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ORIGINAL ARTICLE

Excess dietary salt is associated with an altered bone strain index, degraded bone microarchitecture, vertebral fractures, and increased prevalence of osteoporosis in postmenopausal women—A study from a teaching hospital in southern India

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Abstract

Objectives: Excess dietary salt causes increased urinary calcium and this may lead to bone loss. We proposed to study the association between dietary salt intake and bone health in postmenopausal women from southern India.

Methods: An observational study in which community-dwelling postmenopausal women were recruited. Daily salt intake and urine calcium/creatinine ratio were assessed. Bone biochemistry and densitometric parameters such as bone mineral density (BMD), trabecular bone score (TBS) vertebral fractures, and bone strain index (BSI) were assessed using Dual Energy X-Ray Absorptiometry (DXA).

Results: A total of 383 postmenopausal women with a mean ± SD age of 59.8 ± 7.2 years and BMI of 25.2 ± 4.6 kg/m^2 were recruited. Among the participants, 165/383(43.1%) had osteoporosis at any site and 21% had moderate–severe vertebral fractures. The BMD at lumbar spine and femoral neck, TBS and BSI were significantly ($p < 0.001$) lower and the CTx was significantly ($p = 0.008$) higher among women with high salt intake (7.2 g/day) as compared to those with salt intake of <7.2 g/day. The prevalence of osteoporosis, low TBS, high BSI, and moderate–severe vertebral fractures significantly increased across low to high salt-intake categories. An ROC analysis showed that excess dietary salt was significantly associated with osteoporosis at any site with an AUC of 0.870 (95% CI: 0.832–0.907). On a multivariate analysis, excess salt intake conferred the highest odds of osteoporosis (OR: 2.296; 95% CI: 1.909–2.761).

Conclusions: Excess dietary salt is associated with high urinary calcium and compromised bone health among postmenopausal women from southern India. This may be a modifiable risk factor in osteoporosis and warrants further research.

KEYWORDS
bone strain index, dietary salt, India, osteoporosis, postmenopausal women, trabecular bone score, urine calcium

1 | INTRODUCTION

Osteoporosis is one of the most common bone mineral diseases, and is characterized by decreased bone mass and deterioration of bone microarchitecture.[1] Osteoporosis-related fractures contribute to poor quality of life and to significant morbidity, in the aging population.[2] The mortality rate in the first year following a fragility fracture of the hip can be as high as 20%.[3] Moreover, the quality of life in women with decreased bone mineral density (BMD) was detected to be much lower than their counterparts with normal BMD.[4] The cost-effectiveness of fracture prevention measures has been reasonably proven.[5]

Despite being a common disease, osteoporosis continues to be under-recognized and under-reported. The worldwide prevalence of osteoporosis is about 18.3%, with 23.1% among women and 11.7% among men.[6] It has been extrapolated that one in three women and one in five men above 50 years of age will develop osteoporosis-related fractures in their remaining lifetime.[7] In India, available data show the prevalence of osteoporosis to be about 50% and of prevalent moderate–severe vertebral fractures about 30%.[8]

The gold-standard in diagnosing osteoporosis is the assessment of BMD by the Dual Energy X-ray Absorptiometry (DXA) scan. Other densitometric adjuncts that have been utilized include the Trabecular Bone Score (TBS)[9] which furnishes information about the trabecular microarchitecture of the lumbar spine and the hip structural analysis, which describes characteristics of the proximal hip geometry.[10] Bone Strain Index (BSI) is a newly developed DXA-based tool that provides information of the skeletal resistance to loads, not addressed by the other measures. This has been calculated using the Finite Element Analysis (FEA) on a grayscale of the distribution of bone density measured on both the spine and femoral scans.[11] A high BSI indicates a higher strain in the bone and a reduced resistance to fractures. Various studies have assessed the utility of the BSI in the prediction of fragility fractures; these have shown promising results.[12,13]

