SUMMARY
&
CONCLUSION
SUMMARY AND CONCLUSION

The present study has been undertaken in view of rising incidence of renal stone in Bundelkhand region. The present study comprises of "Prevalance of hypercalciuria in renal stone formers and it's relationship with diet".

In this study 500 cases of renal stone patients were taken whereas 300 cases of normal population. Patients investigations was done in Renal OPD, Medicine and Surgery department of M.L.B Medical College Jhansi whereas cases of normal population studied from various part of Bundelkhand region. The subjects cases were followed 2-year period and were assessed by a thorough history, dietary analysis, and serum and urinary chemistry evaluation.

The aims and objectives

1. To Study the prevalance of calcium stones, their causes and prevention.

2. To study the effects of high and low calcium intake in renal stone formers.

3. To study the role of diet in calcium stone formers, compare with normal population.

4. To study the effects of fluid intake.

5. To study the effect of hypercalcemia on calcium stone formers compare with normal population.

6. To study hypercalciuria, its effect on calcium stone formers and normal population.
Method applied

(i) Usually, 24-hour urine collections are collected and analyzed for calcium while the patient as well as normal population was on a regular diet. This is undertaken to confirm the diagnosis, to determine hypercalciuria.

(ii) Ideally, serum laboratory studies should be drawn at the same time the 24-hour urine sample is being collected.

**BRIEF OUTLINE OF DIETARY FACTORS:**

Several dietary factors besides calcium can contribute to hypercalciuria: These include animal protein, sodium, alcohol, caffeine, refined carbohydrates, fiber, oxalate, and fluids.

**Excessive animal protein:** (>1.7 g/kg of body weight) increases the body's acid load. This additional acid load is buffered or neutralized in part by the bony skeleton, which then releases calcium into the general circulation. This extra serum calcium eventually is excreted by the kidneys into the urine. Acid loading also directly inhibits renal calcium reabsorption, resulting in an increase in urinary calcium excretion. Animal protein also contributes a large purine load. Purines are the precursors of uric acid, which can form uric acid stones, lower the urinary pH, increase the overall acid load, contribute to gouty diatheses (a condition involving both stone disease and elevated uric acid levels), and generally increase urinary calcium excretion and stone formation. **Robertson and Peacock**, (1982) views that vegetarians are at lower risk of forming stones.

**Sodium:** intake is another significant dietary risk factor for kidney
stone disease and hypercalciuria. High dietary sodium is associated with increased calcium release from bone, further contributing to any existing hypercalciuria. It also causes an increase in urinary calcium excretion through a direct effect on the kidneys and reduces or eliminates the hypocalciuric effect of thiazide therapy in hypercalciuria. Each 100-mEq increase in daily sodium intake raises urinary calcium excretion by about 50 mg/d.

**High dietary fiber:** binds to free intestinal calcium, reducing its absorption.

**Refined carbohydrates:** which increase intestinal calcium absorption.

**Effect of Fluid, beverages and alcoholic beverages:**

**Increasing fluid:** Fluid intake lowers urinary calcium concentrations without affecting total calcium excretion. *Shuster, et al. (1992)* views that increases in fluids is helpful. Some research has linked soft drinks (especially cola beverages) that contain phosphoric acid with a higher risk of stone formation.

**Alcohol:** Acute alcohol ingestion causes hypoparathyroidism with hypercalciuria and hypercalcemia. PTH levels can drop by 70% after acute alcohol intoxication. Intake should be limited because ethanol will reduce osteoblastic activity, lower parathyroid hormone (PTH) levels, and contribute to osteoporosis. It also indirectly accelerates osteoclastic activity, increases urinary calcium excretion, and contributes to bone loss.

**Caffeine:** intake also should be limited because caffeine will increase urinary calcium excretion. The ingestion of 34 ounces of caffeine will cause a loss of 1.6 mmol of total calcium, contributing
to both hypercalciuria and osteoporosis.
Another dietary factor that affects calcium excretion is: only undergoes this transformation in urine after the urine has left the body.

**DIETARY TREATMENT GUIDELINES**

(i) Limit daily calcium intake to 600-800 mg/d unless otherwise instructed.

(ii) Limit dietary oxalate whenever calcium intake is reduced.

(iii) High oxalate levels are found in teas, nuts, chocolate, colas, green leafy vegetables (e.g., spinach), and other plant and vegetable products.

