The Prevalence of High Sodium Intake among Hypertensive Patients at Hypertension Clinic, Siriraj Hospital

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Objective: *To find the prevalence of excessive salt intake among hypertensive patients in hypertension (HT) clinic at Siriraj Hospital. In addition, to correlate the epidemiologic characteristics of the studied group with high salt diet.*
Material and Method: *A cross-sectional study was conducted among 320 hypertensive patients in HT clinic at Siriraj Hospital from September 2010 to January 2011. Epidemiologic data, e.g., age, sex, body mass index, duration of treatment, education level, salary, frequency of salty food consumed/week and clinical data, e.g., renal function, (creatinine clearance, CCr) and anti-hypertensive drug(s) received were collected using pre-defined questionnaires. All volunteers were asked to collect 24-hour urine for 2 days to determine average daily amount of urine sodium (UNa). Those 24-hour UNa $\geq$ 100 mmol/day were considered high salt intake. Correlations of high daily salt intake with all data collected were done.*
Results: *The prevalence of high salt intake was 73.4%. The mean total daily Na intake was 148 mmol/day i.e. 3.4 g of Na/day. When the influence of clinical characteristics on the risks of high salt intake was carried out, there were 2.42, 4.00 and 2.88 fold increases among those who have higher education level, those who have CCr $\geq$ 60 ml/min/1.73 m^2, and those who knew that salt could increase blood pressure (BP), respectively. About three-quarters (76.3%) of those patients who knew the effect of salt on BP consumed high salt diet.*
Conclusion: *The present study revealed that nearly three-quarters of hypertensive patients who attended the HT clinic still consumed high salt diet. Most patients who knew the effect of dietary salt on BP level ignored restriction about salt intake. In addition, those patients with higher education and CCr $\geq$ 60 ml/min/1.73 m^2 also consumed high Na diet.*

Keywords: *High sodium intake, Hypertension*

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High salt intake is associated with the development of hypertension which is one of the most important risk factors for cardiovascular disease, stroke and renal failure[1,2]. A reduction in salt intake has been shown to decrease left ventricular mass in hypertensive individuals[3,4] and contribute to risk reduction in developing cardiovascular diseases[5]. A lower salt intake reduced proteinuria and slowed down the progression of renal disease despite a similar BP control between the 2 groups on a high and low salt intake[6]. High salt intake itself is directly related to the risk of stroke, independent of blood pressure (BP)[7,8]. A decrease in sodium intake will prevent the development of hypertension in normotensive patients and reduce the need for pharmacological therapy in hypertensive patients[9-11]. It is widely acknowledged that reductions in sodium intake are likely to be beneficial in the prevention and treatment of hypertension. Moreover, reducing population of high salt intakes is a highly cost effective strategy for reducing BP levels with huge benefits for public health[12]. At Hypertension clinic, Siriraj Hospital, low salt diet is emphasized both routinely and occasionally to patients by a single physician who took care of them. Since most Thai foods are tasty and also contain a lot of salt, surveillance on the prevalence of high salt intake in hypertensive patients should then give us helpful information to enable us to improve our care. The aims of the present study were to find the prevalence of excessive salt intake (Na > 100 mmol/day) among hypertensive patients who attended hypertension clinic, Siriraj

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Hospital and to correlate the epidemiologic characteristics of those patients consumed high salt diet.

Material and Method

A cross-sectional study was carried out in our routine practice from September 2010 to January 2011. This study was approved by the Ethic Committee in Siriraj Hospital. Hypertensive patients of both sexes, aged >18 years, who attended Hypertension clinic, the Outpatient Department, Siriraj Hospital were recruited. All patients were given usual care which included lifestyle modification advice and antihypertensive drugs by a single physician. The major exclusion criteria were those with chronic kidney disease (serum creatinine $\geq$ 1.5 mg/dL), chronic liver disease (serum alanine aminotransferase > 2x upper normal limit), congestive heart failure, diseases which known to affect on the concentrating ability, i.e., urolithiasis, salt losing nephropathy etc. Patients who were taking nonsteroidal anti-inflammatory drug or diuretics were also not included.