Our knowledge regarding risk factors of poor bone health is still incomplete. Identification of new modifiable risk factors is crucial to prevent osteoporosis. Dietary salt has long been postulated as a risk factor for osteoporosis through increasing the urinary calcium excretion, as well as by increasing the extracellular fluid volume and increasing the glomerular filtration rate.[2] Various studies have assessed the possible role of dietary salt leading to bone fragility.[14,15] Carbone et al. showed that the population-based recommendations for sodium intake was unlikely to affect osteoporosis.[14] A preclinical study by Cui et al. on ovariectomized rats showed that a high salt intake can cause excess bone resorption and disrupted microarchitecture.[16] Hong et al. showed that a low sodium intake was associated with osteoporosis.[17] Tiyasatkulkovit et al. showed that a long-term excessive salt consumption accelerated bone loss in rats.[18] Kim et al. showed that among 3635 postmenopausal women, a higher sodium intake was associated with a higher odds of osteoporosis prevalence in Korea.[19] Thus, these results obtained have been conflicting. Hence, this study was aimed to study the association between dietary salt and bone health in postmenopausal women from southern India.

2 | METHODOLOGY

2.1 | Study subjects

The study population was recruited from two villages of Vellore district, Tamil Nadu. Postmenopausal women above the age of 50 years who were living in rural communities between January 2020 and March 2022 were identified prospectively and candidates were selected through a simple random sampling method. The previous medical history and present physical conditions of the selected women were evaluated. Height and weight were assessed by standard methods.

2.2 | Eligibility

Those with underlying rheumatological disorders, malignancies, chronic kidney disease stages 4 and 5, liver disease, those on medications that could adversely affect bone health and those already on treatment for osteoporosis were excluded from the study. Women on drugs that could affect sodium levels such as diuretics and NSAIDs were also excluded from the study. The study was approved by the institutional review board and ethics committee (15843). A written informed consent of the participants was obtained prior to their inclusion in the study. Overall, about 580 women were identified of which 383 were included based on a convenient sample size after applying the eligibility criteria.

2.3 | Assessment

A detailed check on the dietary salt intake of the subjects was carried out through two 24-h dietary recall and a food frequency questionnaire on non-consecutive days. These were based on guidelines pertaining to the nutritive value of foods from the National Institute of Nutrition salt content and was documented on a predesigned proforma.[20] A previous study has suggested that the food record checklist may be a practical instrument for sodium intake.[21] Two spot samples of urinary calcium creatinine ratio (collected as a fasting second-void morning sample), as well as biochemical evaluation of bone mineral parameters, bone turnover markers, and DXA scan was carried out on the selected subjects. Hypercalciuria was defined as a urine calcium–creatinine ratio of more than 0.20. Fasting blood samples were assessed for calcium, phosphate, albumin, alkaline phosphatase, creatinine, 25(OH) vitamin D, and parathyroid hormone (PTH). Samples were also evaluated for procollagen-1 N terminal peptide (P1NP) and C-terminal telopeptide of type 1 collagen (CTX). Among the bone biochemical parameters, colorimetric methods were used for calcium; phosphorus was estimated by the phosphomolybdate method. PTH was measured by chemiluminescent immunoassay (CLIA), 25 hydroxy vitamin D was analyzed using electro-chemiluminescent assay (ECLIA) and alkaline phosphatase was measured by the kinetic PNPP (paranitrophenyl phosphate) method. CTX and P1NP

were measured using ECLIA. Serum creatinine was measured using the modified Jaffe's method.

Dual-energy X-ray absorptiometry (DXA), done using the Hologic Horizon scanner, was utilized to assess BMD at the neck of femur (NOF) and lumbar spine (L1–L4) in antero-posterior projection in accordance with the guidelines of the International Society for Clinical Densitometry (ISCD)[22] TBS and BSI was also determined simultaneously with assessment of BMD. TBS, which assesses the trabecular microarchitecture of the lumbar spine, was assessed using iNsight Software version 3 (Med-Imaps, Bordeaux, France). BSI is a newly developed DXA-based tool that provides information of the skeletal resistance to loads, not addressed by the other measures. This has been calculated using FEA on a grayscale of the distribution of bone density measured on both the spine and femoral scans.[11] BSI was assessed using the Bone Strain Skeletal research software version 1.0.0 (Tecnologie Avanzate s.r.l., Torino, Italy).

