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Case Reports

# Benign Prostate Hyperplasia (BPH) – Induced Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH): A Rare Precipitant of Acute Decompensated Heart Failure

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**Abstract**

**Background:** In Acute Decompensation of Heart Failure (ADHF), precipitating factors must be promptly identified and treated. Urinary retention is rarely recognized as a cause of ADHF. Here, we presented a case of Benign Prostate Hyperplasia (BPH) with urinary retention inducing SIADH, which precipitated a recurrent episode of decompensated heart failure.

**Case Illustration:** a 73-year-old male with a history of hypertension, admitted with signs and symptoms of urinary obstruction. He was scheduled for Transurethral Resection of the Prostate (TURP), but the procedure was delayed due to hyponatremia (serum sodium 119 mmol/l). But while correcting hyponatremia with saline solution, the patient developed worsening dyspnea along with rhonchi and desaturation, which suggested acute heart failure. Chest X-rays showed pulmonary congestion and possible infection; echocardiography indicated concentric LV hypertrophy with normal ejection fraction (LVEF 60%), consistent with HFpEF. He was admitted to the ICU for monitoring, on IV diuretics, fluid restriction, and antibiotics. Intravenous furosemide and tolvaptan improved his symptoms and sodium, leading to ward transfer. Work-up for the hyponatremia showed high urinary sodium without any other cause, supporting the SIADH diagnosis. However, despite treatment and initial improvement, the patient's serum sodium remained below normal (approximately 124 mmol/l) for over a week. He requests removal of the Foley catheter because he already feels much better. However, three days later, he suddenly had severe dyspnea with oxygen saturation dropping to 76% on a nasal cannula and only rising to 89% on 15 liters of oxygen at a non-rebreather mask. He was urgently transferred back into the ICU. Bedside echocardiography showed another episode of decompensated heart failure. Laboratory results showed worsening of hyponatremia (121 mmol/L). The Foley catheter was reinserted because there was evidence of obstruction and inadequate diuresis. The furosemide IV and tolvaptan were continued. The patient's condition and respiratory status improved, with SpO 2 94-97% on 10 L/min via a simple face mask.

**Conclusions:** Benign Prostatic Hyperplasia (BPH), even in rare instances, can induce SIADH, resulting in renal water retention and potentially causing acute decompensated heart failure.

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**Keywords:** Benign Prostate Hyperplasia, SIADH, Acute Heart Failure, HFpEF

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## Introduction

Acute decompensation in patients with previously stable chronic heart failure is common and associated with substantial morbidity.[5] Causes of such decompensation episodes need to be identified and treated promptly.[5-7] While typical precipitating factors are well described, urinary retention is rarely recognized as a cause of heart failure decompensation.[6,8-9] Benign Prostatic Hyperplasia (BPH) with urinary retention can act as a neurogenic trigger for the Syndrome of Inappropriate Antidiuretic Hormone Secretion (SIADH), which can precipitate Acute Decompensated Heart Failure (ADHF), particularly in vulnerable patients. [1-2] We present a rare case of BPH-induced SIADH that resulted in severe, refractory hyponatremia, leading to recurrent, life-threatening episodes of ADHF in a patient with Heart Failure with preserved Ejection Fraction (HFpEF).

## Case Illustration

A 73-year-old male with a history of hypertension and BPH was admitted for an elective urological procedure to address Lower Urinary Tract Symptoms (LUTS) resulting from prostatic hyperplasia. He first came to the urology department in January 2025 and has been diagnosed with BPH since then, with an estimated prostate volume of 32.6 $cm^3$, accompanied by calcification intra-prostate. He had a history of difficulty in urinating for 3 days before hospital admission. He reported sensation to urinate but was only able to produce a small amount with a persistent feeling of residual urine. Due to this symptom, he visited another hospital and got a DC insertion with urinary production of about 500 ml after DC placement. After that, he referred to our hospital and planned to have surgery for his prostate. His vital signs were stable, with blood pressure 151/95 mmHg, heart rate 100 beats per minute, respiratory rate 20 breaths per minute, oxygen saturation 95% on room air, and body temperature 37.5°C. The procedure was subsequently postponed following admission when routine preoperative laboratory investigations revealed severe hyponatremia, with a serum sodium level of 119 mmol/l. Other laboratory and physical findings were normal.