After verbal consents were given, demographic data, e.g., age, sex, weight, height, body mass index (BMI), duration of treatment, education, salary, and clinical data, e.g., creatinine clearance (CCr) and anti-hypertensive drug (s) received were collected using pre-defined questionnaires. A set of questions prepared to identify whether enrolled hypertensive patients had high salt diet, i.e., ate take away food, condiments added to food, ate canned/instant food, consumed seafood which is usually served with hot and ate spicy dip, hot/spicy food, preserved food and food with added sodium bicarbonate or monosodium glutamate was noted as frequencies per week. In addition, conception about salt that could increase BP level and put them on high cardiovascular risk was also asked.

Under daily activity and sweating, approximately 85-90% of ingested sodium is excreted in the urine[13]. Urine sodium (UNa) excretion over 24 hours was accepted therefore as a gold standard method to quantitate daily sodium intake. However, to overcome day-to-day variation in the amount of daily sodium intake, it was measured more accurately by taking the average amount of urine Na collected for 2 consecutive days. Each individual eligible volunteer was given two 5-litre bottles, with a small amount of toluene as preservative added, to collect urine at home and was instructed how to collect 24-hour urine. In brief, each participant was asked to empty his or her urinary bladder at the starting time. Afterward, each amount of urine voided whole day and night had to be collected in the first bottle, including last voided urine, at the same time next day (i.e. 24 hour urine). The same procedure was conducted for another day. In addition, participants were asked to eat and drink as usual.

On the day the enrolled patients returned to submit their 2-day urinary samples, each individual patient was asked to rest in sitting position for 5 minutes before sitting BP was measured with a well calibrated digital automatic BP monitor, OMRON® model: HEM-907. Participants were also examined for weight and height in order to calculate body mass index (BMI). Cigarette smoking and coffee drinking were prohibited for at least 30 minutes before BP measurements.

To determine UNa excretion, each bottle of the 24-hour urine specimen had to be labeled and sent to the central laboratory room within 2 hours. Volume of each of the 24-hour urine sample was determined. UNa was quantified using an Indirect Ion-Selective Electrode method controlled by Biorad Liquichek™ (Urine Chemistry Control), connected to the Roche/Hitachi Modular P800 Analytic system, an automated analyzer (CV = 0.88% for UNa). Each average amount of UNa of > 100 mmol/day was considered a high salt intake. Estimated creatinine clearance (CCr) was determined by using the Cockroft-Gault formula adjusted by body surface area[14,15].

Adequacy of BP control was defined according to the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, the JNC 7 Report[16]. Obesity and overweight were defined according to the proposed classification of weight by BMI in adult Asians by WHO[17].

Statistical analysis

Results were demonstrated as mean $\pm$ standard deviation (SD) or percent (%) where appropriate. Statistical analyses were performed using Statistical Packages for Social Sciences (SPSS 11.5). Independent sample t-test was used to compare the continuous data between 2 variables. Chi-square test and Fisher Exact test were used to compare the categorical data between the high salt intake and other variables. One-sample Kolmogorov-Smirnov test was performed to identify normality or non-normality distribution. Pearson and Spearman correlations of those patients with an average daily amount of UNa excretion with all variables were performed where appropriate. The classification of the magnitude of correlation coefficients was based on Cohen's rule where < 0.3 is considered a low correlation, 0.3 to 0.6

moderate and > 0.6 high[18]. Multiple stepwise logistic regression analyses were performed to identify independent risks. A p-value of less than 0.05 was considered statistically significant.

Results

Clinical characteristics of 320 treated hypertensive patients enrolled in the present study are shown in Table 1. More than half of enrolled patients (59%) were graduates, 48.8% of them were retired or unemployed and 49% had monthly income > 20,000 Baht. Calcium antagonists were used most (81.3%), followed by ACEIs/ARBs (53.4%), beta blockers (47.8%), alpha blocker (12.5%) and others (5.0%).

Enrolled patients had an average age of 61.4 $\pm$ 11.2 years old (range 27-92), duration of hypertension of 5.7 $\pm$ 4.1 years (range 0-21.3), BMI of 25.9 $\pm$ 3.8 kg/m^2 (range 16.6-40.6), SBP of 128.5 $\pm$ 12.2 mmHg (range 97-180), DBP of 72.5 $\pm$ 10.0 (range 48-109), estimated CCr of 65 $\pm$ 39.2 ml/min/1.73 m^2 (range 19-306), and took 2.0 $\pm$ 0.7 items (range 1-5) of antihypertensive drugs. Blood pressures of 257 patients (80.3%) were under control (< 140/90 mmHg). Since the amount of sodium excreted in the urine came from dietary intake, therefore, it reflected the amount of sodium intake. The average amount of daily sodium intake was 148.0 $\pm$ 69.4 mmol (range 38.5-471.5) which was equivalent to a dietary salt (sodium chloride, NaCl) intake of 8.7 $\pm$ 4.1 g/day. The prevalence of high sodium intake was 73.4% according to the criteria mentioned before, 82.1% of men and 65.7% of women. Men consumed much more salt than women i.e. 10.1 $\pm$ 4.7 g/day (equivalent to sodium 172.6 $\pm$ 79.5 mmol/day) and 7.4 $\pm$ 2.9 g/day (equivalent to sodium 125.9 $\pm$ 49.7 mmol/day), respectively. Of those with high sodium intake, 75.3% had 100-200 mmol/day and 24.7% had more than 200 mmol/day of sodium intake.