Vertebral fractures were assessed in the thoracic and the lumbar vertebrae using the vertebral fracture assessment (VFA) tool of the DXA scanner. The severity of vertebral fractures was graded based on height loss of the vertebral bodies in keeping with the Genant's system as follows: mild: <25% height loss; moderate: 25%–40%; severe: >40%.[23] Moderate–severe fractures (vertebral fractures) were considered significant and were included in the analysis. For the DXA derived parameters, a BMD T −score ≤ −2.5 at the NOF or L1-L4 were used for defining osteoporosis. The reference BMD used was that of Caucasian women from the NHANES database both for the hip as well as the lumbar spine. A TBS of <1.200 was considered as degraded bone microarchitecture.[9] A BSI that was ≥2.4 was considered to portend an increased strain in the bone with a reduced resistance to fractures.[24]

2.4 | Statistical analysis

Continuous variables were expressed as mean and standard deviations, while categorical variables were expressed as frequencies and percentages. Statistical significance for continuous variables was calculated using Student's *t*-test for normally distributed variables and the Mann–Whitney *U*-test for non-normally distributed variables. Differences in proportions were analyzed using the chi-squared test. Pearson's correlation coefficient was employed for assessing correlation. Logistic regression as well as ROC curve analysis was also carried out. SPSS Statistics 21.0 software was used for all data analysis. A two-tailed *p*-value <0.05 was considered significant for all comparisons.

3 | RESULTS

3.1 | Characteristics of study population

The mean ± SD age and BMI of our study group was 59.8 ± 7.2 years (Range: 50–79 years) and 25.2 ± 4.6 kg/m^2 respectively. The mean ± SD age of menopause was 45.9 ± 5.2 years. The average salt intake was 7.2 ± 2.2 g/day. Urine sodium was available in 15 participants and this showed a positive correlation with the dietary salt intake ($r = 0.3$). The prevalence of osteoporosis at either the neck of the femur or lumbar spine was 43.1%. Vertebral fractures were seen in 21% of the study population. The mean value of serum albumin adjusted calcium, phosphate, alkaline phosphatase, PTH, and bone turnover markers were all within the normal range except for 25(OH) vitamin D which was 24.9 ± 10.0 ng/mL (insufficient). The percentage of population with vitamin D deficiency <20 ng/mL was 32% and that with severe vitamin D deficiency (<12 ng/mL) was 6.8%. In the study population, 13% had hypertension and 8% had type 2 diabetes mellitus. The patient characteristics are presented in Table 1.

3.2 | Comparison of bone biochemistry between low- and high-salt intake groups

The study population was subsequently categorized into those with low-salt intake (salt intake <7.2 g/day) and those with high-salt intake (salt intake ≥7.2 g/day) based on the mean salt intake of 7.2 g in our population. The high-salt intake group had a higher aged population and low BMI compared to the low-salt intake group. There was a considerably higher level of CTx as well as urinary calcium creatinine ratio in the high-salt intake group which attained statistical significance. No statistical difference with respect to other biochemical parameters including serum calcium, serum 25(OH) vitamin D, and

TABLE 1 Characteristics of study population.

Parameter	Normal ranges	Mean ± SD
Age (years)		59.8 ± 7.2
BMI (kg/m^2)		25.2 ± 4.6
Parity		2.8 ± 1.3
Age of menopause		45.9 ± 5.2
Daily salt intake (g)		7.2 ± 2.2
Daily calcium intake (mg)		464.4 ± 149.5
Physical activity (METS/week) Median (IQR)		1642 (460–1680)
Albumin corrected calcium (mg/dL)	8.3–10.4	9.2 ± 0.3
Phosphate (mg/dL)	2.5–4.5	4.1 ± 0.5
Creatinine (mg/dL) eGFR (mL/min/1.73m^2)	0.5–1.2	0.7 ± 0.1 94.4
Alkaline phosphatase (U/L)	40–125	95.5 ± 23.2
Parathormone (pg/mL)	8–80	71.2 ± 32.3
25(OH) vitamin D (ng/mL)	30–75	24.9 ± 10.0
CTx (pg/mL)	226–1088	687.9 ± 310.9
P1NP (ng/mL)	16.0–73.9	65.4 ± 31.7

Abbreviations: BMI, body mass index; Ctx, C-terminal telopeptide of type 1 collagen; eGFR, estimated glomerular filtration rate; METS, metabolic equivalents; P1NP, N-terminal telopeptide of type 1 procollagen.

