INTRODUCTION
CHAPTER - 1
INTRODUCTION

In Bundelkhand people experience patients with kidney stone disease, with 500 new cases each year and this is a comparative study with 500 renal stone patients and 300 samples of normal population. The percentage of people with hypercalciuria has been estimated out of 500 calcium stone patients (33.4%) were hypercalciuric, (24.8%) were male and (8.6%) were female whereas in normal population out of 300 samples (8%) were having Hypercalciuria (6%) were male and (2%) were female. People who form kidney stones, although some investigators have suggested that hypercalciuria can be found in as many as 65% of all calcium stone formers. It is the most common metabolic abnormality found with calcium nephrolithiasis. Despite a higher incidence of stone disease in the "stone belt," which primarily in the farmers and most common people who exposed to work in direct sunlight. Calcium stones are more common in men; the average age of onset is the third decade. Approximately 60% of people who form a single Calcium stone eventually form another within the next 10 years.

Dent and Sutor (1971) reported that crystals of Calcium Oxalate grow abnormally fast in the urines of patients with recurrent renal stone formation. This was found to be due to the urine containing reduced amounts of an inhibitor presents in all urines.

The data from Bundelkhand region, (including the kidney stone patients and healthy population 'their follow-up study) indicate that, with the current definition of hypercalciuria. The relative risk of
stone production appears to be continuous, with a single arbitrary level differentiating healthy people and those who form stones.

The most common types of clinically significant hypercalciuria are absorptive, renal leak, resorptive, and renal phosphate leak. Each of these conditions is described later. Other causes of hypercalciuria that need to be considered but are not discussed in this practical work, include hyperthyroidism, renal tubular acidosis, sarcoidosis and other granulomatous diseases, vitamin D intoxication, glucocorticoid excess, paget disease, Albright tubular acidosis, prolonged immobilization, multiple myeloma, lymphoma, leukemia, metastatic tumors especially to bone, addison disease, and milk-alkali syndrome.

Hypercalciuria contributes to kidney stone disease and osteoporosis, which explains the need to understand this disorder clearly. Most patients are recurrent stone formers.

Broadus AE et al (1984) reported that hypercalciuria (increased calcium in the urine) is an important risk factor for kidney for formation. Many patients with kidney stones are known to have intestinal hyperabsorption of calcium.

Internationally, overall risk of forming stones differs in various parts of the world. The probability is 1-5% in Asia, 5-9% in Europe, 13% in North America, and 20% in Saudi Arabia. Upper tract stone disease is associated with an affluent lifestyle in developed countries with diets high in animal protein, while bladder stones are predominant in developing countries and are related to poor socioeconomic conditions.
In 60-70% of patients, hypercalciuria will be present (defined by 24-hour urinary calcium excretion of >300 mg in males, >250 mg in females, or >4 mg/kg in males or females). In less than 5%, the hypercalciuria may be associated with hyperparathyroidism or sarcoidosis, with or without hypercalcemia. More often, the hypercalciuria occurs with a normal serum calcium and in the absence of any systemic diseases, and is called idiopathic hypercalciuria.

Parfitt et al (1964) Predisposes to the formation of calculi. An excretion of above 300 mg/24 hours in men and 250 mg /24 hours in women has been termed 'hypercalciuria'. This is present in about 8% of the healthy population in whom balance studies shows low faecal outputs of calcium.

CONTRIBUTING FACTORS OF STONE FORMATION

The main contributing factors that causes stones are age, sex, family history, climate, dietary factors and various disorders of the body:

1. **Age**: The peak age range for calcium kidney stone production generally is 20-60 years. Another peak incidence of hypercalciuria occurs in some postmenopausal women. In this older age group, many are taking supplemental calcium for osteoporosis prophylaxis. The excess absorbed calcium eventually is released into the urine. In addition, an increased risk of hyperparathyroidism exists in postmenopausal women, which can cause hypercalciuria.

In **children**: hypercalciuria often is associated with some degree of hematuria, back or abdominal pain, and, sometimes, voiding
symptoms.