Table 1. Clinical characteristics of hypertensive patients entered into the study (n = 320)

Parameter	n (%)
Education level	
Primary school or under	65 (20)
High school	67 (21)
University or higher	188 (59)
Occupation	
Retired or unemployed	156 (48.8)
Employee	108 (33.7)
Self-own business	56 (17.5)
Monthly income (Baht)	
< 10,000	89 (27.8)
10,000-20,000	73 (22.8)
> 20,000	158 (49.4)
Type of antihypertensive drug received	
ACEI or ARB	171 (53.4)
Beta blocker	153 (47.8)
Calcium antagonist	260 (81.3)
Alpha blocker	40 (12.5)
Others	16 (5.0)

n = number, ACEI = angiotensin converting enzyme inhibitor, ARB = angiotensin receptor blocker

There were significant low to moderate correlations between the amount of daily UNa excretion and age, diastolic BP levels, BMI, estimated CCr, education level, and monthly income (r = -0.31, 0.26, 0.30, 0.46, 0.32 and 0.26 respectively, p < 0.01 for all). Moreover, a significant low correlation was also found between the amount of the UNa excretion and the number of those with high salt intake behavior collected from the questionnaire (r = 0.11, p = 0.04). However, there were no significant correlations between the amount of the UNa excretion and systolic BP levels, duration of hypertension, and numbers of antihypertensive drugs used (Table 2).

Rate of high salt intake was significantly found in those who were < 60 years old, those of male gender, obese patients, those who had estimated CCr $\geq$ 60 ml/min/1.73 m^2, those who graduated from universities, those who had average monthly income > 20,000 Baht, those who were still at work and even in those who knew the effects of salt on BP (Table 3). About three-quarters (76.3%) of those patients who knew the bad effects of salt on BP level still consumed a high salt diet. Borderline significance was also noted among those who had self-own business (p = 0.05) (data not shown). When kinds of high salt containing food correlated with high salt intake was considered, only sea food was significant (Table 4).

Taken into account the influence of clinical characteristics on the likelihood of excreting > 100 mmol sodium/day in the urine, forward and stepwise multiple logistic regression analyses showed that high salt intake was commonly found among those who were graduates, those who had estimated CCr $\geq$ 60ml/min/1.73 m^2, and those who knew the effect of salt on BP but still consumed a high salt diet (Table 5).

Discussion

Average daily salt consumption among Thai individual had decreased progressively from 20.8 g in 1998 to 10.8 g in 2008-2009[19]. The present study

Table 2. Correlation study between clinical data and the amount of daily UNa excretion (n = 320)

Factor	r-value	p-value
[1]Age (yr.)	-0.31	< 0.01*
[1]Systolic BP (mmHg)	0.04	0.53
[1]Diastolic BP (mmHg)	0.26	< 0.01*
[1]BMI (kg/m^2)	0.30	< 0.01*
[2]Duration of hypertension (yr.)	0.02	0.27
[2]Estimated CCr (ml/min/1.73 m^2)	0.46	< 0.01*
[2]Education level	0.32	< 0.01*
[2]Monthly income (Baht)	0.26	< 0.01*
[2]Numbers of antihypertensive drug used	0.04	0.44
[2]Numbers of high salt intake behavior**	0.11	0.04*

UNa = urine sodium, n = number, BMI = body mass index, CCr = Creatinine clearance, BP = blood pressure, *p-value considered significant at < 0.05, **consumed high salt containing food $\geq$ 3 days/week
[1]Pearson correlation, [2]Spearman correlation

Table 3. Comparative study between clinical data and high salt intake (n = 320)