PTH levels were noted between two groups. The particulars of both groups are presented in Table 2.

3.3 | Bone mineral density, trabecular bone score, vertebral fractures, and bone strain index

Assessment of BMD at the NOF and lumbar spine by DXA revealed lower BMD in subjects with higher salt intake. Lower TBS was also noted in the high salt consumption category. Among the 288 study subjects in whom BSI was available, those with a high salt intake were noted to have a higher BSI both at the NOF and at the lumbar spine (higher strain; reduced resistance to fracture) as compared to those with a lower salt intake. Table 3 details the BMD, trabecular bone score, and BSI in both groups.

3.4 | Salt intake and bone health

To identify if an increase in salt intake was associated with an increase in the prevalence of osteoporosis, the subjects were further categorized into three groups based on the tertiles of salt intake, that is. ≤6.0 g/day, 6.1–8.0 g/day and 8.1–13 g/day. The prevalence of osteoporosis, low TBS, high BSI (lumbar spine), vertebral fractures, and hypercalciuria significantly increased across the various salt intake categories and this is depicted in Figure 1.

3.5 | Correlation between salt intake and densitometric parameters

Excess dietary salt showed significant positive correlation with femoral neck ($r=0.143$; $p=0.015$) and lumbar spine BSI ($r=0.443$; $p<0.001$) and significant negative correlation with femoral neck ($r=-0.442$; $p<0.001$) and lumbar spine ($r=-0.619$; $p<0.001$) BMD. There was also a positive correlation between salt intake and urine calcium creatinine ratio ($r=0.169$; $p=0.001$). The dietary calcium intake had no correlation with the urine calcium creatinine ratio ($r=-0.008$; $p=0.882$). However, the bone resorption marker CTx had a significant positive correlation with the urine calcium creatinine ratio ($r=0.168$; $p<0.001$).

3.6 | ROC curve and logistic regression analysis

On doing an ROC curve analysis, it was found that dietary salt intake performed well in predicting osteoporosis at any site with an AUC of 0.870 (95% CI: 0.832–0.907) (Figure 2). At a threshold of >7.0 g/day, the sensitivity and specificity respectively were 85.4% and 68.8%. To determine the factors that were significantly associated with osteoporosis at any site, a logistic regression analysis with candidate covariates salt intake, urine calcium–creatinine ratio, age, BMI, and age of menopause, physical activity, and dietary calcium intake was done. It was found that salt intake,

TABLE 2 Comparison of low-salt intake and high-salt intake groups.

Parameter	Normal ranges	Low-salt group <7.2 g Mean ± SD $n=234$	High-salt group ≥7.2 g Mean ± SD $n=149$	p value
Age (years)		58.5 ± 6.8	61.8 ± 7.3	<0.001
BMI (kg/m^2)		26.3 ± 4.3	23.5 ± 4.8	<0.001
Parity		2.7 ± 1.2	2.7 ± 1.4	0.932
Age of menopause		46.3 ± 5.0	45.3 ± 5.5	0.075
Dietary calcium intake (mg/day)		461.2 ± 151.1	469.6 ± 147.1	0.568
Daily dietary salt intake (g)		5.7 ± 1.0	9.4 ± 1.3	<0.001
Physical activity (METs/week) Median (IQR)		840 (415–1680)	840 (510–1680)	0.869
Albumin corrected calcium (mg/dL)	8.3–10.4	9.1 ± 0.3	9.2 ± 0.3	0.075
Phosphate (mg/dL)	2.5–4.5	4.0 ± 0.5	4.1 ± 0.5	0.602
Creatinine (mg/dL)	0.5–1.2	0.7 ± 0.1	0.7 ± 0.2	0.825
Alkaline phosphatase (U/L)	40–125	96.3 ± 24.2	94.3 ± 21.4	0.400
Parathormone (pg/mL)	8–80	69.1 ± 31.6	75.7 ± 33.2	0.052
25(OH) vitamin D (ng/mL)	30–75	24.4 ± 10.9	25.8 ± 9.0	0.168
CTx (pg/mL)	226–1088	625.6 ± 300.8	785.6 ± 302.2	<0.001
P1NP (ng/mL)	16.0–73.9	63.2 ± 32.5	68.9 ± 30.1	0.081
Urine spot calcium/creatinine ratio	<0.20	0.12 ± 0.09	0.15 ± 0.10	<0.001

