

## Original Research Article

# Hyponatremia in hospitalized patients with chronic kidney disease; aetiology, treatment, and outcome, in a tertiary care hospital, Dubai, UAE

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## ABSTRACT

**Background:** Hyponatremia is common among hospitalized patients. Unfortunately, articles describing the management of profound hyponatremia (serum sodium <125 mEq/l) in the background of kidney disease are scarce. This review focuses on the incidence, prevalence, patient characteristics, and clinical features among hospitalized chronic kidney disease (CKD) patients with particular attention to CKD stage 3 to 5.

**Methods:** 71 adult patients with CKD stage 3 to 5 and had presented with profound hyponatremia (serum sodium <125 mEq/l) were included. Patient demographic data, laboratory parameters and treatment received were recorded. The primary endpoint was the development of central nervous system (CNS) manifestations, while the secondary outcomes included early mortality (death within 30 days).

**Results:** 97 episodes of hyponatremia were recorded in 71 patients. 35 patients (49%) were UAE national and 53.5% patients were females. 52% were in CKD stage-5 refusing dialysis. Diabetic nephropathy was the underlying cause in 66%. The initial Sodium level upon admission ranged from 107-125 mEq/l, with a mean±standard deviation (SD) value of 117.7±4.54 mEq/l. The correction of hyponatremia had ranged between 24 hours in 60% of the patients to 96 hours. Diuretics were used in conjunction with the saline in 85%. Hospital stay ranged between 2-58 days (average 11.7 days). No CNS symptoms were recorded in any of the treated patients. Three patients had died (within 30 days) of sepsis of different sources.

**Conclusions:** Management of hyponatremia in CKD patients remains challenging and should be directed to the underlying cause. Yet, complex patients with advanced CKD particularly in concurrence with heart failure might represent a medical dilemma. Administering hypertonic saline in different mixed concentrations depending on the volume status of the patients appear to be safe, efficient, and suitable for high-risk CKD patients while monitoring Sodium level carefully.

**Keywords:** Hyponatremia, Chronic kidney disease, Hypertonic saline

## INTRODUCTION

Hyponatremia is a common and important electrolyte imbalance with a prevalence of up to 30% in hospitalized patients. It is defined as a serum sodium concentration of

less than 135 mEq/l.<sup>1</sup> Symptom can range from gastrointestinal manifestations like nausea and malaise, with a mild reduction in the serum sodium, to neurological manifestations like lethargy, headache, confusion, disturbed consciousness, and (if severe) seizures and

coma.<sup>2</sup> Overt neurological symptoms most often associated with very low serum sodium levels (usually <115 mEq/l), resulting in life-threatening cerebral edema. The joint European guidelines classify hyponatremia in adults according to the serum sodium concentration, as follows- mild: 130-134 mmol/l, moderate: 125-129 mmol/l, and profound: <125 mmol/l.<sup>3</sup>

Hyponatremia was found to associate with increased mortality, morbidity, and prolonged hospital stay.<sup>4</sup> Early diagnosis of hyponatremia and appropriate rate of correction was found to reduce morbidity and mortality. An approach towards the management of hyponatremia and formulae for correction have been developed by the early 21<sup>st</sup> century and have been adjusted since then with many online calculators to help in adjusting the rate of correction, taking into consideration other osmotically active electrolytes and underlying comorbidities.<sup>5-7</sup> Hanna et al reviewed the commonly used formulae and online calculators for correction of dysnatremia and found a high variability and differences exist between the predicted and the actual Na levels.<sup>8</sup> The safest rate of correction of hyponatremia in most literature was 4-8 mmol/l per day, with a maximum correction of 10-12 mmol/l in any 24 hours; or 18 mmol/l in any 48 hours.<sup>8</sup> This has been achieved through multiple approaches depending on the underlying diagnosis and the volume status of the patient as sodium abnormalities are notably abnormalities of water (solvent) rather than the sodium (solute). Whether fluid restriction in volume overloaded patients (patients with congestive heart failure, liver cirrhosis with fluid accumulation in the third space, or chronic kidney disease (CKD) with disturbed water excretion) or volume-depleted patients with substantial fluid loss, the correction of hypovolemia/hypervolemia remains the main target of management.

Some resistant cases where fluid restriction or replacement of isotonic fluid alone is not sufficient for proper correction of hyponatremia and alleviation of symptoms, necessitating the need for intravenous of hypertonic sodium chloride solution (5.8% or 3%) in conjunction with loop diuretics if indicated in hypervolemic hyponatremia to promote free water excretion while correcting the sodium deficiency. This should be meticulously followed, as rapid correction of hyponatremia carries a substantial risk of development of osmotic demyelination syndrome (ODS) especially in patients with chronic hyponatremia.

