



## A STUDY TO FIND THE CORRELATION BETWEEN HYPERVITAMINOSIS AND SERUM CALCIUM LEVELS IN PATIENTS HAVING RENAI STONES IN SOLAN DIST.

### General Surgery

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### ABSTRACT

#### INTRODUCTION

Hypercalcemia and hypervitaminosis D had been postulated as a cause of renal stones In Children ". Several studies and meta analysis had clearly shown correlation between high serum vitamin D and renal stones. Vitamin D is important for development of bones in children. Deficiency of vitamin D can lead to hypocalcemia, bone demineralisation , Rickets and growth retardation. There is high prevalence of Vitamin D deficiency in India and more so in hilly areas. Prevalence of renal stones is also higher in these areas. This is in contrast to common finding of high vitamin D and calcium levels in stone formers. This study will be conducted to determine the levels of vitamin D and serum calcium levels in children with renal stones. Further in patients of Vitamin D deficiency repletion of vitamin D will be done and effects will be noted after 3 months. Kidney stones are common, with an estimated prevalence of about 10% in the US population. 1 Higher urine calcium excretion is a major risk factor for calcium stone formation, which in turn might be increased by higher circulating levels of 1,25-dihydroxyvitamin D (1,25[OH]2D). In a prospective nested case-control study, the odds of kidney stones were 73% higher for those in the highest quartile of 1,25[OH]2D.5 The association between precursors of 1,25[OH]2D such as circulating 25-hydroxyvitamin D (25[OH]D) and intake of cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2) is less clear.

#### Material and Methods

This study will be conducted In 50 patients coming In surgery 0P0 with renal stones/concretions/ nephrollthiasls.

#### Inclusion criteria

1. Age between 15-50 years . 2. Unilateral or bilateral stones. Exclusion criteria. 1. Anatomncal abnormalities nice PUJO. horse shoe kidney. 5 shaped kidney 2. Metabolic abnormalities which cause hyperoxaluria, hypercalciurla.

#### Aim

1. To study the levels of vitamin D and calcium in patients of renal stones
2. To treat hypovitaminosis if present and to study Its effects on renal stones

#### Summary and conclusion

There is growing evidence that cholecalciferol administration , in the higher ranges, may increase urinary calcium excretion and kidney stone formation in predisposed individuals or specific groups of patients , the observation of an association between "low" levels of circulating vitamin D serum levels and a broad spectrum of diseases has been at the origin of a dramatical increase in the prescription of vitamin D.

### KEYWORDS

#### INTRODUCTION

Hypercalcemia and hypervitaminosis D had been postulated as a cause of renal stones In Children ". Several studies and meta analysis had clearly shown correlation between high serum vitamin D and renal stones. Vitamin D is important for development of bones in children. Deficiency of vitamin D can lead to hypocalcemia, bone demineralisation , Rickets and growth retardation. There is high prevalence of Vitamin D deficiency in India and more so in hilly areas. Prevalence of renal stones is also higher in these areas. This is in contrast to common finding of high vitamin D and calcium levels in stone formers. This study will be conducted to determine the levels of vitamin D and serum calcium levels in children with renal stones. Further in patients of Vitamin D deficiency repletion of vitamin D will be done and effects will be noted after 3 months. Kidney stones are common, with an estimated prevalence of about 10% in the US population. 1 Higher urine calcium excretion is a major risk factor for calcium stone formation, which in turn might be increased by higher circulating levels of 1,25-dihydroxyvitamin D (1,25[OH]2D). In a prospective nested case-control study, the odds of kidney stones were 73% higher for those in the highest quartile of 1,25[OH]2D.5 The association between precursors of 1,25[OH]2D such as circulating 25-hydroxyvitamin D (25[OH]D) and intake of cholecalciferol (vitamin D3) and ergocalciferol (vitamin D2) is less clear. Oral supplementation with cholecalciferol has been associated with increased risk of stones when administered together with calcium; however, administration of ergocalciferol in stone formers with vitamin D deficiency did not cause a significant rise in mean urinary calcium excretion. This is an important issue as vitamin D insufficiency and low bone mineral density are common among stone formers, and also because associations between vitamin D status and other conditions such as high blood pressure, diabetes, and cardiovascular events have been

reported, all frequent among stone formers. To date, only two longitudinal studies investigated the association between intake of vitamin D and risk of kidney stones, reporting no association. Ingested and cutaneously produced vitamin D is rapidly converted to 25(OH)D, but in serum only a fraction of 25(OH)D is converted to its active metabolite 1,25(OH)2D. Thus, measurement of the total 25(OH)D level is the best test to assess body stores of vitamin D. The total 25(OH)D level allows for the diagnosis and monitoring of vitamin D deficiency, whereas quantification of 25(OH)D2 and 25(OH)D3 fractions may facilitate treatment monitoring. For example, in patients without clinical improvement after D2 or D3 supplementation, lack of increase in the corresponding 25(OH)D2 or 25(OH)D3 and total 25(OH)D levels may indicate inadequate dosing, nonadherence, or malabsorption. Some laboratory assays for vitamin D cannot differentiate between 25(OH)D2 and 25(OH)D3 and will only report a total 25(OH)D level. Some laboratory assays underdetect D2 metabolites, which may give the appearance of ineffective D2 supplementation. Two forms of vitamin D can be measured in the blood, 25-hydroxyvitamin D and 1,25-dihydroxyvitamin D. The 25-hydroxyvitamin D is the major form found in the blood and is the relatively inactive precursor to the active hormone, and 1,25-dihydroxyvitamin D. Because of its long half-life and higher concentration, 25-hydroxyvitamin D is commonly measured to assess and monitor vitamin D status in individuals.