(iv) Avoid excessive purines and animal protein (<1.7 g/kg of body weight).

(v) Reduce sodium (salt) and refined sugar to the minimum possible.

(vi) Increase dietary fiber (12-24 g/d).

(vii) Limit alcohol and caffeine intake.

(viii) Increase fluid intake, especially water (sufficient to produce at least 2 L/d of urine).

**Suggestion**

In hypercalciuria, the degree of hypercalciuria is worsened by high dietary sodium intake, potassium, high animal protein intake, carbohydrates, fiber, oxalate, alcohol, caffeine, and low fluid intake. No relationship was found with dietary fat intake as well as dietary restriction of sodium and protein. Several studies have shown that a higher dietary calcium intake has been associated with fewer calcium stone events in both men and women.
CONCLUSION:

1. It is clear that the stone development occurs when the chemical conditions are favourable, regardless of what any arbitrary reference range might be. Calcium Stone disease is the most common form of nephrolithiasis and represents about 70% of all stone forming disease. It occurs most often in the third decade of life, more often in men than women. Calcium Oxalate and Calcium Phosphate Stones are made up of a hard crystal compound. These Stones have become more common in recent years with about 75% to 80% of all Kidney Stones commonly made up of Calcium Oxalate and Calcium Phosphate. The crystals of these Stones grow abnormally fast in the urines of patients with recurrent renal stone formation.

The biomineralization resulting in a urinary stone has a multifactorial origin in which socio-economic, genetic and constitutional factors as well as diet and metabolic abnormalities might act concerned. A supersaturation of urine with the stone forming salt(s) is of fundamental importance and a prerequisite for the necessary precipitation. Calcium stones are favoured by very low urinary pH. Food with high or moderate amounts of Oxalate group are much more sensitive to dietary Calcium than non-stone formers. For most practical purposes, the 250 - mg / d limit for 24 - hour urinary calcium excretion is used regardless of sex when considering the relative risk of calcium Kidney stone production. No clear biochemical difference was found when risk factors were compared between various other regions of the country. Although
nutritional and environmental influences would be expected to produce some variability, stone formers in all of the region tested showed a striking similarity in urinary chemical risk factor profiles with no significant biochemical differences noted that could be attributable to geographical factors.

2. Patients with the history of kidney stones found that those with a relatively high dietary intake of Calcium (more than 1000 mg/day) actually experienced lower incidence of kidney stones compared with those with a relatively low dietary Calcium intake, so it may be tempting to recommend that patients at high risk of kidney stones start taking Calcium supplements. Patients who consumed more dietary Calcium from food also experienced a reduced risk of developing kidney stones. Calcium containing antacids should be avoided and those containing magnesium. However, there appears to be no reason to routinely discourage the consumption of most calcium rich foods unless they are also very high in oxalic acid. A Calcium intake from foods of 800 to 1200 mg/day is probably best for most patients with a history of Calcium-rich kidney stones. Avoidance of an excessively high Calcium diet is an obvious recommendation for calcium stone formers. Most patients with idiopathic hypercalciuria exhibit excessive gastrointestinal absorption of calcium (absorptive hypercalciuria). The patients also have in appropriately high levels of urinary calcium excretion even when on a calcium-restricted diet, which is why a calcium-restricted diet is not advised for these patients.
3. A Calcium intake from foods 800-1200 mg/day is probably best for most patients with a history of calcium-rich kidney stones. Food with high or moderate amounts of oxalate should be reduced or eliminated from the diet. Although there are many foods that contain large amounts of oxalate such as rhubarb, spinach, strawberries, chocolate, wheat bran, nuts, beets and tea. Since calcium oxalate is responsible for most kidney stones, avoiding foods containing oxalate may be helpful. A high sodium intake promotes a variety of effects that enhance urinary calcium excretion and increase overall kidney stone formation rates. These effects include a rise in urinary pH, higher urinary calcium and cystine levels, and a decrease in urinary citrate excretion. Sodium intake among stone formers is equal to or higher than intake in controls of non-stone formers.