Abbreviations: BMI, body mass index; Ctx, C-terminal telopeptide of type 1 collagen; IQR, interquartile range; METs, metabolic equivalents; P1NP, N-terminal telopeptide of type 1 procollagen.

TABLE 3 Bone mineral density and trabecular bone score in the low-salt intake and high-salt intake groups.

Parameter	Low-salt group <7.2 g Mean ± SD $n = 234$	High-salt group ≥7.2 g Mean ± SD $n = 149$	p value
BMD at neck of femur (g/cm^2)	0.702 ± 0.110	0.597 ± 0.079	<0.001
BMD at lumbar spine (g/cm^2)	0.903 ± 0.141	0.712 ± 0.094	<0.001
TBS (unitless)	1.269 ± 0.081	1.193 ± 0.088	<0.001
Parameter	**Low-salt group Mean (SD) $n = 186$**	**High-salt group Mean (SD) $n = 102$**	**p value**
BSI (unitless) (neck of femur)	2.03 ± 0.38	2.13 ± 0.38	0.042
BSI (unitless) (lumbar spine)	2.04 ± 0.50	2.59 ± 0.68	<0.001

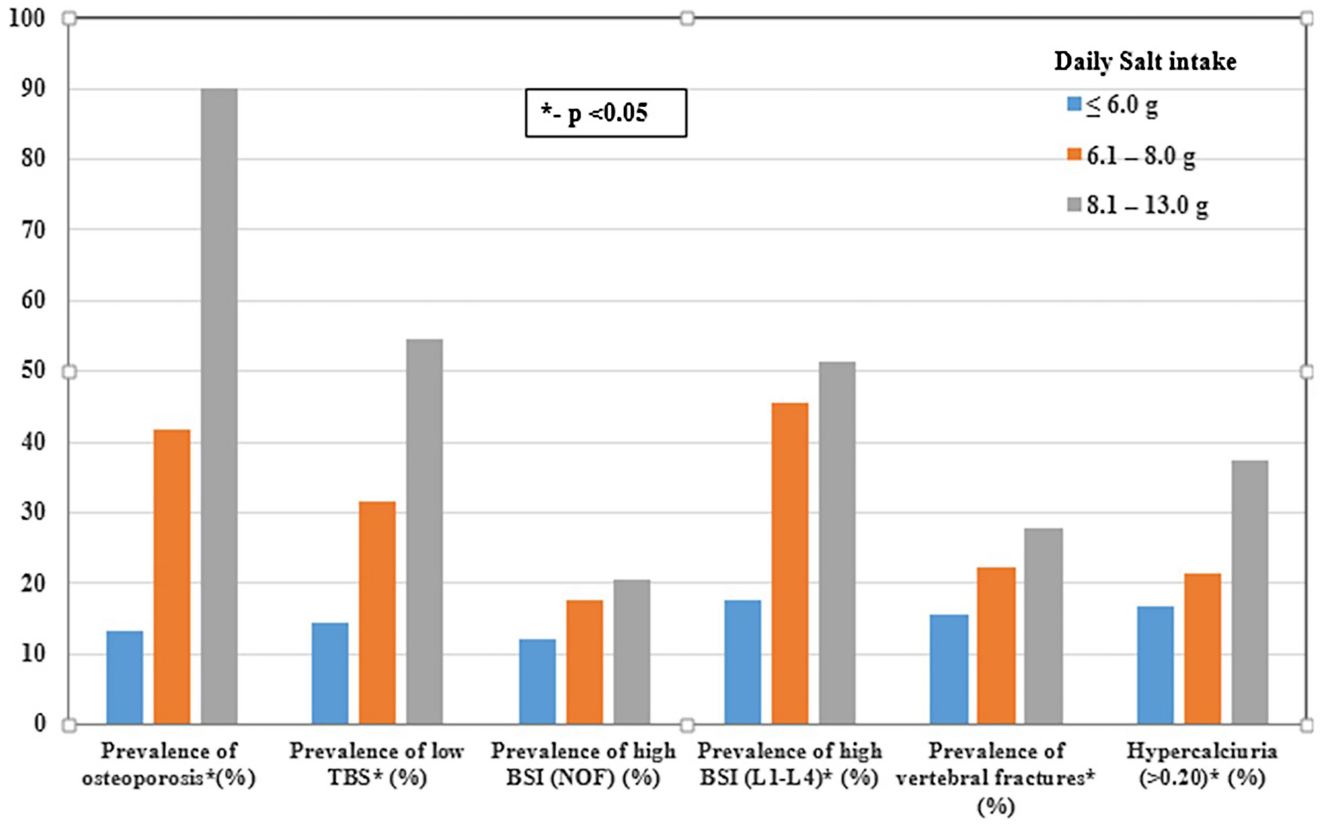

FIGURE 1 Prevalence of osteoporosis, low TBS, high BSI, and hypercalciuria in different salt categories. *indicates significance.

urine calcium–creatinine ratio, age, BMI, and age of menopause were significant on the univariate analysis. On doing a multivariate analysis, age, BMI, and salt intake continued to remain significantly associated, with salt intake having the highest odds ratio [OR = 2.296; 95% CI: 1.909–2.761] among candidate covariates in being associated with osteoporosis (Table 4).

4 | DISCUSSION

In this study, it was seen that high salt intake was associated with higher urine calcium excretion and compromised bone health. This was evidenced by higher bone resorption, lower BMD and TBS, as well as a higher BSI indicating a reduced resistance to fracture in the high-salt categories. The prevalence of osteoporosis as well as vertebral fractures increased significantly from lower to upper tertiles of salt intake. Moreover, among various covariates assessed, dietary salt intake conferred the highest odds in being associated with osteoporosis.

