

Original Research Article

A prospective study of correlation between smoking and serum calcium in newly diagnosed essential hypertension

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ABSTRACT

Background: Smoking significantly reduces the vitamin D and serum parathyroid hormone levels. But no significant difference was observed in serum calcium levels in previous studies. Some studies showed significantly low serum calcium levels in essential hypertension. There are limited international and local studies about the effect of cigarette smoking on the levels of serum calcium in relation to hypertension. Our study aims to find correlation between smoking and serum calcium in newly diagnosed essential hypertensive patients.

Methods: Total 70 cases of newly diagnosed essential hypertension who attended the outpatient department at the government general hospital were included in this study based on inclusion criteria. Base line data including age, gender, risk factors were obtained. Blood biochemistry including serum calcium levels were obtained at the time of diagnosis and results were analysed.

Results: Among 70 cases of essential hypertension there were 24 smokers and 46 nonsmokers. Serum calcium was found low in 37 cases of essential hypertension. Out of 37, 12 were smokers and 25 were nonsmokers, p value is 0.6981 and it is not significant.

Conclusions: Smoking is a major health hazard, with detrimental effects on many organs. In our study serum calcium was significantly less among Hypertensives and correlated inversely with blood pressure. But there was no significant difference in serum calcium among smokers and nonsmokers. Measuring the level of parathyroid hormone in cigarette smokers, may help to clarify the finding in present study.

Keywords: Calcium, Hypertension, Parathyroid hormone, Smoking

INTRODUCTION

The role of calcium in the prevention and treatment of hypertension is controversial, despite decades of study. Essential hypertension is associated with disturbed calcium metabolism, some studies showed significantly low serum calcium levels in essential hypertension.¹ The calcium ion plays a major role as an intracellular second messenger in excitation contraction coupling in cardiac and smooth muscle cells. An increase in peripheral vascular resistance was a uniform finding in all types of

established hypertension. The free intracellular calcium concentration determines the tension in vascular smooth muscle cells thereby resulting in peripheral vascular resistance.² Low levels of calcium, either due to dietary deficiencies or altered calcium metabolism, have been linked by several epidemiological and laboratory studies to higher blood pressure, or hypertension.^{3,4} Essential hypertension is accompanied by abnormalities of calcium homeostasis, including hyperparathyroidism with reduced target organ responses to parathyroid hormone in kidney and bone. Hyperparathyroidism is also due to an intrinsic

defect in renal calcium handling. PTH levels may rise in response to low ionized serum calcium levels but have also been shown to increase in situations where ionized serum calcium levels are normal. Smoking is a major health hazard, with detrimental effects on many organs, including the skeleton. Possible explanations might be that smokers have a low intake of calcium and/or vitamin D, a low calcium absorption, a high calcium resorption from the skeleton or an excessive excretion of calcium in the urine. Smoking significantly reduces vitamin D and serum parathyroid hormone.

Aims and objectives of the study was to find correlation between smoking and serum calcium in newly diagnosed essential hypertensive patients.

METHODS

It is a prospective study in which 70 cases of newly diagnosed essential hypertension who attended the outpatient department of general medicine at the government general hospital (RIMS) were included in this study based on inclusion criteria in support with department of biochemistry, conducted for the study period of five months from July 2019 to December 2019.

Ethics committee clearance was obtained. Patients were explained about the study and informed consent was obtained.

Inclusion criteria

- New Hypertensives
- Age-18-60 yrs
- Not using any anti hypertensives
- No other comorbidities.

Exclusion criteria

- Patients with secondary hypertension.
- Diabetics.
- Readings with abnormal recordings (>20 mmHG in both arms).

Base line data including age and risk factors were identified. Sphygmomanometer readings were taken at two consecutive days at same time in sitting position in both arms and recorded is taken whichever is high, diagnosis was made by JNC 8 criteria.⁵ Blood biochemistry including serum calcium levels were obtained at the time of diagnosis and results were analysed.

RESULTS

The present study was conducted in order to study the correlation between smoking and serum calcium in newly diagnosed essential hypertension, as there are limited studies. We selected 70 patients of newly diagnosed essential hypertension attended to the OPD Government

general hospital kadapa on basis of inclusion criteria. Among them the patients with essential hypertension were categorized based on personal history of smoking into smokers and nonsmokers and serum calcium levels were obtained for all the 70 patients. And results were analysed using chi square test. And results are represented in form of pie charts, bar diagrams and tables.