During the initial hours of electrolyte correction with hypertonic and isotonic saline (3% NaCl at 20 mL/h, administered abruptly and concurrently with 0.9% Nacl at 40 mL/h), the patient suddenly developed dyspnea and orthopnea. Blood pressure was 161/113 mmHg, heart rate was 100 beats per minute, and oxygen saturation was 83% on nasal cannula, improving to 96% with 10 L/min of oxygen delivered via a simple face mask. Bilateral rales were noted on physical examination. Clinical assessment and imaging strongly suggested ADHF. Chest X-ray (CXR) confirmed pulmonary congestion and showed findings suggestive of pulmonary infection. Transthoracic Echocardiography (TTE) demonstrated concentric left ventricular hypertrophy with preserved systolic function (Left Ventricular Ejection Fraction [LVEF] 60%), normal global kinetic wall motion, and normal valvular function, consistent with HFpEF. The Inferior Vena Cava (IVC) diameter (9/12 mmHg) and an estimated Right Arterial Pressure (eRAP) of 8 – 10 mmHg indicated a mild elevation; however, the predominant clinical manifestation was acute pulmonary oedema. The patient was admitted to the intensive care unit for close monitoring and standby for intubation if necessary. He also received intravenous diuretics, fluid restriction, and antibiotics for pneumonia.

The initial treatment of ADHF involved administering intravenous Furosemide and the vasopressin V2 receptor antagonist, Tolvaptan. The patient's dyspnea improved, and his serum sodium levels rose slightly, so he was stepped down to the ward. An investigation into the ongoing hyponatremia showed high urinary sodium (Table 1) without any other appropriate cause, strengthening the evidence of SIADH.

**Table 1.** Urine electrolyte panel.

| Examination | Urine Electrolyte Panel (Na, K, Cl) | | |
|---|---|---|---|
| Volume Urine / 24 Hours | 3600 | — | mL/24 hours |
| Sodium (Na) – Urine | 310 | 54–150 | mEq/L |
| Potassium (K) – Urine | 58 | 25–100 | mEq/24 hours |
| Chloride (Cl) – Urine | 317 | 85–170 | mEq/24 hours |

