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Beyond Salt Intake

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Abstract

Dietary salt restriction was set forth as main agenda internationally as a tool to face the epidemics of hypertension world-wide, with the intention to reduce complications related to hypertension, including stroke, kidney disease, and heart failure. However, there were controversy in studies in regard to cardiovascular risk and survival with salt restriction. And, iodine must be adequately supplemented, especially for people with limited intake of sea product. Because individual metabolic need, urinary and sweat sodium excretion defer according to body surface area, ideally 24 hour urinary sodium excretion should be normalized accordingly, i.e., mmol/day/m^2 or mmol/day/1.73m^2. Further advice of salt reduction was not necessary for those who are already with limited salt intake, especially if there was also thiazide medication, resulting in risk of hyponatraemia. Conversely, presentation of kidney disease and hypernatraemia should not be simply advised on salt restriction.

Lastly, a case report of a 13-year-old girl presented with hypernatraemia and kidney disease was presented, illustrating a wise search for the underlying cause, by looking towards divine wisdom and intervention is essential. After 5 months of regular haemodialysis, the kidneys recover, yet patient developed unexplained "rebound" hypernatraemia and hyperurcaemia. Crude advice of dietary salt restriction is to no avail, and the clinician has to find the final diagnosis with humble heart and give appropriate treatment.

Review of Salt Intake Issues

Reducing salt intake could bring down blood pressure as shown in a large scale study that was published in 1988 [1], and Blood Pressure (BP) reduction is more marked among subjects with high salt intake with additional advantage of reduction of proteinuria [2]. This paper is with emphasis on dietary salt composed by sodium chloride.

In view of potential to reduce prevalence of non-communicable diseases, mainly hypertension, and its related complications including stroke, heart failure and kidney disease, as featured in the guideline of World Health Organization (WHO) [3], with low cost needed, World Health Assembly has set forth a target of 30% reduction of salt intake by year 2025 [4]. However, there was no yet adequate achievement because majority of the countries were not able to achieve this target [5].

Anyway there were controversy in studies in regard to cardiovascular risk and survival with salt restriction: There was phenomenon of J shaped or U shaped curve in cardiovascular events and total mortality portrayed in some observational studies [6,7] while others show linear relationship, casting doubt to extreme dietary sodium restriction [8]. Therefore, it is recommended that more work needs to be done to explain the discrepancy in results, which could

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be due to heterogeneity across studies, bias, measurement error, or unexplained physiological effects [9].

We should also understand that while limiting salt intake in general population, iodine must be adequately supplemented, especially for people who live in the interior area in the mountain with limited access to sea product; otherwise, there might be risk of iodine deficiency [10].

However, because individual metabolic need were increased with the weight increment, while urinary and sweat (though with minute sodium content) sodium excretion might defer according to body surface area, it would be worthwhile to measure 24 hour urinary sodium excretion with normalized by weight, i.e., mmol/day/kg, or in better assessment, normalized by Body Surface Area (BSA), i.e., mmol/day/m^2 or mmol/day/1.73m^2.

Recently, there was study found higher urinary Na excretion among obese subjects [11], and its relationship with hypertension have also been described in another study in different population [12]. This might support the use of above parameters, and of course, it would possibly be more ideal if the urinary Na excretion is normalized by BSA because, basal metabolic rate was much closer in relationship with BSA than weight in the concept of most literatures.

Although our recent study revealed that proteinuria, the marker for the rate of kidney function deterioration would be reduced among subjects with Chronic Kidney Disease (CKD), yet a long term outcome in term of dialysis and survival should be studied [2].

There was no doubt that hypertension was one of the main causes of chronic kidney disease worldwide, and most clinicians used to advise salt restriction right after diagnosis of chronic kidney disease. Because of current thought of salt reduction would likely preserve kidney function better, many subjects of CKD have been advised on salt reduction. Nevertheless, further advice of salt reduction was really not necessary for those who are already with limited salt intake, and thus ideally a careful clinic interview with history clerking including diet would be essential before clinician engages on such advice. In clinical practice, elderly patients with hypertension who have been in extreme dietary salt restriction might be admitted with extreme lethargy due to hyponatraemia especially if they were also on thiazide diuretic. Nevertheless, conversely, even if patients with kidney disease presented with hypernatraemia, an assumption of too high dietary salt intake should not be simply made; this is because subjects with high salt intake generally should achieve homeostasis of stable sodium level with thirst drive, drinking fluid, and renal retention of water resulting in volume expansion and subsequent likelihood of hypertension, while serum sodium level generally should maintain in normal range.