Special consideration appears in Hyponatremia (serum sodium <135 mEq/l) in CKD patients, as it might be associated with either volume overload or volume depletion secondary to diuretics and poor oral intake, making fluids replacement challenging, particularly in concurrence of cardiac diseases with a low ejection fraction (EF) where an infusion of a large amount of fluids are unjustified.<sup>9</sup> Moreover, treatment in cases of dilutional hyponatremia with fluid restriction might be too slow, and infusions of hypertonic saline can reduce patient morbidity and hospital stay. Unfortunately, articles describing the

management of hyponatremia in the background of kidney disease are scarce, hence we hope from this study to elaborate a practical way of managing hyponatremia in high-risk CKD patients.

### ***Aim of the study***

The primary objective is to analyze the incidence, prevalence, patient characteristics, clinical features, and associated morbidity and mortality of individuals presented with profound hyponatremia (serum sodium <125 mEq/l) among hospitalized CKD patients with particular attention to CKD stage 3 to 5. The secondary objective is to imply a practical approach for managing hyponatremia in high-risk CKD patients, particularly in complex patients with advanced CKD in concurrence with heart failure.

## **METHODS**

### ***Settings and design***

This is a retrospective observational study, conducted by the nephrology department at Dubai hospital, one of the tertiary hospitals in Dubai. The study was approved by the scientific research committee of the Dubai Health Authority.

### ***Methods and materials***

All adult patients with CKD stage 3 to 5 on medical therapy, admitted to the general nephrology ward of Dubai hospital at the period of January 2015 till August 2016 with significant (profound) hyponatremia (serum sodium <125 mEq/l) were included. CKD stage-5 patients with hyponatremia and were reluctant for renal replacement therapy (RRT) were enrolled. Our exclusion criteria were hyponatremic patients with normal kidney functions as well as CKD stage 5 patients on maintenance dialysis. Seventy-one adult patients with CKD stage 3 to 5, presented with profound hyponatremia (serum sodium <125 mEq/l), were enrolled in the study and their data were retrospectively collected from health system records using ICD coding system and pharmacy drug codes of hypertonic saline. The following data were collected for each patient; age, gender, race, comorbid diseases (such as diabetes, hypertension, dyslipidemia, ischemic heart disease, and others co-morbid status), etiology of CKD, serum creatinine level/estimated glomerular filtration rate (eGFR) (assessed using CKD epidemiological collaboration (CKD-EPI) method), patients clinical presentation, etiology of hyponatremia, and the length of hospital stay.

Laboratory parameters include the serum sodium level upon presentation (day 1), and the sodium level on the consecutive days (at day 2, 3, and day 4), urinary sodium level and osmolality, treatment received, including the type and volume of intravenous hypertonic saline, diuretic usage, and sodium correction rate in hours were collected.

The clinical endpoint was the development of CNS manifestations, while the secondary outcomes include early mortality (within 30 days). The treatment was given as hypertonic saline (Figure 1), administered as an IV infusion for 24 hours, and to be repeated thereafter as needed. Sodium level was checked at the initial 6, 12 hours, and 24 hours and subsequently every 24 hours till correction of hyponatremia, initiation of dialysis, patient discharge, or mortality had occurred. Patient Data were tabulated, analyzed by the Microsoft statistical package for the social sciences (SPSS)-24 program, using analysis of variance (ANOVA), Mann-Whitney U, and Chi-square tests. A significant value of 0.05, and highly significant if a value is less than 0.001.

**Hypertonic saline protocol in the nephrology department of Dubai hospital**

The nephrology team at Dubai hospital has developed a practical approach for the infusion of hypertonic saline with wider therapeutic safety and ensuring no rapid correction while using high osmolality fluid (saline 5.85%) which requires intensive care unit (ICU) setting in most centers. However, such protocol was used in the general ward with close laboratory monitoring in the view of patient's substantial risk especially with the existence of chronic hyponatremia and impaired water and solute excretion in CKD patients.

Fluids mix and preparation were done in the clinical pharmacy department ahead of infusion and to be used within 24 hours from preparation. Different mixed concentrations of saline infusion were used depending on the volume status of patients and the desired rate of correction. This was calculated as shown in Figure 1.

