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**CASE REPORT**

# Symptomatic Hyponatremia following Acute Urinary Retention

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## Abstract

**Background:** Hyponatremia due to acute urinary retention is not a common condition. Moreover, symptomatic hyponatremia requires management with hypertonic saline and other supportive measures mostly in ICU settings. **Case report:** We report a case of symptomatic hyponatremia secondary to acute urinary retention probably due to release of vasopressin due to urinary bladder distension which was managed with urinary bladder catheterization and other supportive measures leading to resolution of hyponatremia. **Conclusion:** Acute urinary retention can lead to hyponatremia which may be corrected by treatment of the cause and other supportive measures.

**Keywords:** urinary retention, Hyponatremia, Inappropriate ADH syndrome, vasopressins, urinary bladder

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## Introduction

Hyponatremia is a common as well as fatal condition in patients presenting to Emergency Department (ED). But hyponatremia associated with acute urinary retention is not a well-known entity. We hereby report a patient who presented with symptomatic hyponatremia and which improved gradually following relief of urinary retention and other supportive measures.

## Case Report:

A 48 years old female presented to our Emergency Department (ED) with complaints of inability to pass urine for last 2 days. It was associated with 3 bouts of vomiting since last 12 hours before presentation to ED. Her next of kin also reported that she became extremely sleepy and sometimes drowsy over the last 24 hours. They also reported that she developed intermittent episodes of fever 5 days back which subsided after taking antipyretics. She was a known diabetic but not on any regular medication for the same. On examination she was sleepy but arousable, disoriented with a Glasgow Coma Scale score (GCS) of E3V4M6. She was afebrile and recorded a BP of 130/62 mmHg and a pulse rate of 86/min. Abdominal examination revealed distension of urinary bladder extending almost up to the umbilicus. No other abnormalities were noticed. Blood gas analysis showed normal acid-base status but $Na^+$ levels were 107 meq/L. ECG showed normal sinus rhythm. She was immediately catheterized with 14 F Foley catheter following which 1200ml of urine was drained. To look for cerebral edema, NCCT head was done which showed a normal study. After sending blood samples for CBC, KFT, serum electrolytes, she was admitted to the HDU for further evaluation and treatment. Initial laboratory investigation results were as follows:

Table 1. List of initial laboratory investigation reports

| | |
|---|---|
| Hb | 10.9 g/dL |
| TLC | 4400/$mm^3$ |
| Platelet count | 1.67 lakhs/$mm^3$ |
| RBS | 231 mg/dL |
| Blood urea | 16.76 mg/dL |
| Creatinine | 0.73 mg/dL |
| $K^+$ | 3.6 meq/L |
| $Na^+$ | 107 meq/L |
| $Cl^-$ | 71 meq/L |
| Total Protein | 6.5 g/dL |
| Albumin | 4.55 g/dL |
| Total bilirubin | 0.6 mg/dL |
| Direct bilirubin | 0.2 mg/dL |
| SGOT | 65.8 U/L |
| SGPT | 40.1 U/L |
| Alkaline Phosphatase | 154.6 U/L |
| Uric acid | 3.1 mg/dL |
| CRP | 8.4 mg/dL |

Urine analysis showed hazy appearance with large number of pus cells. Ultrasound of abdomen did not reveal any abnormality. Work-up for hyponatremia revealed low Plasma osmolality (220.89 mosm/L), urine spot $Na^+$- 31 meq/L and increased urine osmolality (125.84 mosm/L). as she was clinically euvolemic with no history of diuretic or hormonal therapy, a probable diagnosis of Syndrome of Inappropriate ADH (SIADH) was considered. Her thyroid profile, and Random cortisol levels were in the normal range. She had no history of prior malignancy, pulmonary disease, hormonal deficiency, neurological disorder or insult. She also had no history of recent surgery was not on any drugs. Initially she was infused with 500ml 0.9% NaCl in the ED, but after the diagnosis of SIADH she was kept on fluid restriction. Over the next 5 days, her sodium levels normalized gradually as shown in Table 2.

Table 2. Improvement of serum $Na^+$ levels from day 1 to day 6 of hospitalization.

| | |
|---|---|
| Day 1 $Na^+$ | 107 meq/L |
| Day 2 $Na^+$ | 111 meq/L |
| Day 3 $Na^+$ | 119 meq/L |
| Day 4 $Na^+$ | 121 meq/L |
| Day 5 $Na^+$ | 123 meq/L |
| Day 6 $Na^+$ | 131 meq/L |

She was also started on empirical Intravenous Ceftriaxone in view of possible urinary tract infection (UTI). Her urine output and KFT remained normal throughout. She also became more awake, alert, oriented and remained afebrile. She was discharged on the $6^{th}$ day with $Na^+$ level of 131 meq/L, $K^+$- 3.6 meq/L and $Cl^-$ levels of 104 meq/L and was advised to follow up in Endocrinology OPD after 48 hours.

**Discussion**

SIADH is a disorder related to impaired excretion of water due to inadequate suppression of ADH leading to hyponatremia [1]. Hyponatremic patients with euvolemia, hypoosmolality in plasma and raised urine osmolality and urine $Na^+$ (usually >40 meq/L) should be suspected of SIADH. There are many causes of SIADH viz central nervous system disorders, malignancies, drugs, recent surgery, pulmonary disease, hormonal deficiency, HIV infection, Hereditary SIADH and Idiopathic [2]. In our case the hyponatremia got gradually corrected following urinary catheterization and fluid restriction. The likely mechanism of which could be release of vasopressin in response to distension of urinary bladder. Similar findings were also reported by Parikh J et al and Galperin I et al where hyponatremia ensued secondary to urinary retention [3,4]. The differential diagnoses of such presentation are CNS infections, hypoglycemia, hyperglycemia, uremia etc. which were ruled out by clinical examination, laboratory and imaging modalities. The cause of urinary retention was possibly due to UTI causing urethritis and urethral edema as other causes like pelvic organ prolapse, pelvic mass were ruled by clinical and ultrasound examination [5,6]. Apart from urinary catheterization, fluid restriction was one of

the mainstays of therapy for hyponatremia. The total fluid intake of the patient was 1 litre per day for 6 days aiming for a negative fluid balance of 500-700ml per day and thus a cumulative negative fluid balance of 3600 ml was achieved over a period of 6 days. We have highlighted this case as such conditions do not require aggressive treatment and can be easily managed with simple measures. Further this case also helps us to understand the different mechanisms which can lead to hyponatremia.

## Conclusion

SIADH is one of the most common causes of euvolemic hyponatremia due to varied known etiologies. But SIADH related hyponatremia secondary to urinary retention and urinary bladder distension is less common and should be suspected in patients presenting with altered sensorium and acute urinary retention.

## Conflict of Interest

The authors declare that they do not have conflict of interest.

## Authors Contribution

All authors contributed equally in conceptualization, data curation, formal analysis, methodology, project administration, investigation, resources, software, validation, visualization, writing of original draft, review and editing, and supervision.

## Informed Consent

Informed consent was obtained from patient and legal heirs of the deceased patient involved in the study for publication.

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