**Pediatric hypercalciuria** is limited to dietary or short-term medical therapy because the patients become asymptomatic when the hypercalciuria is corrected and often are lost to follow-up. One recent study looked at the long-term effects of hypercalciuria in children and several possible therapies over a 4-year period, the majority of children with hypercalciuria eventually become asymptomatic even though they remain hypercalciuric. Because limiting calcium intake in children is unwise, the recommended dietary therapy for hypercalciuria is to use a low-sodium/high-potassium diet, which normalizes the hypercalciuria in the majority of pediatric patients.

**Adults:** A study involving only women demonstrated that women who develop calcium kidney stones had an average calcium intake that was 250 mg/d less than non-stone-forming women. This finding agrees with other studies that suggest that calcium stone formers should not restrict their calcium intake too aggressively.

**Postmenopausal women:** are more likely than men to demonstrate hypercalciuria. Hyperparathyroidism, which produces hypercalciuria, is more common in older women. Calcium supplementation also is more popular with women because of their concerns about possible osteoporosis.

**2. Geriatric stone disease:** is relatively uncommon. The risk for newly formed stones in patients older than 65 years is quite low, although once a stone has formed the number and type of risk factors, as well as the risk of recurrent stones, is similar to younger stone formers. The incidence of hyperparathyroidism in particular is higher in older persons and should be considered whenever an
older patient presents with a first calcium kidney stone, particularly if the patient is female.

3. **Obesity:** Urinary chemistry and stone formation rate data were analyzed with the demographic information from a large database of kidney stone formers. This research specifically compared stone formation rates with body weight for men and women and found that obesity is a risk factor for kidney stone disease in women but not in men. Investigators at Harvard conducted two studies found that body size was a positive risk factor for kidney stone disease in women, but the correlation was much less significant in men. The reason for this is unclear, but it may be related to estrogen levels. Whether this increased risk in women disappears when the excess body weight is lost also is unclear.

4. **Climate:** A hot climate contributes to the formation of both renal and vesical calculi, for excessive loss of water in sweating diminishes the volume of urine which then becomes more concentrated. The reference range of urinary calcium excretion for men generally is 275 mg or less per day, while in women the limit is only 250 mg/day. These reference values were created using large numbers of people (not calcium kidney stone formers) to establish a reference range. The most likely reason for the discrepancy is that men generally are larger physically than women and have a correspondingly larger amount of material, such as calcium and uric acid, to excrete.

5. **Dietary factors:** Several dietary factors besides calcium can contribute to stone formation and causes hypercalcuiuria. These include animal protein, sodium, alcohol, caffeine, refined carbohydrates, fiber, oxalate, and fluids.
6. **Family history:** This study also involving children at recurrent abdominal and flank pain associated with hypercalciuria. A family history of kidney stone disease was present in some children. Resolution of the hypercalciuria eliminated the recurrent pain in this patient population. Although the study had a relatively small number of subjects, it still suggested some important differences in the etiology of stone disease between normal population and kidney stone formers.

**STONE FORMATION - TYPES OF STONES AND THEIR CAUSES**

The tendency to form kidney stones is usually inherited. Most normal people form small crystals of calcium phosphate, magnesium phosphate, calcium salts, uric acid, cystine and struvite are the basic constituents of most kidney stones. Calcium oxalate and calcium phosphate stones made up of 75% to 85% of the total stones and may be admixed in the same stone. Calcium phosphate stones are usually hydroxyapatite \( \text{Ca}_6(\text{PO}_4)_3\text{OH} \) or, less commonly brushite \( \text{CaHPO}_4 \cdot \text{H}_2\text{O} \).

**1. Calcium oxalate and phosphate stones** - 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 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 currently made up of calcium oxalate and phosphate. Calcium oxalate is often mixed with phosphate, but either pure calcium oxalate or calcium phosphate
stones may occur.

**Pathophysiology of Calcium Stones:**

In 60-70% of patients, hypercalciuria will be present (defined by 24-hour urinary calcium excretion of >300 mg in males, >250 mg in females, or >4 mg/kg in males or females). More often, the hypercalciuria occurs with a normal serum calcium and in the absence of any systemic diseases, and is called idiopathic hypercalciuria. Most patients with idiopathic hypercalciuria exhibit excessive gastrointestinal absorption of calcium (absorptive hypercalciuria). In many such cases, 1,25-vitamin D levels are slightly elevated and serum phosphorous is slightly low but parathyroid hormone levels are normal. The degree of hypercalciuria is worsened by high dietary sodium intake, high animal protein intake, and loop diuretics; it is reduced by 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.