Some evidence suggests that low potassium intake may be a risk factor for stones, but this has not been confirmed in all studies. The potential influence of a low potassium diet may be due to its relationship to sodium intake in stone formers, who generally have a high sodium-to-potassium ratio than non-stone formers. High dietary proteins intakes are known to increase urinary calcium excretion and if maintained will result in sustained hypercalciuria. Animal protein affects urinary calcium mainly through its acid loading ability. Animal protein is high in purines, which are metabolized to uric acid, further contributing to the acid load. Evidence also indicates that excessive carbohydrate loading can increase endogenous oxalate production. This seems reasonable because glucose is involved in oxalate metabolism through a series of chemical
interactions with glyoxylate calcium stone formers as a group have a lower intake of dietary fiber than non-stone formers. Although no reports of significant problems with increased dietary fiber have been made, some potential risk exists.

4. Water intake is the most important preventive measure for all patients who develop kidney stones. It hinders the formation of stones by diluting the urine.

At least 50% of the total fluid intake should be water. In warmer climates and for physically active people, an even higher fluid intake is recommended.

Studies have shown that, on average, stone formers have a lower overall fluid intake than non-stone formers. Not surprisingly, the highest incidence of kidney stone formation was in the group with the lowest overall fluid intake.

The need for a high fluid intake to increase urinary fluid volume seems obvious because extra water decreases urinary concentration and reduces the likelihood of stones even if the total calcium excretion is unchanged. The amount of extra water to be consumed is variable. In general, the author suggests an amount of water that produces a 24-hour urinary volume of 2000ml or more. This amount may need to be increased in selected cases.

Caffeine has been shown to increase urinary calcium excretion, but the clinical importance is relatively small unless very large amounts of caffeine are ingested. As noted earlier, ingestion of 1/4th teaspoon of caffeine is necessary to cause the loss of 1.6 mmol of total calcium. This caffeine-induced
hypercalciuria seems to parallel changes in urinary prostaglandin F2-alpha (PGF2-alpha), which suggests that prostaglandins may play a role in this entity.

Drinking coffee or other caffeine-containing beverages increases urinary calcium. Coffee and tea consumption is actually associated with a reduced risk of forming a kidney stone. These reports suggest that the helpful effect of consuming more water by drinking coffee or tea may compensate for the theoretically harmful effect that caffeine has in elevating urinary calcium. Therefore, the bulk of current research suggests that it is not important for kidney stone formers to avoid coffee and tea. The findings of some but not all studies suggest that consumption of soft drinks may increase the risk of forming a kidney stone.

Acute alcohol ingestion causes hypoparathyroidism with hypercalciuria and hypocalcemia. PTH levels can drop by 70% after acute alcohol intoxication. Prolonged but moderate alcohol intake eventually will raise PTH levels. People with chronic alcoholism develop low serum vitamin D levels, which cause impaired intestinal calcium absorption and hypocalciuria. A direct inhibitory effect on osteoblast activity by alcohol ingestion also appears to exist. This effect is enhanced in smokers. Urinary calcium excretion during periods of alcohol consumption can increase by over 200% over controls. Osteopenia also has been linked to alcohol consumption.

Potassium-rich citrus fruits and juices, such as oranges, grapefruit, and cranberries, are recommended. Orange juice, for example, has natural potassium citrate. Lemon juice also
has a very high citrate content, so lemonade made from real lemon juice is recommended. In contrast, lime juice contains mostly citric acid and does not increase urinary citrate substantially.

5. Hormone which rapidly lowers an artificially raised plasma calcium is produced by the parafollicular or C cells present in the thyroid, parathyroid and thymus glands and is known as calcitonin. The concentration of ionised calcium in the blood is raised, as may be the blood urea, plasma cholesterol and blood pressure. Hypercalcemia occurs in adults as a result of hyperparathyroidism or through excessive intake of vitamin D.

6. Many patients with kidney stones are known to have intestinal hyperabsorption of calcium. Each 100-mEq increase in daily dietary sodium raises the urinary calcium level by about 50 mg/d. Increased calcium excretion is thought to be due to an increase in the extracellular fluid volume, which ultimately results in an inhibition of calcium reabsorption in the distal renal tubule. Reducing dietary sodium has been shown to decrease urinary calcium excretion in hypercalciuric stone formers, while high dietary sodium is associated with both increased urinary calcium excretion and low bone density.

Sodium intake among stone formers is equal to or higher than intake in control groups of non-stone formers. Sodium and calcium share common sites for reabsorption in the renal tubules. Patients with recurrent nephrolithiasis and hypercalciuria also are the most sensitive to the hypercalciuric actions of a high-sodium diet. Finally, in postmenopausal women, high sodium intake has been directly associated with low bone