Salt Intake and Hypertension: Walking the Tight Rope

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Hypertension, a disease often correlated to affluence, is becoming increasingly common in urban population in developing countries. Recent epidemiological studies in India show that prevalence was 1.2 to 4.2% in 1940’s and has remarkably increased to nearly 15 to 25% in 1990’s in urban areas. A recent study by Mohan et al reports a prevalence of 20% in Chennai in the CURES study. Rural areas also showed a similar increase in prevalence of hypertension (~5-10%) although the rise was not as steep as in urban areas.

The etiology of hypertension is considered to be multifactorial which includes genetic, dietary, racial, metabolic and psychological factors, often in combination. Hypertension is considered to be etiologically related to obesity and insulin resistance and is a component of the metabolic syndrome. A genetic role is implicated as hypertension is seen to be about twice as common in subjects who have one or both hypertensive parents. Perinatal influences may also play a role. Low birth weight and catch-up obesity seems to adversely affect blood pressure in adult life. Further, low grade inflammation manifesting as raised C-reactive protein levels may also be a novel correlate of hypertension.

Salt or body sodium level is considered to be one of the important contributing factors in the pathogenesis of hypertension. Going as far back as 2,000 B.C., the famous Chinese “Yellow Emperor” Huang Ti demonstrated the association of salt with a “hardened pulse”. Since then, evidence from many population-based studies have demonstrated a positive association. The “Dietary Approaches to Stop Hypertension (DASH) study” demonstrated a direct relationship between salt intake and blood pressure. The subjects put on DASH diet, containing high amount of fiber, vegetables and fruits, with low fat dairy products and low salt processed foods experienced a reduction of blood pressure by 11/6 mm Hg. In this study, diets at three sodium levels were compared: high (3,300 mg), intermediate (2,400 mg) and low (1,500 mg). The difference in blood pressure in those taking lowest sodium-containing diet to highest sodium-containing diets was 8.3/4.4 mm Hg in patients with hypertension and by 5.6/2.8 mm Hg in normotensive people regardless of race and sex. In the INTERSALT study (a multinational study of more than 10,000 persons in 52 centers in 32 countries including India), a direct relationship between average sodium intake in a population and increase in degree of blood pressure with aging was seen. The Multiple Risk Factor Intervention Trial (MRFIT) showed that the mortality rates from cardiovascular disease (CVD) were greater in the patients with higher quintiles of sodium intake. In Trial of non-pharmacologic Interventions in the Elderly (TONE), obese persons randomized to sodium reduction showed an effective reduction of blood pressure which was comparable to that seen with weight loss. Overall, the evidence was strong that restriction of salt consumption is an effective measure of primary prevention of hypertension.

The general perception is that a high salt concentration in the body is a predisposing factor to hypertension and increases cardiovascular risk, and a low salt intake would be beneficial. However, it is still considered debatable whether lowering the salt intake is an important strategy in the treatment of hypertension.

First, salt restriction produces only small, even though detectable changes in blood pressure. Meta-analysis of randomized trials of sodium restriction in subjects with hypertension has shown a blood pressure lowering of 3-6 mm Hg systolic and 1-3 mm Hg diastolic for a decrease of 2.3 gm in sodium intake. However, it may still turn out to be significant as even small reduction in blood pressure may lead to substantial reduction in mortality. A reduction of 2 mm Hg in systolic blood pressure is expected to reduce mortality from stroke by 6%, coronary heart disease by 4%, and all cause mortality by 3%.

Second, the effects of salt depletion in hypertension are restricted to subjects with a salt-sensitive phenotype. Ducher et al reported that only a limited proportion of individuals (5 to 16%), classified as “salt-dependent” would benefit from dietary salt restriction. It has been also argued that factors other than sodium intake may be important environmental and behavioral determinants of blood pressure. In a counterpoint to the observations of INTERSALT study, it was stressed that salt intakes neither very high nor too low (despite being freely accessible) was significantly correlated with hypertension in 48/52 sites of the study. On re-analysis of NHANES I data, Alderman et al reported...
heterogeneity of blood pressure response to sodium restriction, and opined that obesity was a stronger correlate than salt intake in relation to hypertension.

It has been argued that body has adaptive mechanisms that make physiological adjustments to changes in sodium levels in the blood e.g. through renin-angiotensin system. Interestingly, low salt intake could be linked to higher incidence of adverse coronary events as reduction of sodium intake increases plasma renin activity and aldosterone activity. Further, it has also been shown that sodium restriction stimulates the sympathetic nervous system. All these processes singly or in combination may cause tissue hypoperfusion, vasoconstriction or insulin resistance leading to adverse cardiovascular outcomes. Alderman et al showed that patients with lowest quartile of measured sodium excretion had a highest incidence of myocardial infarction and total cardiovascular events as compared to highest quartile of salt intake. Importantly low salt intake was related to high all-cause morbidity. The findings of the study were, however, contentious as the patients with lower salt intake were older and had preexisting CVD. Finally, low plasma sodium concentrations have been also seen to be associated with chronic heart failure. The data, therefore, point out to adverse cardiovascular outcomes of both high and too low dietary salt intake. Lowering sodium intake decreases blood pressure and there is a deemed necessity of salt restriction and intake, even though one has to walk a tight rope as too low salt intake may also lead to adverse cardiovascular outcomes.

Dietary habits of Asian Indians indicate that the salt intake in them may be higher than some other populations. Excess salt intake occurs due to intake of pickles, papads, salty snacks, and chutneys which are popular household dietary choices, in addition to increasing consumption of salted potato chips by children. The mean daily salt intake of salted tea drinking people residing in Jammu and Kashmir was shown to be 3.5 g, and daily salt intake was significantly higher in hypertensives than that of normotensive subjects (mean 3.9 g vs. 3.3 g, respectively; p <0.001), with intake of salt tea showing a correlation to hypertension. A higher salt intake was also reported in the children of hypertensive families as compared to normotensive families in Kashmir. Salt consumption was found as a significant predictor of hypertension even in a rural community in north India. Increased mean arterial pressure was seen to correlate with increased sodium consumption, in both urban as well as rural tribals, and hypertensives from both the groups showed higher urinary sodium excretion. Despite these anecdotal data, average salt intake of people living in different regions of India remain poorly investigated.

In this issue of the Journal, Radhika et al in a follow up report of the recent paper by Mohan et al report that the mean dietary salt intake was 8.5 g/day in urban people residing in Chennai, which was higher than recommended by World Health Organization (5 gm/day). The authors have further reported that higher salt intake was associated with older age and higher socio-economic status, possibly due to an increased intake of ‘westernized foods’ high in salt content. Subjects in the highest quintile of salt intake had significantly higher prevalence of hypertension than did those in the lowest quintile. An odds ratio of 1.16 was reported in subjects showing positive associations of salt intake with hypertension, even after adjusting for multiple factors.

What are the implications of this well-conducted study? First, we now know that Asian Indians in South India have high average salt intake. Importantly, this fact alone would be vital for primary prevention of hypertension in this population, due to the high prevalence of stroke and CVD in South India. Second, the diet intake in this population needs further scrutiny, given that the prevalence rates of the metabolic syndrome and type 2 diabetes are also high. Clearly, these observations also stress that key to primary prevention of several non-communicable diseases in Asian Indians lies in dietary modulation. Given the projected high incidence and prevalence of CVD in India, the findings of this study assume importance for formulation of primary prevention strategies and National CVD Control Program. Finally, although we could roughly project these data to any urban area in India, documentation of salt intake is necessary in populations residing in different agro-climatic zones of India which may help to prepare location-specific future guidelines for salt intake.

**REFERENCES**


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