SUMMARY AND CONCLUSION

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Abnormalities of Calcium metabolism have been described in patients with essential hypertension. The role of calcium in the regulation of blood pressure is receiving increased attention

The aim of this work is to study calcium homeostasis in essential hypertensive patients to shed light on the mechanisms played by calciotropic hormones in the pathogenesis of essential hypertension. Thirty essential hypertensive patients as well as ten well matched healthy volunteers as control had been selected for this study.

Diagnosis of secondary hypertension was adequately excluded by meticulous history and clinical examination and by the finding of normal urine analysis, serum creatinine and serum electrolytes, and by radiologic studies, and electrocardiography (ECG), when clinically indicated.

Patients taking contraceptive pills, or injections, glucocorticoids, phosphate binding antacids, sex hormones or those on weight reducing diets, or pregnant were excluded from the study.

Patients and controls were subjected to full medical history and examination and the following investigations were done:

1-Serum creatinine.

2-Serum sodium and potassium

3-serum calcium, 1.25 $(OH)_2D$ and Parathyroid hormone (PTH).

4-Plasma renin activity (PRA).

5-24 hours urinary calcium.

The results of this work showed:

1-No significant difference between the mean serum calcium values in the hypertensive group and the control group.

2-Significant elevation of the mean value of 24 hours urinary calcium in the hypertensive group when compared to the control group, with hypercalciuria in 70% of patients.

3-Significant elevation of the mean value of serum 1,25 $(OH)_2D$ in the hypertensive group when compared to the control group with elevated serum 1,25$(OH)_2D$ in 40% of patients.

4-Significant elevation of the mean value of serum PTH in the hypertensive group when compared to the control group, with elevated serum PTH in 60% of patients.

5-No correlation was observed among serum PTH, serum 1,25 $(OH)_2D$ and urinary calcium in the hypertenisve group.

From this study, we come to the conclusion that:

-Hypercalciuria is a major derangement of calcium metabolism in patients with essential hypertension. This may be due to a defective renal tubular calcium transport.

-The elevated PTH and 1,25 $(OH)_2D$ in essential hypertension might be a compensatory response in trial to restore calcium balance to normal.

-These calciotropic hormones directly facilitate calcium transport from the extracellular space in the cell resulting in intracellular calcium accumulation and increase in cytosolic free ca^{++} concentration found in patients with essential hypertension. The cytosolic free calcium concentration is crucial for the vascular contractile activity and hence, elevation in blood pressure.

Further research is recommended to study the association among systemic markers of calcium metabolism, cellular calcium metabolism and arterial blood pressure regulation.