were ruled out as he did not have malignancy, lung or intracranial pathology, and was not on any medications associated with SIADH. He did note significant pain with the insertion of the Foley catheter, which may have contributed to SIADH. However, the presence

of acute urinary retention, with significant pain, suggests the possibility that urinary retention and bladder distension were the causes of his hyponatremia.

The relationship between urinary retention and hypotonic hyponatremia is incompletely understood. Hypotheses include bladder distension causing vasopressin release from the posterior pituitary in addition to pain associated with urinary retention causes vasopressin release.[1] Another contributing factor is possible significant free water ingestion patients to relieve their urinary retention, exacerbating hyponatremia.[2,3] Hyponatremia evaluation, should include assessment for urinary retention. This may be particularly useful in euvolemic patients without apparent cause of SIADH.

Risk factors for acute urinary retention need to be assessed on presentation. Some causes of acute urinary retention include obstruction (such as benign prostatic hyperplasia, prostate cancer, ovarian cancer),[4] infection (such as urinary tract infection, prostatitis, and pelvic inflammatory disease), neurologic (such as spinal cord injury),[5] and post-operative in the setting of anesthesia, which can be determined via patient history and examination. Urinalysis and urine culture should be obtained on bladder decompression.[7] Both over-the-counter and prescription drugs should be assessed with specific attention to anticholinergics, antihistamines, analgesics, benzodiazepines, calcium channel antagonists, antidepressants, antipsychotics, antiparkinsonians, antiarrhythmics, and muscle relaxants.[1-3]

Acute urinary retention should be approached with prompt bladder decompression, typically via urethral or suprapubic catheterization.[7] In circumstances where patients are asymptomatic or have mild hyponatremia, it may reasonable to relieve the retention with catheterization and monitor sodium levels and possible auto-diuresis. However, if patients are symptomatic with significant symptoms, particularly neurologic symptoms suggestive of impending brain herniation, hypertonic saline may be indicated with low risk of osmotic demyelination syndrome.[8]

This case highlights several principles in the diagnosis and management of hyponatremia. Urinary retention is an independent risk factor for the development of acute hyponatremia, with laboratory tests consistent with SIADH. Patients with no identifiable cause of hyponatremia, particularly euvolemic patients, should be evaluated for urinary retention. Correction of hyponatremia can be achieved with bladder decompression and resolution of urinary retention. Hypertonic saline may be used if patients are symptomatic from hyponatremia. Risk factors for acute urinary retention should be addressed with patients to avoid reoccurrences.

REFERENCES

1. **Galperin I, Friedmann R, Feldman H, Sonnenblick M**. Urinary retention: a cause of hyponatremia? *Gerontology*. 2007;53(3):121-4. doi: 10.1159/000097138. Epub 2006 Nov 17. PMID: 17119339.
2. **Hilton CMH, Boesby L, Nelveg-Kristensen KE**. Severe Hyponatremia Precipitated by Acute Urinary Retention in a Patient with Psychogenic Polydipsia. *Case Rep Nephrol*. 2020 Jul 30;2020:8792897. doi: 10.1155/2020/8792897. PMID: 32802531; PMCID: PMC7414331.
3. **Pyle R, Scott M, Bartholomew J, McGrath S, Moffett B**. Accidental polydipsia and hyponatremia from diphenhydramine urinary retention. *Am J Med*. 2011 Oct;124(10):e5-6. doi: 10.1016/j.amjmed.2011.02.019. Epub 2011 Apr 30. PMID: 21531365.
4. **Mahajan R, Simon EG**. Urinary retention as a cause of hyponatremia in an elderly man. *Indian J Clin Biochem*. 2014 Apr;29(2):260-1. doi: 10.1007/s12291-013-0378-0. Epub 2013 Sep 4. PMID: 24757315; PMCID: PMC3990804.
5. **Parikh J, Dhareshwar S, Nayak-Rao S, Ramaiah I**. Hyponatremia secondary to acute urinary retention. *Saudi J Kidney Dis Transpl*. 2017 Mar-Apr;28(2):392-395. doi: 10.4103/1319-2442.202768. PMID: 28352026.
6. **Saleh AO, Al-Shokri SD, Ahmed AO, Musa AE, Mohamed MF**. Urinary Retention and Severe Hyponatremia: An Unusual Presentation of COVID-19. *Eur J Case Rep Intern Med*. 2020 Sep 11;7(10):001905. doi: 10.12890/2020_001905. PMID: 33083368; PMCID: PMC7546571.
7. **Billet M, Windsor TA**. Urinary Retention. *Emerg Med Clin North Am*. 2019 Nov;37(4):649-660. doi: 10.1016/j.emc.2019.07.005. Epub 2019 Aug 16. PMID: 31563200.
8. **Rondon-Berrios H, Sterns RH**. Hypertonic Saline for Hyponatremia: Meeting Goals and Avoiding Harm. *Am J Kidney Dis*. 2022 Jun;79(6):890-896. doi: 10.1053/j.ajkd.2021.07.020. Epub 2021 Sep 8. PMID: 34508830.