Among the 70 cases of essential hypertension there were 24 smokers and 46 nonsmokers (Figure 1). Serum calcium was found low in 37 cases of essential hypertension (Table 1). Out of 37 patients with low serum calcium, 12 were smokers and 25 were nonsmokers, p value is 0.6981 and it is not significant. In this study serum calcium was significantly less among hypertensives and correlated inversely with blood pressure. But there was no significant difference in serum calcium among smokers and nonsmokers.

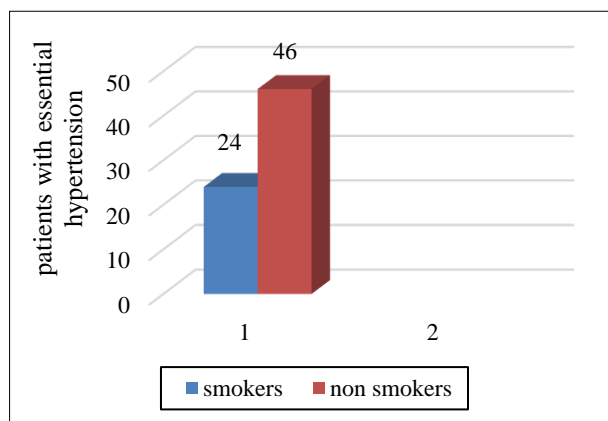


Figure 1: Patients with essential hypertension number of smokers and nonsmokers.

Table 1: Serum calcium levels among smokers and nonsmokers.

Findings	Smokers	Nonsmokers	Total
Decreased levels of serum calcium	12	25	37
Normal levels of serum calcium	12	21	33
Total	24	46	70

DISCUSSION

In subject with a genetically determined predisposition to the development of hypertension, a high sodium consumption leads to volume overload, which results in the appearance of a natriuretic hormone in the circulation.⁶ It has been suggested that the excessive excretion of calcium and phosphorus associated with exaggerated natriuresis may participate in aberration of calcium metabolism in low rennin hypertensive seniles. By definition, natriuretic hormone causes natriuresis by

blocking sodium reabsorption in the renal tubules and this blockade appears to involve inhibition of the ouabain sensitive Na-K pump that activity extrudes sodium into the extracellular fluid. This natriuretic hormone influences membrane permeability which leads to an increase in intracellular sodium and by inhibiting sodium calcium exchange, causes an accumulation of calcium in vascular smooth muscle cells. The increase in intracellular calcium then leads to an increased contractility and vascular tone resulting in an augmented peripheral vascular tone resulting in an augmented peripheral vascular resistance and consequently in raised blood pressure.⁷ Increased intracellular sodium will raise the concentration of free calcium within the vascular smooth muscle cells. A number of relations exists between sodium and calcium that would explain how this happens.

Blaustein portrays some of these.⁸

- An inhibition of sodium potassium exchange pumps would depolarize the muscle fiber and thereby increase calcium entry through voltage - sensitive calcium channels.
- An increase in intracellular sodium will result in a smaller sodium (Na) electrochemical gradient between the sarcoplasm and external medium, thereby decreasing the extrusion of calcium from the cell via the Na-Ca exchange which derives its energy from this gradient.
- An increase in intracellular sodium in the presynaptic terminals of sympathetic neurons promotes calcium dependent norepinephrine release. The norepinephrine releases calcium from intracellular stores.

In summary, vascular smooth muscle intracellular calcium is likely elevated in primary hypertension. Since intracellular calcium directly controls vascular contraction and relaxation, the connection to hypertension is obvious. Increased vascular tone and reactivity are likely responsible for much of the increased total peripheral vascular resistance that is the primary cause of sustained hypertension.

The significance of the reduced binding of calcium to the inner aspect of the plasma membrane is not clear. Calcium exerts an important regulatory effect on various aspects of membrane function. It is possible that the reduction in binding is a cause of some of the abnormalities of ion transport. It may be, however, that the reduction in binding is merely a marker of an alteration in membrane composition. Which results in a reduced availability of binding sites.

Relation between whole body calcium metabolism and events at cellular level

Evidences suggest some mechanism by which the calcium balance of the body as a whole can influence

events at a cellular level. The link may involve change in the plasma ionized calcium concentration or changes in calcium regulating substances such as parathyroid hormone or 1-25 dihydroxy vitamin D. A rising concentration of calcium in the extracellular fluid has been shown to inactivate certain potential operated calcium channels in tenia coil and a falling concentration must have contrary effects. It is probable that human resistance vessels respond in a similar way, although the manifest response to a rise in calcium concentration is contraction.