Factor	Total	High UNa		p-value
		n	%	
Age (yrs)				
< 60	135	109	80.7	0.01*
$\geq$ 60	185	126	68.1	
Sex				
Male	151	124	82.1	< 0.01*
Female	169	111	65.7	
BMI (kg/m^2)				
$\geq$ 25	175	137	78.3	0.03*
< 25	145	98	67.6	
Estimated CCr (ml/min/1.73 m^2)				
< 60	180	112	62.2	< 0.01*
$\geq$ 60	140	123	87.9	
Education level				
$\geq$ University	188	153	81.4	< 0.01*
< University	132	82	62.1	
Monthly income (Baht)				
$\leq$ 20,000	162	104	64.2	< 0.01*
> 20,000	158	131	82.9	
Working status				
No	156	100	64.1	< 0.01*
Yes	164	135	82.3	
Knew that salt could increase BP level				
Yes	266	203	76.3	0.01*
No	54	32	59.3	

UNa = urine sodium, High UNa = UNa excretion $\geq$100 mmol/l, n = number, BMI = body mass index, CCr = Creatinine clearance, BP = blood pressure, *p-value considered significant at < 0.05

observed among hypertensive patients has shown a lower daily salt intake, 8.7 $\pm$ 4.1 g. Salt intake in the present study is generally lower than that of the Thai National Survey and that reported of 10.7 g/day/person

Table 4. The predefined questionnaire that designed to detect the frequency of eating various kinds of high salt containing food in correlation with high salt intake (n = 320)

Predefined questionnaire that designed to detect high salt intake behaviours	day per week	Total	High UNa		p-value
			n	%	
Eating of food that using condiments	< 3	133	97	72.9	0.86
	≥ 3	187	138	73.8	
Eating of instant cooking food	< 3	261	192	73.6	0.92
	≥ 3	59	43	72.9	
Eating of sea food	< 3	223	156	70.0	0.03*
	≥ 3	97	79	81.4	
Eating of hot/spicy food	< 3	182	131	72.0	0.50
	≥ 3	138	104	75.4	
Eating of processed/preserved/salted food	< 3	243	181	74.5	0.45
	≥ 3	77	54	70.1	
Eating of food contained sodium bicarbonate or	< 3	102	77	75.5	0.57
monosodium glutamate	≥ 3	218	158	72.5	

UNa = urine sodium, High UNa = UNa excretion ≥100 mmol/l, n = number, * p-value considered significant at < 0.05

Table 5. The factors in all of significant factors that could predict high salt intake in multiple stepwise logistic regression analyses

Factor (s) (significant)	Univariate analyses			Multivariate analyses		
	CrudeOR	95% CI	p-value	Adjusted	OR 95% CI	p-value
Graduated from university or higher	2.67	1.60-4.43	< 0.01*	2.42	1.37-4.26	< 0.01*
Estimated CCr (≥ 60 ml/min/1.73 m^2)	4.39	2.44-7.92	< 0.01*	4.00	2.11-7.58	< 0.01*
Knew that salt could increase BP level	2.22	1.20-4.09	0.01*	2.88	1.39-5.96	< 0.01*

OR = odd ratio, 95%CI = 95% confidence interval, CCr = Creatinine clearance, BP = blood pressure
*p-value considered significant at < 0.05

from Japan at the same period[20]. This could be due to patient education on the benefit of the low salt intake periodically provided by the physician at the clinic. An average reduction in UNa of 39.1 mmol/day (95% CI: 31.1-47.1) among hypertensive individual was known to be related to SBP reduction by 4.1 mmHg (95%CI: 2.4-5.8) in hypertensive individuals[21]. Moreover, the reduction of sodium intake can lower BP in the setting of antihypertensive medication and facilitate hypertension control[22]. Benefits from salt restriction which led to slight BP reduction were noted and definitely have positive effects on clinical events. Therefore, an average reduction of daily salt intake of 2.1 g which is equivalent to UNa of 35.9 mmol/day in the present study as compared to that of the National Survey should be beneficial to patients in hypertension clinic.