**OTHER RISK FACTORS FOR CALCIUM STONES INCLUDE:**

1. Hypercalciuria (excessive urinary excretion),
2. Chronic low urine output,
3. Low urine citrate, which occurs most often in patients with inflammatory bowel disease, chronic metabolic acidosis, and renal tubular acidosis (RTA). Renal stones occur in the distal form of RTA, are frequently composed of calcium phosphate, present as multiple stones on radiography (nephrocalcinosis), and occur in the presence of a persistently alkaline urine (pH >5.5) despite metabolic acidosis.
SIGNS AND SYMPTOMS:

Patients often present with episodes of flank pain that radiates to the anterior abdomen or even to the genitalia. The pain is often severe and comes in waves. Often there is microscopic or gross hematuria. Calcium oxalate crystals may be seen with urine microscopy. In some patients the renal stones are completely asymptomatic or may produce painless hematuria.

Hypercalciuria, or excessive urinary calcium excretion, is the most common identifiable cause of calcium kidney stone disease. (The other significant causes are hyperoxaluria, hyperuricosuria, low urinary volume, and hypocitraturia.).

Hypercalciuria is defined as urinary excretion of more than 250 mg of calcium per day for women or more than 275-300 mg of calcium per day for men while on a regular, unrestricted diet. It also can be defined as the excretion of urinary calcium in excess of 4 mg/kg of body weight per day. An alternate definition is daily urinary excretion of more than 3 mg of calcium per kg of body weight or more than 200 mg of calcium per day while on a restricted (400 mg calcium and 100 mEq sodium) diet.

DIAGNOSIS OF CALCIUM STONE:

Stone analysis is the surest way to diagnose calcium oxalate or calcium phosphate stones. Calcium-containing stones are radiopaque on routine radiography but show up as bright objects on computed helical tomography without contrast. Ultrasonography will detect all types of renal stones if the stone is larger than 3 to 5 mm and the ultrasound is technically satisfactory. At the present time,
helical computed tomography without contrast is the procedure of choice for the initial radiographic investigation. All types of stones located anywhere in the kidneys, ureters, or bladder will be demonstrated with this technology. In addition, the anatomic status of the urinary tract will be clarified and other possible non-stone causes for the patient's symptoms or signs may be identified. Of the conditions associated with calcium stones, only pyelotubular ectasia (medullary sponge kidney) is better demonstrated by intravenous urography.

SHAPE, SIZE AND COLOUR OF THE STONES & TYPE OF RENAL CALCULUS:

(1) **Calcium Stones**: Calcium usually combines with other substances to form stones, most often with oxalate and phosphate. Calcium oxalate is the most common form. Calcifications can occur and accumulate in the collecting system resulting in nephrolithiasis. 80–85% of all urinary stones are calcerous.

(i) **Calcium Oxalate (Oxalate Calculus)**: Approximately 80% of cases. These stones are irregular in shape and converted with sharp projections which tend to cause bleeding. In the urine, oxalate monohydrate crystals (whewellite) usually grow as biconcave oval shaped that resembles red blood cells in shape and size but may occur in a larger, "dumbbell" form. In polarized light the crystals appear bright against a dark background, with an intensity that is dependent on orientation, a property known as birefringence. The surface of the calculus is discoloured by the pigments of altered blood. Calcium oxalate monohydrate stone is very hard and absorbs X-rays well, it is easy to see radiologically. Calcium oxalate dihydrate
crystals (wheddellite) are bipyramidal. Apatite crystals do not exhibit birefringence and appear amorphous because the actual crystals are too small to be resolved by light microscopy. Oxalate is a byproduct of metabolism. It is also found in a number of foods.

(ii) Calcium Phosphate (Phosphate Calculus): Usually calcium phosphate, although sometimes combined with ammonium magnesium phosphate (Struvite) is smooth and dirty white. These stone tends to grown in alkaline urine especially when proteus organisms are present which split urea to ammonium. As a result, the calculus may enlarge to fill most of the renal collecting system forming a staghorn calculus.