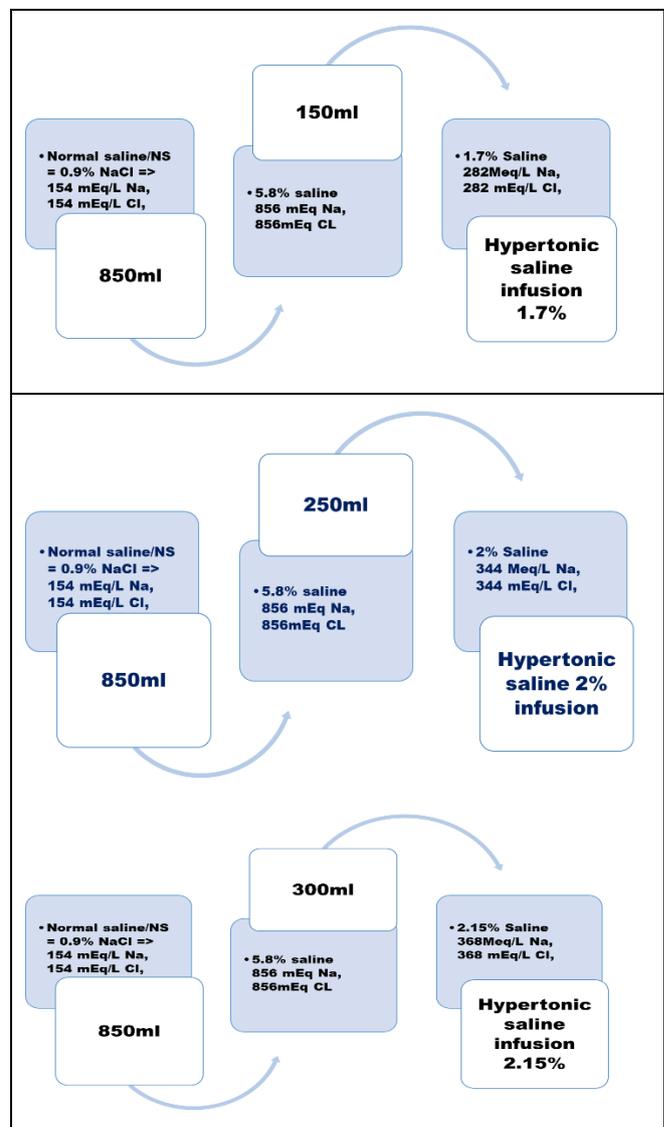
**RESULTS**

As shown in the following Tables 1-4 and Figures 2-6, eventy-one adult patients with CKD stage 3 to 5 (CKD stage 5 who were refusing RRT) and had presented with hyponatremia were enrolled in the study. 97 episodes of hyponatremia were recorded in 71 patients. 35 patients (49%) were UAE national and 38 patients were females (53.5%). The patient's age had ranged between 21 to 102 years (median 64 years). 37 (52%) patients were in CKD stage 5, 22 (30.9%) in CKD stage 4, while 12 had CKD stage 3 (16.6%). Diabetic nephropathy was the cause of CKD in 47 (66%) of the total population, (Figure 2 and 3). 45 of the hyponatremia episodes (42%) were asymptomatic, while in the rest; the symptoms varied between confusion, lethargy, dizziness, weakness, nausea, and vomiting.

Initial sodium level upon admission ranged from 107-125 mEq/l, with a mean±standard deviation (SD) value of 117.7±4.54 mEq/l. Urinary sodium was done in 31 patients and had ranged from <10 to 114 mEq/day with a median of 42, and a mean±SD of 46.5±24.6 mEq/da. The correction of hyponatremia had ranged between 24 hours

in 60% of the patients to 96 hours. In the rest, with a mean of 48±25.3 hours. Diuretics were used in conjunction with the saline in 61 patients (85%).

CNS symptoms at admission were found in 11 patients (15.5%) and disappeared with the correction of hyponatremia. The presence of neurological symptoms at admission was associated with statistically significant lower Serum sodium upon admission (mean±SD of 112.4±3.2 and 120.11± 2.78 mEq/l for patients with and without neurological manifestations on admission respectively). Moreover, the presence of neurological manifestations upon admission was found proportionately the same amid the range of CKD stages. No CNS symptoms were recorded in any of the treated patients, who were on hypertonic saline infusion. On the other hand, Gastrointestinal manifestations at admission were found to be significantly higher in patients with lower initial serum sodium 114±4.8 and 119±3.3 mEq/l respectively p value ≤0.05.