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Exclusion criteria. 1. Anatomical abnormalities like PUJO, horse shoe kidney, 5 shaped kidney 2. Metabolic abnormalities which cause hyperoxaluria, hypercalciuria.

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### OBSERVATIONS

Total number of patients :-50

**Table 1**

Age group	Total number of patients
15-20years	2
20_30 years	7
30-40 years	25
40-50 years	16

**Table 2 Level of calcium in patients**

Level of calcium	Number of patients
<8.5mg/dl	2
8.5-10.5mg/dl	47
>10.5mg/dl	1

**Table 3 Level of vitamin D**

Level of vitamin D	Number of patients
<30ng/dl	1
30-50ng/dl	49
>50ng/dl	0

**Table 4 Vitamin C levels**

Vitamin C level	Number of patients
<0.6mg/dl	0
>or=0.6mg/dl	50

### DISCUSSION

Vitamin D is essential for calcium absorption and for maintaining bone health in paediatric population. The causes of Vitamin D deficiency can be nutritional deficiency, malabsorption, patients with chronic illnesses, dark pigmented skin. Deficiency of Vitamin D leads to hypocalcaemia, rickets and osteomalacia in children and adults. Vitamin D toxicity, hypervitaminosis which occurs due to giving too much vitamin D can lead to hypercalcaemia and hyperphosphatemia. There is strong correlation between levels of vitamin D and incidence of urolithiasis in children (2, 3).

In majority of children causes of stones are due to metabolic abnormalities like hypercalcaemia, hypercalciuria or hyperoxaluria. Out of these hypercalcaemia is the most common cause of primary urinary cause in children. Hypercalcaemia can occur due to excess calcium absorption from intestine which occurs in the patients of hypervitaminosis D (4,5). Several studies have suggested that increased levels of circulating vitamin D and C is associated with urinary stones especially calcium stones (6).

In India study was conducted in 2017, it was shown that vitamin D deficiency is prevalent in children residing in higher altitude region (7). This is contrary to the fact that they have adequate sunshine and low air pollution as compared to plain areas. Also people residing in these areas have high incidence of renal stones. This could be probably these people are drinking less water. It has been proven that all types of calculi are less likely to occur in dilute urine. Some recent studies have shown low levels of vitamin D in children of nephrolithiasis (8,9). Another study conducted in US has shown no correlation between high serum vitamin D and renal stones (10).

We are conducting this study to find correlation between serum calcium and vitamin D levels in children with renal stones in hilly areas.

Without medical treatment, the 5-year recurrence rate is high, ranging from 35–50% after an initial stone event. A high fluid intake, enough to

produce at least 2.5 L of urine per day, should be the initial therapy to prevent stone recurrence. Recommendations for preventing stone formation depend on the stone type and the results of metabolic evaluation. After remediable secondary causes of stone formation (e.g. primary hyperparathyroidism) are excluded, the focus should turn to modification of the urine composition to reduce the risk of new stone formation. Dietary modifications have a major role in the management of recurrent stones that are due to hypercalcaemia. Dietary calcium should not be restricted, since calcium reduces the excretion of urinary oxalate by decreasing intestinal absorption of oxalate. Guidelines from the AUA recommend a daily calcium intake of 1,000–1,200 mg. Moreover, restriction of dietary calcium to <800 mg/day (the current recommended daily allowance for adults) can lead to negative calcium balance and bone loss. Sodium intake also influences hypercalcaemia. Calcium is reabsorbed passively in the proximal tubule due to the concentration gradient created by active reabsorption of sodium. A high sodium intake causes volume expansion, leading to a decrease in proximal sodium and calcium reabsorption and enhancing calcium excretion. A low-sodium diet (80–100 mmol/day, or 1,800–2,300 mg/day) is recommended. This enhances proximal sodium and passive calcium absorption and leads to a decrease in calcium excretion. Dietary protein increases the acid load by production of sulphuric acid and leads to hypercalcaemia by its action on bone and kidney. Animal protein has a higher content of sulphur and generates a higher acid load compared to vegetable protein, with animal protein associated with an increased incidence of stone formation. It has been seen that the combination of restricted intake of animal protein (52 g/day), restricted salt intake (50 mmol, or 2,900 mg/day of sodium chloride), and normal calcium intake (30 mmol/day, or 1,200 mg/day) was associated with a lower incidence of stone recurrence in men with hypercalcaemia, compared with traditional low-calcium intake (10 mmol, or 400 mg/day). Patients should therefore be advised to avoid excessive intake of animal protein.

### Summary and conclusion

There is growing evidence that cholecalciferol administration, in the higher ranges, may increase urinary calcium excretion and kidney stone formation in predisposed individuals or specific groups of patients, the observation of an association between "low" levels of circulating vitamin D serum levels and a broad spectrum of diseases has been at the origin of a dramatical increase in the prescription of vitamin D. Increase vitamin C levels can predispose to renal stones and the reason for it are multifactorial.

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