(2) Struvite Stones (infection stones): Magnesium ammonium phosphates (struvite) stones almost always occur in patients with a persistently alkaline urine, owing to urinary tract infections. In particulars the urea splitting bacteria such as Proteus Vulgaris and the Staphylococci, predispose the patients towards urolithiases. These stones are often large and have a characteristic stag's horn shape that can seriously damage your kidneys. Struvite formations are more common in women than men, mainly because the female urinary tract system is more susceptible to infections. The stones are jagged, and can reach large sizes. "Staghorn" calculi are a form of struvite deposit that grows very large, and can fill the kidney cavity.

(3) Uric acid stones: These are hard, smooth, and often multiple. Their colour varies from yellow to reddish to brown and sometimes have an attractive multifaceted appearance. Pure uric acid stones are radiolucent appear on an excretion urogram as a filling defect, which can be mistaken for a transitional tumour of the
<table>
<thead>
<tr>
<th>Stones</th>
<th>Percentage of all Stones</th>
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<tbody>
<tr>
<td><strong>1. Calcium Oxalate (Phosphate)</strong></td>
<td>75</td>
</tr>
<tr>
<td>- Idiopathic hypercalciuria (50%)</td>
<td></td>
</tr>
<tr>
<td>- Hypercalcemia &amp; Hypercalciuria (10%)</td>
<td></td>
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<tr>
<td>- Hyperoxaluria (5%)</td>
<td></td>
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<tr>
<td>- Enteric (4.5%)</td>
<td></td>
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<tr>
<td>- Primary (0.5%)</td>
<td></td>
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<tr>
<td>- Hyperuricosuria (20%)</td>
<td></td>
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<tr>
<td>- No known metabolic abnormality (15% - 20%)</td>
<td></td>
</tr>
<tr>
<td><strong>2. Struvite (Mg, NH₃, Ca, PO₄)</strong></td>
<td>10 - 15</td>
</tr>
<tr>
<td>- Renal infection</td>
<td></td>
</tr>
<tr>
<td><strong>3. Uric acid</strong></td>
<td>6</td>
</tr>
<tr>
<td>- Associated with hyperuricemia</td>
<td></td>
</tr>
<tr>
<td>- Associated with hyperuricosuria</td>
<td></td>
</tr>
<tr>
<td>- Idiopathic (50% of uric acid stones)</td>
<td></td>
</tr>
<tr>
<td><strong>4. Cystine</strong></td>
<td>1 - 2</td>
</tr>
<tr>
<td><strong>5. Xanthine, silicate</strong></td>
<td>+10</td>
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urinary tract. These are soft and fiable. These are radiolucent unless they are contaminated with calcium salts. Uric acid stones are formed of uric acid, a byproduct of protein metabolism. These stones are produced when nucleic acids (the building blocks of DNA) are recycled, with very low urine pH (below pH 5.0).

(4) **Cystine stones:** These are uncommon and appear in the urinary tract of patients with a cognitor error of metabolism which leads to cystinuria. Cystine crystals are hexagonal translucent white and appear only in acid urine. Cystine stones are often multiple and may grow to form a cast of renal pelvis and calyces. These are pink or yellow when first removed but they change colour to a greenish hue when exposed to air. Cystine stones are radio opaque because of the sulphur that they contain and these stones are very hard. Only one to two percent of calculi are formed from cystine, an amino acid that is found in protein. Most cases stem from cystinuria. Cystinuria is a rare congenital disorder that produces unusually high levels of cystine. Stones formed due to cystinuria occur throughout life.

These stones represent only about 1% of kidney stones. They form in people with a hereditary disorder that causes the kidneys to excrete excessive amounts of certain amino acids (cystinuria).

(5) **Xanthine and Silicon:** These are very rare stones.

(6) **Urinary Bladder or Vesical Stones:** These stones usually occur in males; in adults they are generally associated with prostatic obstruction or other causes of urinary stagnation. Stagnation and infection of urine and prolonged confinement to bed, each
predispose to stone formation. Epidemiological evidence suggests that a single cereal diet (wheat, rice or millet) may be a significant causative factor of endemic bladder calculi.