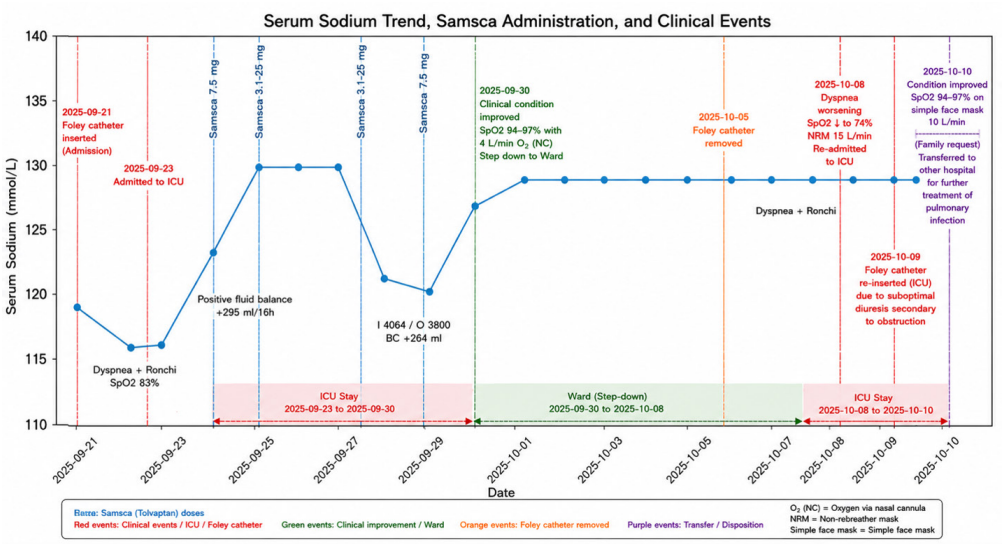


**Figure 1.** Temporal relationship between serum sodium levels, tolvaptan administration, and clinical events.

Despite appropriate medical treatment and an initially favorable response, the patient's serum sodium remained below normal, around 124 mmol/L, for more than one week of hospitalization. The patient clinically improved and requested removal of the Foley catheter on the 5th of October. On the fourteenth day, however, he suddenly experienced a severe recurrence of dyspnea, accompanied by hypoxemia, with oxygen saturation falling to 76% on a nasal cannula and rising only to 89% with a non-rebreather mask at 15 L/min. He was urgently transferred again to the ICU for stabilization. Bedside echocardiography revealed an IVC diameter of 13/17 mmHg with an estimated eRAP of 8-10 mm$H_2O$. Laboratory tests showed worsening hyponatremia with 121 mmol/L. Hypertonic 3% Nacl was continued intermittently along with intravenous furosemide. The Foley catheter was reinserted, and this time we ensured it remained in place to prevent urinary retention and maintain continuous urinary monitoring.

In Figure 1, the graph shows the serial trend of serum sodium levels during hospitalization, along with the timing and dosage of tolvaptan (Samsca) administration and major clinical events. The patient was admitted on September 21, 2025, with signs and symptoms of urinary obstruction and was scheduled for Transurethral Resection of the Prostate (TURP). A Foley catheter was inserted at admission. While correcting hyponatremia with normal saline and 3% NaCl, the patient developed worsening dyspnea, ronchi, and severe desaturation to $SpO_2$ 83% on September 23, 2025, which led to ICU admission. After starting tolvaptan, serum sodium gradually rose from 116–119 mmol/L to 130 mmol/L between September 25–27, 2025, though intermittent positive fluid balance persisted. On September 30, 2025, the patient showed clinical improvement, with $SpO_2$ 94–97% on 4 L/min oxygen via nasal cannula, and was transferred out of the ICU. Following Foley catheter removal on October 5, 2025, serum sodium remained relatively stable at approximately 129 mmol/L, but ongoing dyspnea and rhonchi indicated continued congestion despite biochemical improvements. On October 8, 2025, the patient's respiratory condition worsened, with severe desaturation to 74%, requiring a non-rebreather mask oxygen at 15 L/min and ICU re-admission. On October 9, 2025, Foley catheter re-insertion was performed due to suspected urinary obstruction and insufficient diuresis. By October 10, 2025, respiratory status had improved, with $SpO_2$ 94–97% on a simple face mask at 10 L/min; however, the patient was ultimately transferred to another hospital for further treatment of a pulmonary infection at the family's request.

## Discussion

Chronic heart failure leads to frequent hospitalizations and increasing healthcare costs.[5] Effective management requires not only diagnosing heart failure but also identifying and treating the factors that precipitate clinical deterioration in both outpatient and inpatient settings.[5-7] In cases of decompensated chronic heart failure, determining the trigger of the

episode is essential, regardless of whether systolic or diastolic dysfunction is present.[9]

Common precipitating factors for heart failure decompensation include medication non-adherence, myocardial ischemia, cardiac arrhythmias, sepsis, uncontrolled hypertension, and suboptimal medical therapy before admission.[9] In contrast, urinary retention has not been well described in the literature as a trigger for decompensated heart failure.[1,8-9] Our case report aims to highlight this connection, where SIADH caused by Benign Prostatic Hypertrophy was identified as the cause of urinary retention, which in turn led to refractory decompensated heart failure.

We observed that the clinical course following diuretic administration, hypersaline infusion, and catheter insertion was both dramatic and revealing (Figure 2). Bladder decompression led to a sustained increase in serum sodium levels. Within a couple of days, sodium levels nearly normalized (134 mmol/L), and the patients' respiratory status improved. The whole history of it became evident clinically that, despite the ongoing pulmonary infection, the earlier refractory hyponatremia and recurrent episodes of ADHF were primarily attributable to neurogenically induced SIADH, which resolved only after the mechanical trigger of urinary retention was eliminated.