Parathyroid hormone probably acts on cell of many types apart from those of renal tubules and bone and increase in cytosolic calcium. At concentration, greatly in excess of those occurring physiologically. Parathyroid hormone dilates resistance vessels, but the effect, if any, of the very small increases observed in patients with primary hypertension is unknown. It is possible that a mechanism exists by which the free calcium in the intake, but it seems likely that the disorder of calcium handling at a cellular level predominantly from other causes.⁹ Effect of alteration in calcium intake. If a calcium intake that is suboptimal relative to need plays any part in the causation of primary hypertension, it would be expected that the administration of calcium supplements would lower blood pressure, at least in the early stages of the hypertensive process.

The majority of observational studies show a clear inverse relationship between calcium and both the prevalence of hypertension and the level of blood pressure.⁴ PTH levels may rise in response to low ionized serum calcium levels, but have also been shown to increase in situations where ionized serum calcium levels are normal.¹⁰ Smoking is a major health hazard, with detrimental effects on many organs, including the skeleton. Possible explanations might be that smokers have a low intake of calcium and/or vitamin D, a low calcium absorption, a high calcium resorption from the skeleton or an excessive excretion of calcium in the urine.¹¹ Smoking significantly reduces vitamin D and serum parathyroid hormone.¹¹

CONCLUSION

Hypertension is one of the leading causes of death and disability among all over the world. It remains the major risk factor for coronary, central and peripheral vascular disease. Smoking is a major health hazard, with detrimental effects on many organs including skeleton. Smoking significantly reduces vitamin D and serum parathyroid hormone. Hence the present study is undertaken to find correlation between serum calcium, smoking, hypertension. In our study serum calcium was significantly less among hypertensives and correlated inversely with blood pressure. But there was no significant difference in serum calcium among smokers and nonsmokers. From our study it was concluded that in patients with essential hypertension there was no

significant difference in correlation with smoking and serum calcium. Measuring the level of parathyroid hormone in cigarette smokers, may help to clarify the finding in our study.

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Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Grobbee, D.E., I.M.S. van Hooft, and A. Hofman. Calcium Metabolism and familial Risk of Hypertension. *Sem Nephrol.* 1995;15(6):512- 18 .
2. Sudhakar K, Sujatha M, Babu SR, Padmavathi P, Reddy PP. Serum calcium levels in patients with essential hypertension and their first degree relatives. *Ind J Clini Biochem.* 2004;19(1):21-3.
3. McCarron DA. Calcium. Symposium Notes: Non pharmacologic Management of Hypertension (National Kidney Foundation). 1994:34-8.
4. Morris CD, Reusser ME. Calcium Intake and Blood Pressure: Epidemiology Revisited. *Sem Nephrol.* 1995;15(6):490-5.
5. James PA, Oparil S, Carter BL, Cushman WC, Dennison-Himmelfarb C, Handler J, et al. 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8). *JAMA.* 2014 Feb 5;311(5):507-20.
6. Anchala R, Kannuri NK, Pant H, Khan H, Franco OH, Di Angelantonio E, et al. Hypertension in India: a systematic review and meta-analysis of prevalence, awareness, and control of hypertension. *J Hypertension.* 2014 Jun;32(6):1170-7.
7. Hugo K, Jozer G. Calcium and blood pressure. *Lancet.* 1982:813-5.
8. Blaustein MP. How does sodium cause hypertension? An hypothesis. In: Zunkley H, Losse H (eds) *Intracellular electrolytes and arterial hypertension.* Thieme: Stuttgart; 1980:151–157.
9. Laragh JH, Brenner BM. *Hypertension pathophysiology, Diagnosis and Management.* 2nd Edition. New York: Raven Press, Ltd.; 1995:1169-1189.
10. Oshima T, Young EW. Systemic and Cellular Calcium Metabolism and Hypertension. *Sem Nephrol.* 1995 Nov;15(6):496-503.
11. Jorde R, Saleh F, Figenschau Y, Kamycheva E, Haug E, Sundsfjord J. Serum parathyroid hormone (PTH) levels in smokers and non-smokers. The fifth Tromsø study. *Europ J Endocrinol.* 2005 Jan 1;152(1):39-45.

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