An unpublished pilot study conducted in 2005-2006 among 214 hypertensive individuals attended at the Outpatient Department, Siriraj Hospital found that average salt intake was 9 g/day. Moreover, 71% of them had daily salt intake of > 6 g. A minimal change in salt intake behavior after a 4 year follow-up was rather disappointing. As noted in a previous Cochrane review, the degree of sodium restriction is likely to attenuate over time[23]. In spite of an attempt to reduce salt intake, the minimal diminution of daily salt intake overtime is similar to that found in the epidemiological study from the United Kingdom. After an effort put into a national campaign, initial salt intake was 9.0 g/day/person in

2006 which only reduced to 8.66 g/day/person in 2008[24]. The proportion of English people who consumed salt < 6 g/day was also comparable to ours (26% vs. 26.6%). Notably, the difficulty in cutting down salt intake was accentuated in a report from Australia, since their average daily salt intake had increased from 8.2 g/day in 2006 to 10.9 g/day in 2008[25,26].

High daily salt intake was associated with high diastolic BP level, but not systolic BP level, in this study. Similar findings were also found in the Third National Health and Nutritional Examination Survey (NHANES III)[27]. These findings might imply that lowering salt intake would be associated with lowering diastolic BP level. Non elderly (< 60 years), men and those obese patients (BMI $\geq$ 25 kg/m^2) took more salt as compared to the others. The univariate studies had shown comparable findings to those reported using 24 hours UNa excretion in the analyses[24,28,29]. Those hypertensive patients whose estimated CCr < 60 ml/min/1.73 m^2 paid more attention to minimizing their salt intake. Surprisingly, hypertensive patients who graduated from university were ignorant about salt restriction[29]. Similarly, those who knew the effect of salt on BP still consume a high salt diet. The usual explanations were that Thai people preferred tasty food and food to take away. Fewer people currently prepared food by themselves. However, multivariate analysis confirmed only 2.42, 4.00 and 2.88 fold increases in risk of high salt intake among those who graduated, those who had estimated CCr $\geq$ 60 ml/min/1.73 m^2, and those who knew the effect of salt on BP, respectively.

Most of the salt consumed in Thailand comes from salt added during cooking or from condiments[19]. A public health campaign to take less salt e.g. replacing sodium salt with potassium salt, using fewer condiments, avoiding salt preserved food and instant food, etc., is going on. A modest reduction in salt intake across the hypertensive individuals will result in the improvement in BP control and less use of antihypertensive drugs. Monitoring of 24-hour UNa will be beneficial for certain cases, e.g. those with resistant hypertension and those with high/very high risk of cardiovascular disease. Measurement of 24-hour UNa is an important evidence to convince them, especially university educated patients. Most physicians tend to avoid measuring 24-hour UNa since it is a rather cumbersome procedure. They usually overcome this problem by adding diuretic to treatment regimen. However, this will put patients at risk of the side effects of diuretics. Patient education on taking low salt diet was usually given by verbal with no example of high salt containing food. In addition, those who consumed a high salt diet were not aware that they already did so. Information of the amount of NaCl in various common Thai foods and those branded as fast food should be made available to all hypertensive patients to help them avoid a high salt diet. This concept is likely to work in educated patients.

Conclusion

The present study revealed that nearly three-quarters of hypertensive patients attended at hypertension clinic still consumed high salt diet. More efforts to lower salt intake should be done for those patients with certain epidemiologic characteristics related to high sodium diet. Simple verbal instructions for patients on low salt diet seem to be insufficient in clinical practice.

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Potential conflicts of interest

None.

References

1. Ezzati M, Lopez AD, Rodgers A, Vander HS, Murray CJ. Selected major risk factors and global and regional burden of disease. Lancet 2002; 360: 1347-60.
2. Xie JX, Sasaki S, Joossens JV, Kesteloot H. The relationship between urinary cations obtained from the INTERSALT study and cerebrovascular mortality. J Hum Hypertens 1992; 6: 17-21.
3. Jula AM, Karanko HM. Effects on left ventricular hypertrophy of long-term nonpharmacological treatment with sodium restriction in mild-to-moderate essential hypertension. Circulation 1994; 89: 1023-31.
4. He FJ, MacGregor GA. How far should salt intake be reduced? Hypertension 2003; 42: 1093-9.
5. Strazzullo P, D'Elia L, Kandala NB, Cappuccio FP. Salt intake, stroke, and cardiovascular disease: meta-analysis of prospective studies. BMJ 2009; 339: b4567.
6. Cianciaruso B, Bellizzi V, Minutolo R, Tavera A, Capuano A, Conte G, et al. Salt intake and renal outcome in patients with progressive renal disease. Miner Electrolyte Metab 1998; 24: 296-301.