ROLE OF CALCIUM IN THE BODY:

Calcium is the fifth most abundant mineral element in the human body. The total amount of calcium in body is about 1000-1200mg, 98% of which is present in skeleton in the form of hydroxyappetite, which is a crystal lattice composed of calcium, phosphorus and hydroxide, held in a cellular matrix provide the hard structure of the bones and teeth. Half part of remaining calcium is present in extracellular fluid and remaining present in skeletal muscles. Besides this much amount of $Ca$ is present in our body, only 1% of total skeletal reservoir of $Ca$ is readily exchangeable with extracellular fluid. The amount of calcium in the extracellular fluids and soft tissues of a normal adult does not exceed 10g. Obviously all of this calcium comes from the diet. Among common foods, milk is much the richest source, which is one reason why milk and cheese are especially valuable for growing children. Half a litre (just under a pint) of cow's milk contains about 0.6 g of calcium. Most other foods contribute much smaller amounts. The calcium retained in the normal human skeletal has therefore been extracted from a very large volume of food, even when the diet has contained abundant milk and cheese. Calcium has an important role in determining the excitability of peripheral nerves and muscles. Calcium plays vital role in:

1. Basic physiologic processes as blood coagulation - The amount of calcium in the plasma is about 10mg/100ml or 4.5mEq/
litre. About half of this amount is bound to the serum albumin and half is in ionised solution. If the level of albumin in the serum is lower than normal in consequence of protein deficiency, there is a corresponding reduction in the level of protein-bound calcium. This produces no metabolic disturbance in marked contrast to the effects of a reduction in the ionised portion of serum calcium.

2. **Neural transmission and maintainence of normal tone and extitability of skeleton and cardiac muscles** - The motor nerves become over susceptible to stimuli. This particularly affects the face, hands and feet, producing the spasms known clinically as tetany. Muscles lose tone and become flaccid, so that infants with rickets are late in sitting, standing and walking. The isolated animal heart ceases to beat when transfused with fluid lacking calcium; however, there is no evidence that calcium deficiency in the myocardium is ever a cause of heart failure in man. Calcium salts provide rigidity to the skeleton and calcium ions play a role in many if not most metabolic processes. In the primitive exoskeleton and in shells, rigidity is generally provided by calcium carbonate, but in the vertebrate skeleton it is provided by a form of calcium phosphate which approximates hydroxyapatite[Ca10(OH)2(PO4)6] and is embedded in collagen fibrils.

3. **Enzyme activity** - The enzyme rennin is dependent on calcium for its activity. This enzyme is secreted by the stomach of young mammals and causes the curdling of milk (i.e. clotting of caseinogen) as a preliminary to digestion. The ionisation of calcium can be suppressed by sodium citrate. This is sometimes added to cow's milk for infant and invalid feeding in the mistaken belief that, by
inhibiting the normal curdling of caseinogen in the stomach, the milk will be rendered more digestible.

4. **It regulates the transport of Sodium and Potassium across the cell membrane** - In a normal man the extracellular fluids contains just under $2175\text{mWq}$ of Na and the intracellular fluids about $375\text{mEq}$. In addition there are some $900\text{mEq}$ present in the bônes, making a total of about $3450\text{mEq}$ (200g of NaCl) in the whole body. The total exchangeable sodium in adult men and women are found to vary from 1880 to 3140mEq. Only about 25% of the Sodium in bone is exchangeable, and most is held in a chemical form which is unavailable as an immediate reserve. It has been known at least since 1961 that urinary calcium is related to urinary sodium and that sodium administration raises calcium excretion, presumably because sodium competes with calcium for reabsorption in the renal tubules. Regarding the quantitative relationships between the renal handling of sodium and calcium, the filtered load of sodium is about 100 times that of calcium (in molar terms) but the clearance of these two elements is similar at about 1 ml/min, which yeilds about 99% reabsorption and 1% excretion for both. However these are approximations, which conceal the close dependence of urinary sodium on sodium intake and the weaker dependence of urinary calcium on calcium intake. It is an empirical fact that urinary sodium and calcium are significantly related in to normal and hypercalciuric subjects on freely chosen diets. By contrast, the Potassium content of the extracellular fluids is only about $80\text{mEq}$, whereas the tissues contain about $3500 \text{mEq}$. The store of Potassium in the bones is negligible. Potassium is an important electrolyte involved in nerve
transmission and the contraction of all muscles including the heart. Potassium is essential for maintaining normal blood pressure and heart function. A one cup of cow’s milk provides 10.8% of the daily value for potassium.

