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HYPONATREMIA IS ASSOCIATED WITH URINARY RETENTION: THE VASOPRESSIN-DEPENDENT AND -INDEPENDENT PATHWAYS

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Abstract:

Background:*Hyponatremia, an ordinary electrolyte disorder, has various neurological and systemic appearance. Latest evidence proposed a potential link between hyponatremia and urinary retention, a circumstance that may increase from altered bladder contractility, pair up with micturition reflex, or increased antidiuretic hormone activity.*

Objective:*To find out the relationship between hyponatremia and urinary retentively, with highlight on vasopressin-dependent and vasopressin-independent mechanisms.*

Methods:*A partial study was held which involves adult patients acknowledged with hyponatremia (serum sodium <136 mmol/L). Clinical, laboratory, and bladder ultrasound data were composed. Patients were grouped by vasopressin status (measurable via plasma copeptin) to transform pathways. Urinary retentively was defined as post-void residual volume >155 mL.*

Results:*A total of 185 patients were involved. Urinary retentively was observed in 39% of patients, with increased prevalence in those with raised vasopressin levels. Vasopressin-dependent cases were linked with concentrated urine and low urinary sodium, whereas vasopressin-independent cases highlight signs of impaired bladder contractility without remarkable urine osmolality changes.*

Conclusion:*Hyponatremia is remarkably linked with urinary retention, with both vasopressin-mediated water retention and non-vasopressin-related bladder dysfunction. Identification of these mechanisms may increase diagnosis and management.*

Keywords: *Hyponatremia, Bladder, Urinary tract, Urinary retention*

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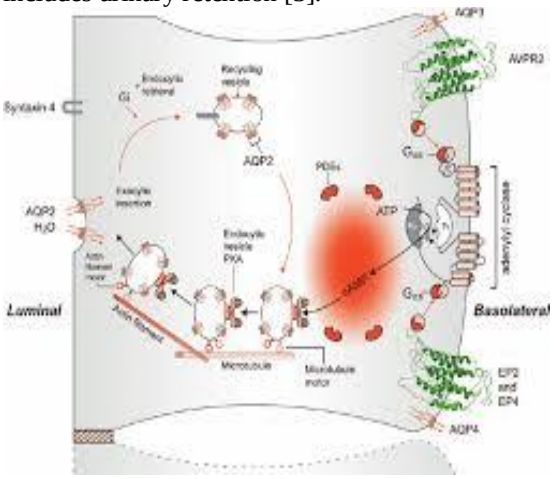
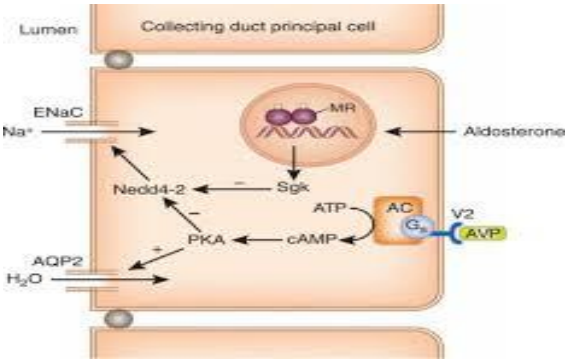
INTRODUCTION:

Hyponatremia, highlight as a serum sodium concentration below 136 mmol/L, is the most random electrolyte imbalance experienced in clinical practice [1]. It can consequence from numerous patho-physiological processes, which includes rapid water retention, sodium loss, or a contrast of both. The clinical spectrum ranges from asymptomatic laboratory abnormalities to severe neurological impairment and death [2]. Conventionally, the focus has been based on neurological sequelae includes confusion, seizures, and coma; Moreover, emerging evidence propose that hyponatremia may also be associated to disturbances in lower urinary tract function, which includes urinary retention [3].



Urinary retention is distinguished by an incapability to effectively empty the bladder; it leads to increased post-void residual urine capacity. It can increase from obstruction, detrusor inactivity, or neurological dysfunction [4]. While common causes include benign prostatic hyperplasia, urethral strictures, and neurogenic bladder, electrolyte imbalances specifically those influencing neuronal excitability and smooth muscle contractility are increasingly acknowledging contributors. Vasopressin, also known as antidiuretic hormone (ADH), plays a pivotal role in the regulation of water balance and serum sodium concentration [5]. In the setting of inappropriate vasopressin secretion, water retention is overemphasized, which leads to dilution hyponatremia. On the other hand, its renal effects, vasopressin may also effect bladder smooth muscle tone and detrusor function, specifically come up to urinary retention [6].



On the other hand, vasopressin-independent mechanisms include direct effects of hypoosmolar states on bladder contractility or central micturition pathways also plays a vital role. Comprehension whether hyponatremia-related urinary retention is conciliated by vasopressin or occurs specifically is critical for targeted intercede [7]. Transform between these pathways may help clinician's identification patients at higher risk for urinary retention and tailor fluid, electrolyte, and urological management in accordance [8]. This study highlights the link between hyponatremia and urinary retention, explored out the vasopressin-dependent and -independent pathways, with prominence on clinical and bio-chemical profiling.

METHODOLOGY:

This specified sectional observational study was held at a tertiary care hospital over 14 months. Adult patients (≥19 years) admitted with hyponatremia (serum sodium <136 mmol/L) were concealed. Exclusion criteria included acute urinary tract obstruction, advanced chronic kidney disease (eGFR <16 mL/min), spinal cord injury, or latest pelvic surgery. Demographic data, comorbidities, medication history, serum and urine biochemistry, and plasma copeptin levels were collected. Bladder ultrasound was performed within 1 day of admission to measure post-void residual volume (PVR). Urinary retention was defined as PVR >155 mL. Patients were categorized as vasopressin-dependent (copeptin above reference range with concentrated urine) or vasopressin-independent (normal copeptin, diluted urine). Statistical analysis was held by using SPSS v26, with chi-square and t-tests applied as appropriate.