This case describes a rare but important association between severe urinary retention and ADHF in a patient with HFpEF. Prolonged and/or marked bladder distention due to urinary tract obstruction activates stretch receptors that stimulate afferent pathways projecting to the hypothalamus, constituting a neurogenic reflex. This, in turn, triggers non-osmotic release of Antidiuretic Hormone (ADH, also called vasopressin), resulting in SIADH. The ensuing inappropriate ADH secretion leads to renal water retention, dilution of serum sodium, and subsequent hyponatremia. In addition, pain caused by bladder distention may further enhance ADH release, thereby worsening the disorder. Impairment of free water excretion due to ADH induces intravascular volume expansion, culminating in volume overload. These mechanisms highlight how urinary tract obstruction can act as an extracardiac neurohormonal trigger that precipitates ADHF (Figure 2).[2-3]

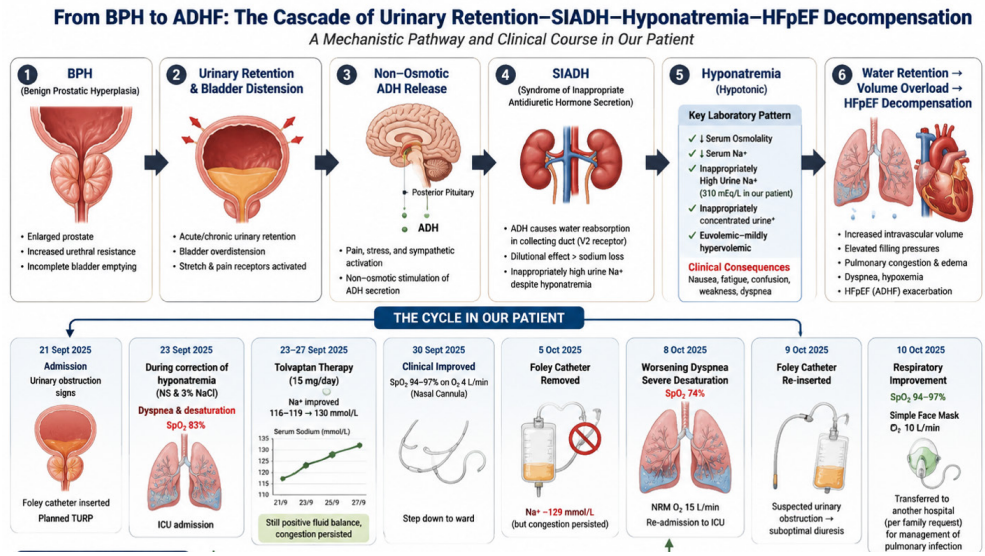


**Figure 2.** Central illustration.

While there is limited literature specifically addressing ADHF caused by SIADH due to urinary obstruction, some physiologically plausible links exist. Scattered case reports describe urinary obstruction-induced SIADH-like hyponatremia and water retention, which can lead to volume overload. Manappallil et al. (2019) documented a case involving a 60-year-old male patient with a history of BPH who presented with acute urinary retention, confusion, and symptomatic severe hyponatremia. Laboratory assessments revealed findings consistent with SIADH, including decreased serum osmolarity and inappropriately concentrated urine. Imaging and further diagnostic investigations excluded other common etiologies of hyponatremia. Following bladder catheterization and resolution of urinary obstruction, the patient exhibited rapid diuresis and gradual normalization of serum sodium levels, supporting the hypothesis that bladder distention from BPH-associated urinary retention may have

triggered non-osmotic ADH release, resulting in a transient SIADH-like condition.[10] Mahnič et al. (2024) reported the case of an elderly man presenting with severe hypoosmotic hyponatremia associated with acute urinary retention and bladder distension. The laboratory findings were compatible with SIADH, while other common causes of hyponatremia were excluded. The authors proposed that urinary tract obstruction and bladder overdistension triggered non-osmotic ADH release, likely mediated by pain and sympathetic nervous system activation, resulting in water retention and dilutional hyponatremia. Following insertion of a urinary catheter and relief of the obstruction, the patient developed marked post-obstructive diuresis, accompanied by a rapid, spontaneous correction of serum sodium levels without the need for aggressive additional therapy. The report emphasizes that urinary retention is an underrecognized reversible cause of SIADH-like hyponatremia, and that catheter decompression can lead to significant clinical and biochemical improvement, as in our case.[11]