Osteoporosis is severely under reported and underdiagnosed in Indian postmenopausal women. Excess dietary salt intake is postulated to be associated with poor bone health.[14,15] The mean salt intake of our study group was 7.2 g/day. In India, the overall mean weighted salt intake in a systematic review has been reported to be about 10.98 g/day, which is much higher than the average salt intake among most other nations and well above the WHO recommendation of <5 g/day.[25,26] Women with a higher salt intake were older as compared to women whose salt consumption was <7.2 g/day. This seems to be in keeping with the finding by Sucheka et al., which suggested that the salt taste intensity decreased with advancing age.

The hedonic response to salt taste increases, likely leading to higher salt consumption in elderly.[27]

Women with a higher salt intake were seen to have greater bone turnover and higher urinary calcium excretion as compared to women with a salt intake <7.2 g. This was evidenced by the higher levels of CTx in the former group. Jones et al. in their study of 154 subjects showed that salt intake was associated with a rise in markers of bone resorption.[28] Massey LK in her review proposed that urinary calcium excretion increases approximately 40 mg for each 2300 mg increase in dietary sodium in normal adults.[29] High urine calcium excretion with increase in the markers of bone resorption have also been demonstrated in postmenopausal women by Sellmeyer et al.[30] and Evans et al.[31] Teucher et al. conducted a randomized cross-over trial consisting of four successive 5-week periods of controlled dietary intervention, each separated by a minimum 4-week washout in 11 women. Moderately low- and high-salt (3.9 vs. 11.2 gram) diets, reflecting lower and upper intakes in postmenopausal women was included in the diet. They concluded that salt was responsible for a significant change in bone calcium balance, from positive to negative, when consumed as part of a high calcium diet, but with a low calcium intake, the bone calcium balance was negative on both high- and low-salt diets.[15] A study at Parma Kidney Stone clinic conducted in 743 males and 429 females demonstrated that progressive increase in dietary salt intake was associated with increasing hypercalciuria in stone formers.[32]