7. Perry IJ, Beevers DG. Salt intake and stroke: a possible direct effect. J Hum Hypertens 1992; 6: 23-5.
8. Nagata C, Takatsuka N, Shimizu N, Shimizu H. Sodium intake and risk of death from stroke in Japanese men and women. Stroke 2004; 35: 1543-7.
9. MacGregor GA, Markandu ND, Sagnella GA, Singer DR, Cappuccio FP. Double-blind study of three sodium intakes and long-term effects of sodium restriction in essential hypertension. Lancet 1989; 2: 1244-7.
10. Whelton PK, Appel LJ, Espeland MA, Applegate WB, Ettinger WH Jr, Kostis JB, et al. Sodium reduction and weight loss in the treatment of hypertension in older persons: a randomized controlled trial of nonpharmacologic interventions in the elderly (TONE). TONE Collaborative Research Group. JAMA 1998; 279: 839-46.
11. He J, Whelton PK, Appel LJ, Charleston J, Klag MJ. Long-term effects of weight loss and dietary sodium reduction on incidence of hypertension. Hypertension 2000; 35: 544-9.
12. Webster JL, Dunford EK, Hawkes C, Neal BC. Salt reduction initiatives around the world. J Hypertens 2011; 29: 1043-50.
13. Hunter D. Biochemical indicators of dietary intake. In: Willett W, editor. Nutritional epidemiology. 2nd ed. New York: Oxford University Press; 1998: 174-243.
14. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. Nephron 1976; 16: 31-41.
15. Du Bois D, Du Bois EF. A formula to estimate the approximate surface area if height and weight be known. 1916. Nutrition 1989; 5: 303-11.
16. Chobanian AV, Bakris GL, Black HR, Cushman WC, Green LA, Izzo JL Jr, et al. The seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure: the JNC 7 report. JAMA 2003; 289: 2560-72.
17. WHO/IASO/IOTF. The Asia-Pacific perspective: redefining obesity and its treatment. Melbourne: Health Communications Australia; 2000.
18. Cohen J. Statistical power analysis for the behavioral sciences. 2nd ed. Hillsdale, NJ: Lawrence Erlbaum Associates; 1988.
19. World Health Organization. Creating an enabling environment for population-based salt reduction strategies. Report of a joint technical meeting held by WHO and the Food Standard Agency, United Kingdom, July 2010. Geneva: WHO; 2010.
20. Miura K. Epidemiology and prevention of hypertension in Japanese: how could Japan get longevity? EPMA J 2011; 2: 59-64.
21. Taylor RS, Ashton KE, Moxham T, Hooper L, Ebrahim S. Reduced dietary salt for the prevention of cardiovascular disease: a meta-analysis of randomized controlled trials (Cochrane review). Am J Hypertens 2011; 24: 843-53.
22. Appel LJ, Espeland MA, Easter L, Wilson AC, Folmar S, Lacy CR. Effects of reduced sodium intake on hypertension control in older individuals: results from the Trial of Nonpharmacologic Interventions in the Elderly (TONE). Arch Intern Med 2001; 161: 685-93.
23. Hooper L, Bartlett C, Davey SG, Ebrahim S. Advice to reduce dietary salt for prevention of cardiovascular disease. Cochrane Database Syst Rev 2004; (1): CD003656.
24. National Centre for Social Research and Medical Research Council Human Nutrition Research. An assessment of dietary sodium levels among adults (aged 19-64) in the UK general population in 2008, based on analysis of dietary sodium in 24 hour urine samples. London, UK: National Centre for Social Research; 2008.
25. Keogh JB, Clifton PM. Salt intake and health in the Australian population. Med J Aust 2008; 189: 526.
26. Margerison C, Nowson C. Dietary intake and 24-hour excretion of sodium and potassium. Asia Pac J Clin Nutr 2006; 15 (Suppl): S37.
27. Cohen HW, Hailpern SM, Alderman MH. Sodium intake and mortality follow-up in the Third National Health and Nutrition Examination Survey (NHANES III). J Gen Intern Med 2008; 23: 1297-302.
28. Anderson CA, Appel LJ, Okuda N, Brown IJ, Chan Q, Zhao L, et al. Dietary sources of sodium in China, Japan, the United Kingdom, and the United States, women and men aged 40 to 59 years: the INTERMAP study. J Am Diet Assoc 2010; 110: 736-45.
29. Venezia A, Barba G, Russo O, Capasso C, De Luca V, Farinaro E, et al. Dietary sodium intake in a sample of adult male population in southern Italy: results of the Olivetti Heart Study. Eur J Clin Nutr 2010; 64: 518-24.