Given the overall clinical picture, we considered the possibility of an alternative mechanism of hyponatremia, particularly in the context of heart failure, and evaluated the impact of saline administration on clinical outcomes. The patient developed worsening congestion and respiratory deterioration during saline correction, indicating hypervolemic hyponatremia associated with ADHF. Nonetheless, the overall evidence indicated that SIADH secondary to urinary tract obstruction remained the primary etiology. At presentation, the patient had urinary obstruction requiring Foley catheter placement – a well – recognized trigger for non-osmotic ADH release via bladder distention and sympathetic nervous system activation. Moreover, serum sodium levels improved substantially following bladder decompression and tolvaptan therapy, as in the previously reported case. Furthermore, episodes of clinical deterioration recurred after Foley catheter removal but were alleviated upon reinsertion, likely due to persistent obstructive pathology limiting effective diuresis. Finally, persistently elevated urinary sodium excretion despite hyponatremia was atypical for classic hypervolemic hyponatremia solely attributable to heart failure, in which renal sodium retention is usually expected.

In a patient with underlying HFpEF and limited diastolic reserve, marked by concentric hypertrophy, the slight increase in volume caused by refractory SIADH was sufficient to push the patient past the Frank-Starling threshold. This resulted in two separate episodes of ADHF, each requiring urgent medical care. The fact that hyponatremia did not improve with fluid restriction and tolvaptan therapy only suggested that the primary trigger, bladder distension, persisted. The rapid and sustained improvement of SIADH, combined with the stabilization of cardiac function after Foley catheter insertion, clearly indicated that BPH-induced urinary retention was the main cause of this complex multisystem presentation. Although definitive confirmation of SIADH was not achieved due to the absence of urine osmolality measurements, the clinical and biochemical improvement following relief of urinary obstruction supports a plausible association between urinary retention, hyponatremia, and recurrent episodes of heart failure decompensation.[1-2,4]

## Conclusion

BPH can, on rare occasions, precipitate SIADH, leading to severe and treatment-refractory hyponatremia. In patients with pre-existing cardiac disease, the resulting volume expansion from SIADH may trigger critical, potentially life-threatening episodes of ADHF. This case highlights the importance of recognizing urinary retention as a potential cause of SIADH. It also demonstrates that targeted, non-pharmacological interventions such as bladder decompression can effectively correct the electrolyte disturbance and ameliorate the associated cardiac decompensation.

## List of Abbreviations

| | |
|---|---|
| ADH | Antidiuretic Hormone |
| ADHF | Acute Decompensated Heart Failure |
| BPH | Benign Prostatic Hyperplasia |
| CXR | Chest X-Ray |
| eRAP | estimated Right Arterial Pressure |
| HFpEF | Heart Failure with preserved Ejection Fraction |
| IVC | Inferior Vena Cava |
| LUTS | Lower Urinary Tract Symptoms |
| LVEF | Left Ventricular Ejection Fraction |
| TURP | Transurethral Resection of the Prostate |
| TTE | Transthoracic Echocardiography |
| SIADH | Syndrome of Inappropriate Antidiuretic Hormone Secretion |

## Ethical Clearance

Not applicable.

## Publication Approval

All authors consent to the publication of this manuscript.

## Author Contributions

All authors contributed to the literature search and review. All authors read and approved the final manuscript.

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## Conflict of Interest

The authors declared that they have no competing interests.

## Availability of Data and Materials

All data backing this case are included in the manuscript. No extra datasets were created or examined in this study.

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## Generative AI and AI-Assisted Technologies in the Writing Process

Authors acknowledge that artificial intelligence (AI) tools were only used to assist in language editing and did not generate or alter the scientific content, analyses, or conclusions presented in this manuscript.

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