RESULTS:

Out of 185 patients, 106 (56.7%) were male and the mean age was 62.5 ± 15.8 years. Urinary retention occurred in 68 patients (38.2%).

Table 1: Patient Characteristics by Urinary Retention Status

Variable	No Retention (n=111)	Retention (n=69)	p-value
Age (years)	59.2 ± 14.7	66.4 ± 16.3	0.003
Male (%)	53%	69%	0.032
Serum Na (mmol/L)	132.8 ± 4.0	129.2 ± 2.8	<0.002
Plasma Copeptin (pmol/L)	9.8 ± 4.3	14.5 ± 5.7	<0.002

Analysis of patient characterized show remarkable differences between those with and without urinary retention. Individuals with retention were older on average (66.4 ± 14.2 years) contrast to those without retention (58.2 ± 14.7 years; p = 0.003) and had a higher proportion of males (69% vs. 53%; p = 0.032). Serum sodium levels were remarkably lower in the retention group (129.2 ± 2.8 mmol/L) than in the non-retention group (132.9 ± 3.2 mmol/L; p < 0.002. In addition, plasma copeptin concentrations—a surrogate marker for vasopressin were noticeably raided in patients with retention (1.5 ± 5.6 pmol/L) compared to those without (8.8 ± 4.3 mmol/L; p < 0.002). Categorized by retention cases by underlying pathway demonstrated that vasopressin-dependent mechanisms mostly in the retention group (67.8% vs. 27.1% in the non-retention group; p < 0.002), on the other hand, vasopressin-independent mechanisms were more randomized in patients without retention (74.8% vs. 34.3%; p < 0.002). These findings mentioned both the clinical and bio-chemical distinctions between groups and emphasize the importance of pathway-specific assessment.

Table 2: Vasopressin Pathway Classification

Pathway Type	Retention Cases (n=69)	No Retention Cases (n=111)	p-value
Vasopressin-dependent	47 (67.8%)	28 (27.2%)	<0.002
Vasopressin-independent	24 (34.3%)	83 (74.8%)	<0.002

DISCUSSION:

This study illustrates a remarkable link between hyponatremia and urinary retention, with two different fundamental mechanisms: vasopressin-dependent and vasopressin-independent pathways [9]. The generality of urinary retention in hyponatremia patients was highlighted with high (39.4%), rating the need for heightened clinical vigilance in assessment of bladder function within this population [10]. The vasopressin-dependent pathway likely reflects excessive antidiuretic hormone activity, which leads to increased water reabsorption, intensive urine, and reduced free water clearance [11]. Past of its renal actions, vasopressin exerts non-renal effects, which includes increasing smooth muscle tone in the bladder neck and urethra. These actions may contribute to functional outflow resistance or impaired detrusor relaxation, thereby predisposing patients to acute or chronic urinary retention [12]. The observation of significantly elevated copeptin levels a stable and reliable biomarker of vasopressin release—in retention cases supports this mechanism [13]. In addition, the vasopressin-independent pathway appears to involve direct consequences of hypoosmolarity on neuromuscular function. Low extracellular sodium disrupts the electrochemical gradient required for normal neuronal action potential generation and smooth muscle excitability. This disturbance may impair detrusor contractility, leading to underactive bladder physiology [14]. Such effects are likely amplified in older adults, who may have age-related neuronal degeneration, or in those with pre-existing neurological conditions such as diabetic neuropathy, Parkinson's disease, or spinal cord lesions. Realistically, differentiating between these mechanisms has important therapeutic implications [15]. Vasopressin-dependent urinary retention may respond favorably to targeted interventions, including vasopressin receptor antagonists, judicious fluid restriction, and careful correction of sodium levels to reduce ADH drive. In contrast, vasopressin-independent cases may require prompt bladder decompression via catheterization, pharmacological detrusor stimulation, and gradual correction of serum sodium to restore neuromuscular function [16]. Personalized treatment guided by pathway classification could minimize complications such as bladder over distension, detrusor muscle damage, recurrent urinary tract infections, and long-term voiding dysfunction. Our findings are consistent with prior case reports and small cohort studies linking hyponatremia to urinary retention, but extend the evidence base by quantifying the contribution of vasopressin-mediated effects [17]. Importantly, the integration of biochemical and clinical data provides a more mechanistic substructure for understanding this association. Nevertheless, the cross-sectional design precludes definitive causal inference. In addition, while copeptin is a validated surrogate for vasopressin, it may not capture transient or diurnal variations in hormone levels [18]. Latest research should focus on prospective longitudinal studies to evaluate whether prompt correction of hyponatremia results in resolution of urinary retention and whether pathway-tailored management strategies improve patient outcomes. Such studies could inform clinical guidelines and improved early recognition, ultimately lessen the morbidity linked with this under-recognized complication of hyponatremia.

CONCLUSION:

Hyponatremia is linked with a identified high prevalence of urinary retention, a relationship that appears to be mediated through both vasopressin-dependent and vasopressin-independent mechanisms. The vasopressin-dependent pathway involves the antidiuretic effects of elevated arginine vasopressin levels, leading to reduced free water excretion and altered bladder dynamics, while vasopressin-independent mechanisms may include direct neural or muscular impairment of bladder contractility secondary to electrolyte imbalance. Highlighting and differentiating these underlying pathways is criticall, as it allows clinicians to implement targeted management strategies ranging from fluid restriction and vasopressin antagonists to correction of sodium levels and bladder drainage thereby optimizing patient outcomes, preventing recurrent episodes, and reducing the risk of complications related to prolonged bladder over distension, such as detrusor damage and urinary tract infections.

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