**Figure 1: Dubai Hospital hypertonic saline protocol.**

**Table 1: Patient’s characteristics and demographic data (n=71).**

Number of patients	N (%)
<b>Age</b>	21-102 years (median 65 years)
<b>Gender</b>	Females 38 (53.5)
<b>Etiology of CKD among the study patients</b>	
Diabetes mellitus	47 (66.5)
Chronic glomerulonephritis	4 (5.6)
Hypertension	8 (11.6)
Rejected transplant	7 (9.8)
Others	5 (7)
<b>CKD stage</b>	
CKD stage 3	12 (16.6)
CKD stage 4	22 (30.9)
CKD stage 5	37 (52)
<b>Clinical features in 97 episodes of hyponatremia (in 71 patients)</b>	
Asymptomatic	45 (46.3)
Symptomatic	52 (53.6)
<b>Etiology of hyponatremia</b>	
Diuretics	71.4
Heart failure	12.8
CKD	10
Hypovolemia (other than diuretics)	5.7

**Table 2: Admission symptoms and complications during therapy, per CKD stages.**

CKD stage, admission symptoms and complications during therapy		CKD stage						Chi-square p value
		3.00		4.00		5.00		
		Count	Row (%)	Count	Row (%)	Count	Row (%)	
<b>Admission CNS symptoms</b>	No	5	11.1	13	28.9	27	60.0	0.0841
	Yes	8	30.8	8	30.8	10	38.5	
<b>Admission git symptoms</b>	No	5	11.4	14	31.8	25	56.8	0.154
	Yes	8	29.6	7	25.9	12	44.4	
<b>CNS complications of therapy</b>	No	13	18.3	21	29.6	37	52.1	Non to correlate
<b>Mortality</b>	No	13	20.0	20	29.2	35	50.8	0.827
	Yes	0	0.0	1	33.3	2	66.7	

**Table 3: Sodium levels (initial sodium levels and sodium level following correction), urinary Na, and hospital stay per CKD stage.**

Sodium levels	CKD stage											
	3.00				4.00				5.00			
	Mean	SD	Medi-an	Range	Mean	SD	Medi-an	Range	Mean	SD	Medi-an	Range
<b>Initial sodium</b>	114	5	114	15	118	4	119	13	118	4	118	18
<b>Sodium at 24 hours</b>	121	5	121	16	124	6	123	22	124	5	124	21
<b>Sodium at 48 hours</b>	125	4	124	12	129	5	127	19	129	5	131	20
<b>Sodium at 72 hours</b>	127	2	128	7	132	5	132	20	131	5	131	21
<b>Urinary sodium</b>	43	22	48	52	47	33	38	104	48	17	43	65

Continued.

Sodium levels	CKD stage											
	3.00				4.00				5.00			
	Mean	SD	Medi-an	Range	Mean	SD	Medi-an	Range	Mean	SD	Medi-an	Range
Duration of correction in hours	61	27	72	72	54	27	48	72	41	22	24	72
Hospital stay in days	11	6	11	20	13	15	7	57	11	9	7	39

Table 4: CKD stages, use of hypertonic saline and diuretic use during hospitalization.

Diuretics use	CKD stage						Chi-square p value
	3.00		4.00		5.00		
	Count	Row (%)	Count	Row (%)	Count	Row (%)	
No	3	30.0	3	30.0	4	40.0	0.550
Yes	10	16.4	18	29.5	33	54.1	