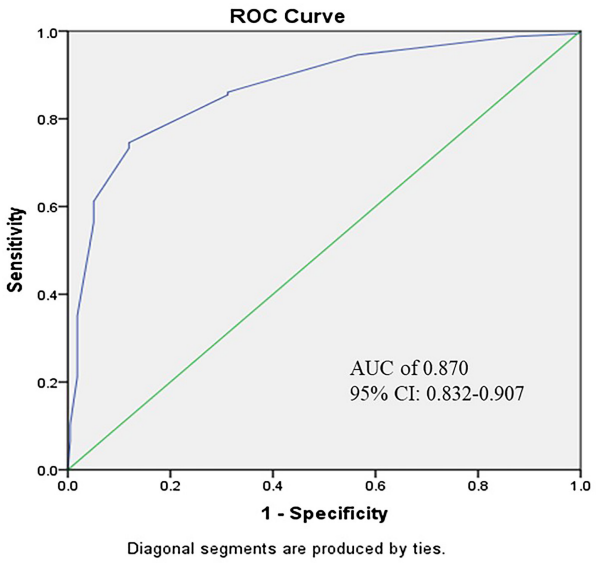

FIGURE 2 ROC curve between salt intake and osteoporosis at any site.

A high dietary salt (sodium chloride) intake has been considered detrimental to the bone as high salt intake increases urinary calcium excretion. The relationship between urinary calcium and urinary sodium excretion has been attributed to the presence of common pathways for reabsorption of both ions in the proximal convoluted tubule and the thick ascending limb of the loop of Henle. Hence, when dietary sodium chloride is increased, the fractional reabsorption of sodium is decreased, causing a simultaneous reduction in reabsorption of calcium.[33] Once again, this is one of the key reasons why low-sodium diets are prescribed among those with calcium stones of the renal system.

The BMD was significantly lower among women with a higher salt intake. It was also noted that the prevalence of osteoporosis increased across categories of low to high salt intake. Moreover, there existed a significant negative correlation between salt intake and BMD at the lumbar spine and femoral neck. This is in keeping with the findings by Kwon et al. on 4733 postmenopausal women from Korea, where high sodium intake was negatively associated with BMD of the lumbar spine.[34] However, there are studies that have been conflicting in this regard. Hong et al. demonstrated that a lower sodium diet below 2 g per day was an independent predictor for developing incipient osteoporosis and there was a sex disparity in the association between reduced sodium intake and the risk of incipient osteoporosis.[17] In the present study, however, none of the women had a salt intake that was less than 2 g/day. Carbone et al. in the data from the Women's Health Initiative reported that in adjusted models, there was no association of calibrated sodium intake with changes in BMD at the hip or lumbar spine from baseline to 3 or 6 years.[14] However, the mean calibrated salt intake in this large cohort was much lower than the present study; the two studies therefore may not be comparable. It hence appears that either end of the salt intake spectrum may be detrimental to bone health.

Excess salt intake was also noted to be associated with degraded bone microarchitecture and a higher BSI. This is indicative of a higher strain in the bone and an increased tendency to fracture. Preclinical studies have shown an enhanced bone loss and impaired bone microarchitecture in mice. This was attributed to an uncoupling of bone remodeling with increased bone resorption and decreased formation. Also, histomorphometric studies showed that in mice on a high-salt diet, there was increased osteoclastic activity suggesting

TABLE 4 Univariate and multivariate analyses to determine factors significantly associated with osteoporosis at any site.