การศึกษาความชุกของการรับประทานเค็มในผู้ป่วยความดันโลหิตสูง ที่คลินิกความดันโลหิตสูง โรงพยาบาลศิริราช

พีระ บูรณะกิจเจริญ, เมธา ผู้เจริญชนะชัย

วัตถุประสงค์: *เพื่อประเมินความชุกของการรับประทานอาหารเค็มในผู้ป่วยความดันโลหิตสูงที่คลินิกความดันโลหิตสูง โรงพยาบาลศิริราช ในขณะเดียวกันได้หาความสัมพันธ์ของลักษณะทางระบาดวิทยาของประชากรที่ศึกษาที่รับประทานเค็ม*

วัสดุและวิธีการ: *เป็นการศึกษาแบบภาคตัดขวาง ในผู้ป่วยความดันโลหิตสูง 320 ราย ที่คลินิกความดันโลหิตสูง โรงพยาบาลศิริราช ตั้งแต่เดือน กันยายน พ.ศ. 2553 ถึง มกราคม พ.ศ. 2554 โดยการเก็บข้อมูลทางระบาดวิทยาเช่น อายุ, เพศ, ดัชนีมวลกาย, ระยะเวลาที่ได้รับการรักษา, ระดับการศึกษา, อาชีพ, รายได้ต่อเดือน ความถี่ของการรับประทานอาหารเค็มต่อสัปดาห์โดยใช้แบบสอบถามที่เตรียมไว้ล่วงหน้า และข้อมูลทางคลินิก เช่น การทำงานของไต (อัตราการขับครีอะตินิน) ยาลดความดันโลหิตที่ได้รับ อาสาสมัครทุกรายจะต้องเก็บปัสสาวะ 24 ชั่วโมงเป็นเวลา 2 วัน เพื่อหาค่าเฉลี่ยปริมาณโซเดียมในปัสสาวะต่อวัน ปริมาณโซเดียมในปัสสาวะต่อวันมากกว่าหรือเท่ากับ 100 มิลลิโมลต่อวัน จัดว่ารับประทานเค็ม ศึกษาหาความสัมพันธ์ของการรับประทานเค็มกับข้อมูลที่เก็บรวบรวมจากแบบสอบถาม*

ผลการศึกษา: *พบว่าผู้ป่วยที่ รับประทานอาหารเค็มร้อยละ 73.4 ค่าเฉลี่ยของปริมาณโซเดียมในปัสสาวะ 148 มิลลิโมลต่อวัน ซึ่งเท่ากับ ปริมาณโซเดียมที่รับประทาน 3.4 กรัมต่อวัน เมื่อศึกษาบทบาทของลักษณะทางคลินิกต่อการเพิ่มความเสี่ยงของการรับประทานเค็มพบ 2.42, 4.00 และ 2.88 เท่าในกลุ่มผู้ป่วยที่มีการศึกษาสูง, ผู้ป่วยที่มีอัตราการขับครีอะตินินที่ไตมากกว่าหรือเท่ากับ 60 มิลลิลิตรต่อนาทีต่อพื้นที่ผิวกาย 1.73 ตารางเมตร และผู้ป่วยที่ทราบว่าเกลือมีผลต่อการเพิ่มความดันโลหิตตามลำดับ ประมาณ 3 ใน 4 (ร้อยละ 76.3) ของผู้ป่วยทราบว่าเกลือมีผลต่อความดันโลหิต ยังรับประทานอาหารเค็ม*

สรุป: *การศึกษานี้พบว่าเกือบ 3 ใน 4 ของผู้ป่วยความดันโลหิตสูงที่รักษาในคลินิกความดันโลหิตสูงยังรับประทานเค็ม ผู้ป่วยส่วนมากทราบว่าเกลือมีผลต่อระดับความดันโลหิตยังละเลยการควบคุมอาหารเค็ม รวมทั้งผู้ป่วยกลุ่มที่มีการศึกษาสูงและผู้ป่วยที่มีอัตราการขับสารครีอะตินินที่ไตมากกว่าหรือเท่ากับ 60 มิลลิลิตรต่อนาทีต่อพื้นที่ผิวกาย 1.73 ตารางเมตรก็เป็นกลุ่มที่รับประทานอาหารเค็ม*