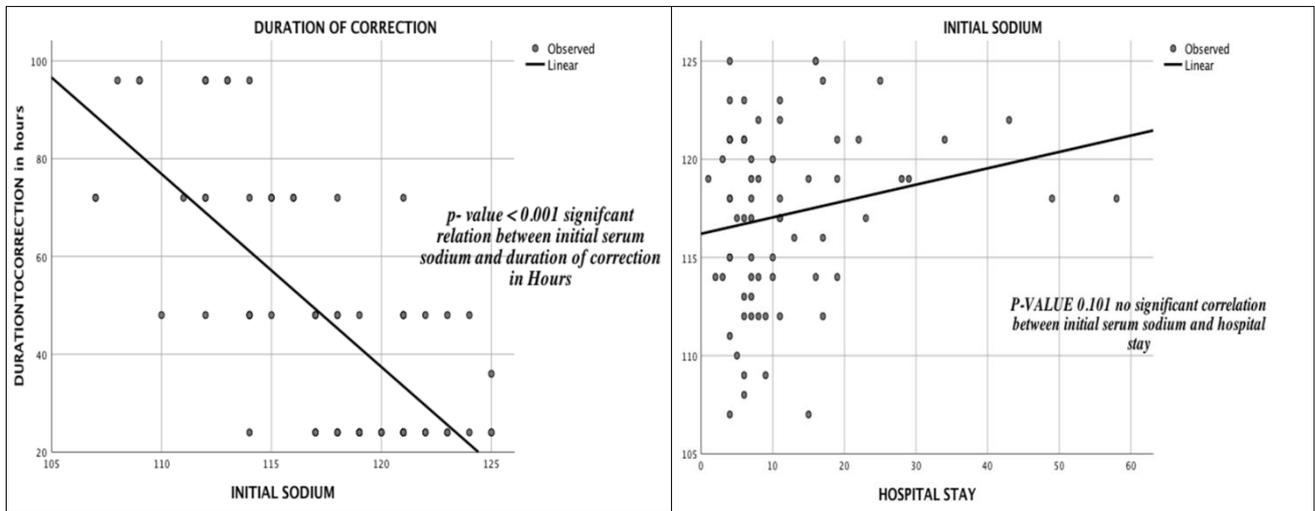


Figure 2: The relation between the initial serum sodium, duration of correction, and the overall hospital stay.

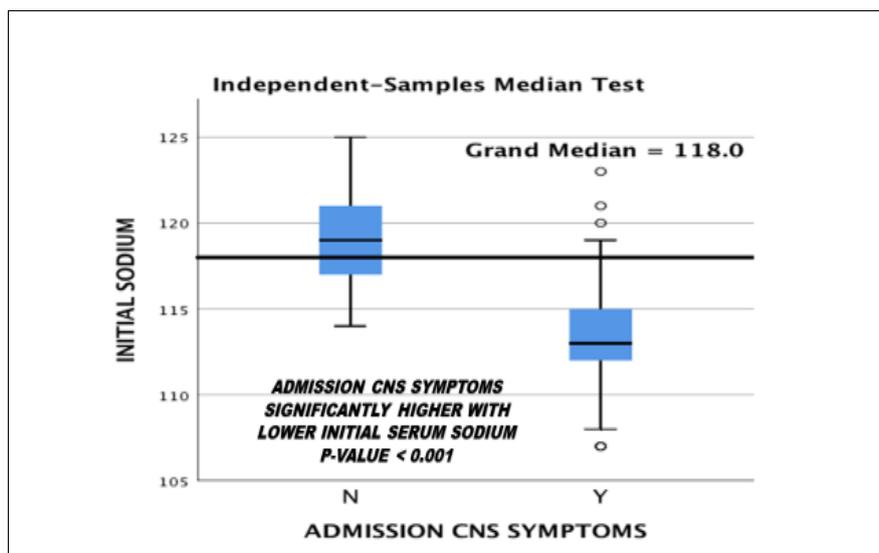
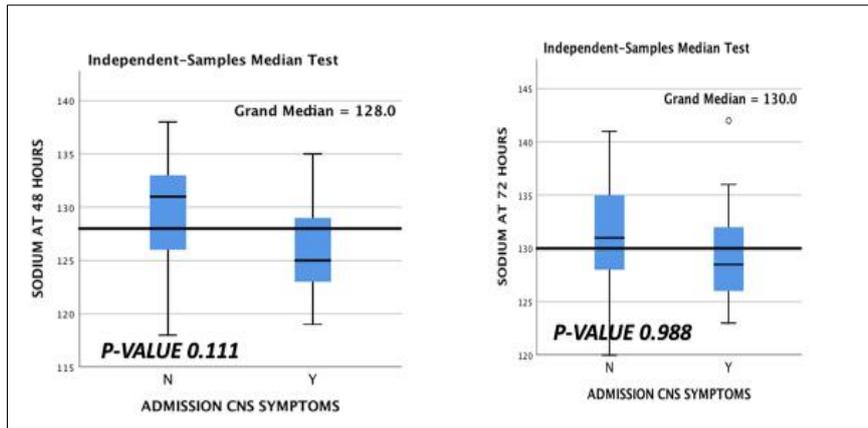
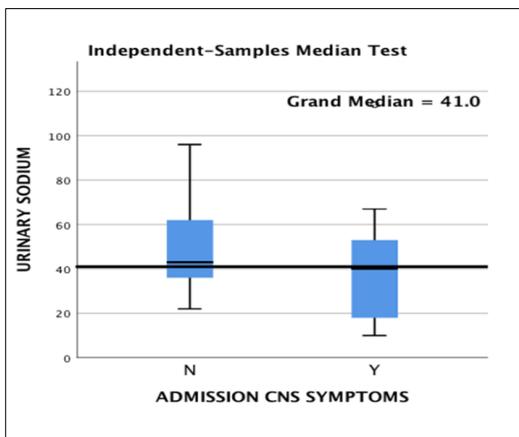