Clinical covariate	Univariate unadjusted OR (95% CI)	*p* value	Multivariate adjusted OR (95% CI)	*p* value
Age (years)	1.095 (1.061–1.129)	<0.001	1.082 (1.038–1.129)	<0.001
BMI (kg/m^2)	0.805 (0.760–0.853)	<0.001	0.834 (0.775–0.896)	<0.001
Age of menopause (years)	0.954 (0.917–0.992)	0.018	0.952 (0.919–1.029)	0.326
Urine spot calcium creatinine ratio (unitless)	46.793 (5.059–432.785)	<0.001	3.400 (0.122–69.392)	0.462
Dietary salt intake (g)	2.411 (2.029–2.864)	<0.001	2.296 (1.909–2.761)	<0.001

that excess salt stimulated osteoclastogenesis.[35] Similar studies in humans in this regard were not available in literature till date. BSI is an innovative DXA based tool for assessing the level of strain in the bone and studies in this regard are overall limited in literature.

India is home to more than 100 million postmenopausal women. The authors have done several community-based studies pertaining to osteoporosis. The prevalence of osteoporosis in India is as high as 40%–60% at any site.[36] Moreover, the prevalence of asymptomatic vertebral fractures is about 30%.[8] Even in regularly menstruating premenopausal women, the prevalence of vertebral fractures is about 17%.[37] Notwithstanding, the knowledge about osteoporosis among these women is far from adequate. The average daily salt consumption in India is about 8.0–8.7 g[38] which is far above the WHO recommended guidelines of <5 g daily. Furthermore, the dietary calcium intake in Indian women is grossly inadequate.[39,40] The consumption of excess salt coupled with a poor dietary calcium intake heightens one's risk for osteoporosis. The relationship between salt intake and bone health have previously not been studied in Asian Indians. Salt intake is potentially a modifiable risk factor as is dietary calcium intake. Thus, in this study the authors have attempted to assess the relationship between salt-intake and osteoporosis; if such a relationship does exist, the question arises as to whether ongoing bone loss could be curtailed through limiting the salt intake at a younger age.

This is the first Indian study that has assessed the association between salt consumption and osteoporosis in postmenopausal women. To the best of our knowledge, it is also the first study evaluating the association between excess dietary salt and other densitometric indices such as TBS and the novel BSI. The major limitation of the study was that urine sodium, which is a less biased index of sodium intake, was available only in 15 participants. However, the population in which the study was carried out is homogeneous; they have similar eating habits, consume a predominantly vegetarian diet, with consumption of meat more commonly over the weekend. The socio-economic and cultural background of these rural women is such that most are unwilling for a 24-h urine collection. While it is well documented that excess intake of dietary salt can result in decreased renal reabsorption of calcium and consequent hypercalciuria, it is not known to what extent this hypercalciuria is compensated by increased intestinal absorption of calcium. Also, the sample size is small and BSI was not available in the entire cohort. As such, the present study is of a cross-sectional design that has shown an association between salt intake, calcium excretion and bone mass. Further studies assessing calcium excretion after dietary salt restriction followed by assessment of bone turnover markers would lend further validity to the study.

5 | CONCLUSION

Excess dietary salt intake appears to be a significant risk factor contributing to a compromised bone health in postmenopausal women. There is a progressive increase in prevalence of hypercalciuria as well as osteoporosis with increasing dietary salt. Further prospective studies looking into the impact of dietary salt restriction on bone health may be the way forward.

AUTHOR CONTRIBUTIONS

Conceptualization: Kripa Elizabeth Cherian, Thomas Vizhalil Paul, Nitin Kapoor. *Data curation*: Thomas Vizhalil Paul, Kripa Elizabeth Cherian. *Data analysis*: Rebecca John, Kripa Elizabeth Cherian, Nitin Kapoor, Thomas Vizhalil Paul. *Manuscript*: Rebecca John, Kripa Elizabeth Cherian. *Approval of final version of manuscript*: Rebecca John, Kripa Elizabeth Cherian, Nitin Kapoor, Thomas Vizhalil Paul.

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The authors have nothing to report.

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CONFLICT OF INTEREST STATEMENT

The authors declare that they have no conflict of interests.

ETHICS STATEMENT

The study was approved by the institutional review board and ethics committee (15843).

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