Finally, the author wishes to report a case of kidney failure presenting with hypernatraemia, in order to illustrate the necessity of wise and deep thought into individual case

Case Report

A 13-year-old girl presented with generalized weakness for a week and fever for 3 days. She was with history of mycobacterium tuberculosis meningitis resulted in hydrocephalus needing ventriculo-peritoneal shunt at the age of 11 year-old and thus with residual complication of right 3rd cranial nerve palsy, limited speech function and all limbs were with feature of upper motor neuron lesion with partial recovery. Her blood test revealed haemoglobin level (Hb) 13.9g/dL, leucocytosis: white blood cell count 19,110/μL and subsequent mild thrombocytopenia, with platelet count dropping from 152,000 to 52,000/μL within a day. Empirical antibiotic treatment was commenced though no pathogen was being identified in serial culture. A diagnosis of anuric acute kidney injury and rhabdomyolisis due to sepsis was made, with extremely high creatinine kinase and hypernatraemia on 16 Nov 2022. Clinicians thus dialyze her with Continuous Renal Replacement Therapy (CRRT) with dilution of NaCl 3% (154 mmol/L) of calculated amount into each 5 litres of prismasol (140 mmol/L), in order to limit changes in serum sodium level and osmolarity, using software invention of the author [13]: https://drive.google.com/drive/folders/1O3DD81gwO_PVQF3moPw6zk-qZXUtds20

Table 1: Serial Investigation and Clinical Management.

		16 Nov 22	17 Nov 22	18 Nov 22	19 Nov 22	9 Jan 23	13 Feb 23	25 Apr 23	19 May 23	17 Jul 23
Creatinine	µmol/L	928	949	534	224	221	150	130	77	67
Na	mmol/L	183	171	160	155	143	137	158	145	129
Urea	mmol/L	32.3	34.1	20.6	9	20.8	23.5	21.3	2.3	4.1
AST	U/L	1,262	1,364	962	637	55	73	73	51	
ALT	U/L	329	318	291	250	72	117	85	54	
Albumin	g/L	38	28	25	22	49	49	48	38	41
LDH	U/L	10,394	9,937	7,270	2,941		346			
Creatine Kinase	U/L	30,849	175,882	116,663	43,150	180	150			
Uric Acid	µmol/L							1080	679	386
Urine osmolarity	mOsm/kg							635	97	403
Clinical Management		Hydration was commenced.	CRRT was initiated.			On-going regular haemodialysis		Withhold HD since 20th Apr. Desmopressin 0.1 mg od was then commenced.	Desmopressin was then up-titrated to 0.1mg bd	Then, desmopressin was reduced to 0.15 mg/day; Patient was encouraged to increased salt intake.

Abbreviations: AST: Aspartate Transaminase; ALT: Alanine Transaminase; LDH: Lactate Dehydrogenase; HD: Haemodialysis; CRRT: Continuous Renal Replacement Therapy

Figure 1: Software of Calculating Regime of Dialysis and/or Intravenous Solute Infusion for Subjects with Hypernatraemia or Hyponatraemia and/or Hyperkalaemia, with or without Risk of Hypoglycaemia, invented by Keng-Hee Koh.
(Clinicians of emergency/general physician/anaesthetist/intensivist discipline could freely download 1empty...xls and utilize it for treatment of critically ill patients with electrolyte imbalance. Dialysis model in software includes haemodialysis or continuous renal replacement therapy or slow efficiency haemodiafiltration, and with concurrent calculated intravenous solute infusion).

This resulted in gradual reduction reduction of serum Na to the safe level with gradualy reduction of serum Na by about 6 mmol/L per day, until normal range, as shown in Table 1. Patient thus recovered fully without untowards neurological complication.

Renal ultrasound showed kidneys of normal renal size, normal echogenicity and normal cortico-medullary differentiation. This signified possibility of acute kidney injury, and thus clinician forsees speedy recovery. Nevertheless, patient remained dependent on dialysis and thus a conversion to regular haemodialysis. And the father being a person believing in The Lord, had come forward to the physician in-charge requesting for prayer for the patient, and thus the physician was fully commited to prayer for her, demanding wisdom from The Lord to search for the underlying cause of kidney failure, and appropriate treatment. Then a miracle occurred, after 5 months, patient's kidney function recovered and did not need dialysis anymore. Subsequently, the patient has unexplained "rebound" hypernatraemia 158 mmol/L upon cessation of haemodialysis with low urine sodium level 10 mmol/L. Taking into consideration of her past baseline urinary sodium concentration was moderate with median of 61 mmol/L before her presentation of acute kidney injury, and patient has difficulty in collecting 24 hour urine sodium due to her disability, an assumption of high salt intake resulting in hypertension and kidney disease was made. A crude advice of low salt diet was given, but it was certainly not the solution for this precious girl with complex scenario.