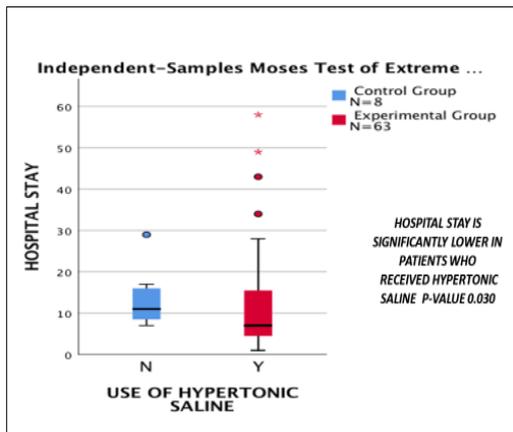
Figure 3: Relation between the CNS manifestations at presentation and initial serum sodium.



**Figure 4: No significant difference of serum sodium correction at 48 and 72 hours concerning CNS symptoms.**



**Figure 5: Relation between urinary sodium and CNS manifestations at presentation shows urinary sodium is significantly lower in patients who presented with CNS manifestations on admission (p value<0.001).**



**Figure 6: Hospital stay and the use of hypertonic saline.**

Haemodialysis was used in 8 patients (11.26%) with CKD-5, who accepted finally the RRT, as a part of treatment in patients with CKD stage 5 with intractable volume overload and dilutional hyponatremia where the infusion

of a hypertonic solution therapy was unjustified. Hospital stay ranged between 2-58 days (median 7 days, average 11.7 days). No significant relation was found concerning the duration of hospitalization and Sodium level upon presentation (p value=0.10), denoting that the diversity of the underlying admission diagnosis has no significant impact on the duration of hospitalization concerning the initial serum NA level. No recorded morbidity from the correction of hyponatremia, however, three patients had died (within 30 days) of sepsis of different sources.

**DISCUSSION**

Hyponatremia is noticed in 15 to 30% of hospitalized patients, especially in the sittings of intensive care units.<sup>10</sup> Correction of hyponatremia must consider the chronicity of the condition. Acute hyponatremia (<48 hours) can be corrected safely and rapidly than chronic hyponatremia. However, rapid correction of serum sodium can precipitate severe neurological sequels.<sup>11</sup> On the other hand; profound hyponatremia (<125 mEq/l) has a higher mortality rate, which can exceed 50% if serum sodium level had fallen further to below 105 mEq/l.<sup>12-15</sup> A study by Huang et al, involved 45,333 patients with CKD stage 3 to 4, revealed that hyponatremia and hypernatremia were associated with an increased risk of all-cause mortality, unrelated to cardiovascular events or malignancy in patients with CKD.<sup>16</sup>

In our analysis for CKD patients, females were more common than males (51%), though it was not statistically significant, and were more symptomatic of hyponatremia than males (23/41(56%) versus 12/39 (30%)). Similarly, a female preponderance was also seen in other studies, while others showed no sexual preference for hyponatremia.<sup>17-19</sup> Grikinienė et al demonstrated a clear-cut predilection for the feminine gender in cases with hyponatremia. Moreover, females are most vulnerable to brain damage from hyponatremia and are more liable to have brain demyelination (ODS), as a result of excessive therapeutic correction of hyponatremia.<sup>20</sup> The authors attributed this to differences in the mechanisms of Na<sup>+</sup> transport (Na<sup>+</sup>/H<sup>+</sup>-exchange, Na<sup>+</sup>/K<sup>+</sup>/2Cl<sup>-</sup> co-transport, Na<sup>+</sup>, K<sup>+</sup>-ATPase).

Diabetic patients were prevalent in our analysis for hyponatremia (66%) compared to others. Lim et al reported 65.5% of his hyponatremic patients to be diabetics, and were related to diuretic use.<sup>9</sup> Diabetics with CKD are prone to hyponatremia for different reasons, such as pseudohyponatremia secondary to overt hyperglycemia/severe hypertriglyceridemia, volume depletion as a result of drug-induced (particularly diuretics), diabetic nephropathy with gross anasarca, causing dilutional hyponatremia, or as an association with the syndrome of hyporeninemic hypoaldosteronism.<sup>21</sup>

The management of hyponatremia in the existence of CKD remains challenging.