As the old saying of the wise, "Trust in the Lord with all your heart and lean not on your own understanding; in all your ways submit to Him, and He will make your paths straight." [14] - With the wisdom from The Lord, the physician noticed high uric acid and thus makes a presumptive diagnosis of cranial diabetes insipidus [15], though urine osmolarity test was normal 635 mOsm/kg and a diagnostic serum copeptin test was not available in local setting. Oral desmopressin was prescribed and was adjusted based on her serum [Na] and urine osmolarity test result. Patient was then fully recovering her kidney function and electrolyte stability. On hind sight, the initial presentation of rhabdomyolysis was likely part of the puzzle as complication from diabetes insipidus [16].

This case illustrated a humble heart was greatly needed searching for wisdom from The Lord, in managing patient, and this would then ensure good clinical outcome despite facing desperate critically ill patients even with limited clinical resources. In the midst of uncertainty with complex issue clouding the thought, that high salt intake might elevate blood pressure and potentially increase proteinuria, resulting in renal failure, the simple solution of salt restriction might not be applicable for all patients. Rather than making a fix firm statement in treatment, clinicians should humbly search for guidance from The Lord, looking for His wisdom, sailing through the rough sea to bring patients to a safe strand ahead.

Author Statements

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Conflict of Interest

There was no conflict of interest.

References

1. Intersalt. Intersalt: an international study of electrolyte excre-

tion and blood pressure. Results for 24 hour urinary sodium and potassium excretion. Intersalt cooperative research group. BMJ. 1988; 297: 319-28.

2. Koh K-H, Hii W-SL, Lee J, Ngu L-SL, Tan H-HC. Study of low salt diet in hypertensive patients with chronic kidney disease. Med J Malays. 2018; 73: 399-404.

3. WHO [guideline]. Sodium intake for adults and children. 2012.

4. WHO. Global action plan for the prevention and control of NCDs. 2013-2020.

5. Looi MK. Salt intake: 97% of the world is failing to cut consumption, warns WHO. BMJ. 2023; 380: 570.

6. O'Donnell MJ, Yusuf S, Mente A, Gao P, Mann JF, Teo K, et al. Urinary sodium and potassium excretion and risk of cardiovascular events. JAMA. 2011; 306: 2229-38.

7. Stolarz-Skrzypek K, Kuznetsova T, Thijs L, Tikhonoff V, Seidlerová J, Richart T, et al. Fatal and nonfatal outcomes, incidence of hypertension, and blood pressure changes in relation to urinary sodium excretion. JAMA. 2011; 305: 1777-85.

8. Cook NR, Appel LJ, Whelton PK. Lower levels of sodium intake and reduced cardiovascular risk. Circulation. 2014; 129: 981-9.

9. Cook NR, He FJ, MacGregor GA, Graudal N. Sodium and health-concordance and controversy. BMJ. 2020: m2440.

10. World Health Organization. Salt reduction and iodine fortification strategies in public health. Geneva, Switzerland: World Health Organization; 2014.

11. Mohammadifard N, Haghighatdoost F, Nouri F, Khosravi A, Sarrafzadegan N. Is urinary sodium excretion related to anthropometric indicators of adiposity in adults? J Res Med Sci. 2020; 25: 50.

12. Han W, Wang W, Sun N, Li M, Chen L, Jiang S, et al. Relationship between 24-hour urinary sodium excretion and blood pressure in the adult population in Shandong, China. J Clin Hypertens (Greenwich). 2019; 21: 1370-6.

13. Koh KH. WCN23-0902 Mathematical model for conventional haemodialysis and continuous renal replacement therapy in subjects with hyponatraemia or hypernatraemia with or without hyperkalaemia. Kidney Int Rep. 2023; 8: S333.

14. Proverbs 3: 5-6.

15. Decaux G, Prospert F, Namias B, Soupart A. Hyperuricemia as a clue for central diabetes insipidus (lack of V1 effect) in the differential diagnosis of polydipsia. Am J Med. 1997; 103: 376-82.

16. Kung AW, Pun KK, Lam KS, Yeung RT. Rhabdomyolysis associated with cranial diabetes insipidus. Postgrad Med J. 1991; 67: 912-3.