Correction of hyponatremia varies according to its source, severity, and its duration. Moreover, the treatment of hyponatremia should be directed at the underlying disorder. Generally, treatment must be calibrated to avoid osmotic demyelination syndrome (ODS), which may result from overly rapid correction.

The fluid restriction might be beneficial in patients with hypervolemia in addition to loop diuretics, and correction of the underlying condition. However, such maneuver can be slow and the patient might be severely symptomatic in the absence of V2 receptor antagonists, hence prescribing hypertonic saline might be necessary in such cases. On the other hand, isotonic saline's can be used safely in hypovolemic patients to replace the contracted intravascular volume, yet complex patients with advanced CKD particularly in concurrence with heart failure, who appear depleted from diuretic use, might not tolerate large volume infusion which may precipitate heart failure, a medical dilemma that can be solved probably through administering low volume of fluid (in the form of hypertonic saline), like the treatment opted in figure-1. Sodium level should be monitored carefully and diuretics can be added thereafter as needed. We do not use hypertonic saline if the sodium level is above 125 mEq/L.

The rate of hyponatremia correction should be parallel to the international guidelines for diagnosis, evaluation, and treatment of hyponatremia (updated in 2013), where a recommendation of minimum correction of serum sodium is 4-8 mmol/l per day, with a lower goal of 4-6 mmol/l per day if the risk of ODS is high, and a maximum correction of 10-12 mmol/l in any 24 hours; 18 mmol/l in any 48 hours for patients at normal risk of ODS to prevent brain herniation and neurological damage from cerebral ischemia.<sup>22</sup>

Similarly, the European society of intensive care medicine, the European renal association, and the European Society of Endocrinology have released guidelines on the diagnosis, classification, and treatment of hyponatremia, that recommend the use of intravenous infusion of hypertonic saline, with a target raise of 6 mmol/l over 24 hours (and not to exceed 12 mmol/L) and an additional 8 mmol/L for every 24 hours thereafter until the patient's

serum sodium concentration reaches 130 mmol/l, for managing serious symptomatic hyponatremia.<sup>4,23</sup>

There is little information about the use and the doses of hypertonic saline in the literature, particularly in the setting of CKD.<sup>24-26</sup> We found our modified formulae of administering hypertonic saline in different mixed concentrations depending on the volume status of the patients and the desired rate of correction to be useful, safe, efficient, and suitable for high-risk CKD patients with no recordable neurological manifestation.

Our study has certain limitations, being a retrospective study; it could not remove any potential bias. Yet; it is the first study of its kind in the local context and has provided valuable evidence regarding the prevalence, patient characteristics, clinical features, and associated morbidity and mortality among hospitalized CKD patients, presented with profound hyponatremia (serum sodium <125 mEq/l) and the treatment provided.

## CONCLUSION

Management of hyponatremia in CKD patients remains challenging and should be directed to the underlying cause. Yet, complex patients with advanced CKD particularly in concurrence of heart diseases might represent a medical dilemma. We hope from this review to imply a practical approach in managing hyponatremia in high-risk CKD patients.

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## REFERENCES

1. Hoorn EJ, Zietse R. Diagnosis and Treatment of Hyponatremia: Compilation of the Guidelines. J Am Soc Nephrol. 2017;28(5):1340-9.
2. Giuliani C, Peri A. Effects of Hyponatremia on the Brain. J Clin Med. 2014;3(4):1163-77.
3. Spasovski G, Vanholder R, Allolio B, Annane D, Ball S, Bichet D, Decaux G, Fenske W, Hoorn EJ, Ichai C, Joannidis M. Clinical practice guideline on diagnosis and treatment of hyponatremia. Nephrol Dialysis Transplant. 2014;29(2):11-39.
4. Peri A, Thompson CJ, Verbalis JG. Disorders of Fluid and Electrolyte Metabolism. Focus on Hyponatremia. Front Horm Res. Basel, Karger. 2019;52:36-48.
5. Hyponatremia Correction Infusate Rate. Available at: <https://www.msdmanuals.com/medical-calculator-for-hyponatremia>. Accessed on 26 October 2020.
6. Adroge HJ, Madias NE. Hyponatremia. N Engl J Med. 2000;342(21):1581-9.
7. Sodium Correction Rate for Hyponatremia. Available at: [https://qxmd.com/calculate/calculator\\_643/sodium-](https://qxmd.com/calculate/calculator_643/sodium-)

- correction-rate-for-hyponatremia. Accessed on 26 November 2020.
8. Hanna RM, Yang WT, Lopez EA, Riad JN, Wilson J. The utility and accuracy of four equations in predicting sodium levels in dysnatremic patients. *Clin Kidney J.* 2016;9(4):530-9.
  9. Lim LM, Tsai NC, Lin MY, Hwang DY, Lin HY, Lee JJ, Hwang SJ, Hung CC, Chen HC. Hyponatremia is Associated with Fluid Imbalance and Adverse Renal Outcome in Chronic Kidney Disease Patients Treated with Diuretics. *Sci Rep.* 2016;6:36817.
  10. Upadhyay A, Jaber BL, Madias NE. Incidence and prevalence of hyponatremia. *Am J Med.* 2006;119(1):30-5.
  11. Sahay M, Sahay R. Hyponatremia: a practical approach. *Indian J Endocrinol Metab.* 2014;18(6):760-71.
  12. Doshi SM, Shah P, Lei X, Lahoti A, Salahudeen AK. Hyponatremia in hospitalized cancer patients and its impact on clinical outcomes. *Am J Kidney Dis.* 2012;59(2):222-8.
  13. Corona G, Giuliani C, Verbalis JG, Forti G, Maggi M, Peri A. Hyponatremia improvement is associated with a reduced risk of mortality: evidence from a meta-analysis. *PLoS One.* 2015;10(4):e0124105.
  14. Dubois GD, Arieff AI. Treatment of hyponatremia: the case for rapid correction. Narins RG, ed. *Controversies in Nephrology and Hypertension.* New York: Churchill Livingstone Inc. 1984;393-407.
  15. Waikar SS, Mount DB, Curhan GC. Mortality after hospitalization with mild, moderate, and severe hyponatremia. *Am J Med.* 2009;122(9):857-65.
  16. Huang H, Jolly SE, Airy M. Associations of dysnatremias with mortality in chronic kidney disease. *Nephrol Dial Transplant.* 2016.
  17. Ahmed SS, Nur F, Ullah MR, Al Mamun A, Chowdhury MT, Laila TR, Khan MA. Factors Influencing Hyponatremia in Hospitalized Diabetic Patients-A Cross Sectional Study. *Medicine Today.* 2014;26(1):4-8.
  18. Rao MY, Sudhir U, Anil Kumar T, Saravanan S, Mahesh E, Punith K. Hospital-based descriptive study of symptomatic hyponatremia in elderly patients. *J Assoc Physicians India.* 2010;58:667-9.
  19. Hawkins RC. Age and gender as risk factors for hyponatremia and hypernatremia. *Clinica Chimica Acta.* 2003;337(1-2):169-72.
  20. Grikinienė J, Volbekas V, Stakišaitis D. Gender differences of sodium metabolism and hyponatremia as an adverse drug effect. *Medicina (Kaunas).* 2004;40(10):935-42.
  21. Liamis G, Tsimihodimos V, Elisaf M. Hyponatremia in diabetes mellitus: clues to diagnosis and treatment. *J Diabetes Metab.* 2015;6(5):559-61.
  22. Soupart A, Decaux G. Therapeutic recommendations for management of severe hyponatremia: current concepts on pathogenesis and prevention of neurologic complications. *Clin Nephrol.* 1996;46(3):149.
  23. Spasovski G, Vanholder R, Allolio B, Annane D, Ball S, Bichet D, et al. Clinical practice guideline on diagnosis and treatment of hyponatraemia. *Intensive Care Med.* 2014;40(3):320-31.
  24. Mohmand HK, Issa D, Ahmad Z, Cappuccio JD, Kouides RW, Sterns RH. Hypertonic saline for hyponatremia: risk of inadvertent overcorrection. *Clin J Am Soc Nephrol.* 2007;2(6):1110-7.
  25. Bhaskar E, Kumar B, Ramalakshmi S. Evaluation of a protocol for hypertonic saline administration in acute euvolemic symptomatic hyponatremia: A prospective observational trial. *Indian J Crit Care Med.* 2010;14(4):170-4.
  26. Assadi F. Hyponatremia: a problem-solving approach to clinical cases. *J Nephrol.* 2012;25(4):473-80.

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