5. It maintains the integrity of cell membrane - Calcium also plays an important role in cell membrane function and blood pressure regulation. Because these activities are essential to life, the body utilizes complex regulatory systems to tightly control the amount of calcium in the blood, so that sufficient calcium is always available. As a result, when dietary intake of calcium is too low to maintain adequate blood levels of calcium, calcium stores are drawn out of the bones to maintain normal blood concentrations. Vitamin D ensures calcium availability, although typically categorized as a fat soluble vitamin, vitamin D actually functions more like a hormone than a vitamin. Calcitriol, the most metabolically active form of vitamin D, works with parathyroid hormone (PTH) to maintain proper levels of calcium in the blood. In addition, calcitriol participates in the regulation of cell proliferation, differentiation, and growth which suggests that vitamin D may play a role in the prevention and treatment of various cancers. A cup of cow’s milk supplies 24.4% of the daily value for this important vitamin. The vitamin K provided by cow’s milk is also important for maintaining strong bones. Vitamin K1 activates osteocalcin, the major non-collagen protein in bone. Osteocalcin anchors calcium molecules inside the bone. Therefore, without enough vitamin K1, osteocalcin levels are inadequate, and bone mineralization is impaired. A cup of cow’s milk provides 12.2% of the daily value for vitamin K. It’s not just calcium
and vitamin K that makes milk a bone-friendly food, cow's milk and fermented milk products such as yogurt also contain lactoferrin, an iron-binding protein that boosts the growth and activity of osteoblasts (the cells that build bone). Not only does lactoferrin increase osteoblast differentiation, it also reduces the rate at which these cells die by up to 50-70% and decrease the formation of osteoclasts (the cells responsible for breaking down bone) thus helping to prevent or reverse osteoporosis. In addition, lactoferrin also increases the proliferation of chondrocytes, the cells that build cartilage. For building bone, enjoying both milk and yogurt seems a good idea since lactoferrin’s effects were found to be dose dependent, stimulating an up to a 5-fold increase in osteoblasts at higher doses. Cow's milk is a good source of low-cost high-quality protein, providing 8.1 g of protein (16.3% of the daily value for protein) in one cup. The structures of humans and animals are built on protein.

Daily intake of dietary Calcium varies from 150-200mg/day, major parts of which is derived from milk and dairy products. In both adults, male and female minimum daily dietary requirement of Calcium is 400mg/day. During pregnancy and lactation 1000 mg/day, during childhood 400-600 mg/day calcium is absorbed by active transport process that occurs in duodenum and upper jejunum. The absorbed calcium becomes the part of blood. The normal serum calcium level 9.1-10.5mg% (4.5-5.0m eq/L).

1. Calcium exist in serum (plasma) in three distinct forms, free or ionised calcium which is physiologically active form accounts for 50% of total calcium.

2. About 5% of total Calcium is composed with variety of anions
particularly phosphate and citrate.

3. The remaining 95% of Calcium is bound to plasma proteins especially to albumin. Both ionised calcium and the calcium complexes are freely dialyzable. The relative distributions of the three forms altered as a result of change either in pH of the extracellular fluids or in the protein concentration. Acidosis promotes an increase in ionised calcium where alkalosis causes a corresponding decrease.

Calcium Metabolism.
MAINTANANCE OF CALCIUM HOMEOSTASIS INVOLVES THE PARTICIPATION OF THREE MAJOR ORGANS:

1. **Small intestine** - Ingested calcium mixes with digestive juice calcium in the proximal small intestine from where it is absorbed by a process, which has an active saturable component and a diffusion component. At low calcium intakes calcium is mainly absorbed by active (transcellular) transport, but at higher intakes an increasing proportion of calcium is absorbed by simple (paracellular) diffusion. The unabsorbed component appears in the faeces together with the unabsorbed dietary calcium and unreabsorbed digestive juice calcium. True absorbed calcium is the total calcium absorbed from the calcium pool in the intestines and therefore contains both dietary and digestive juice components. Net absorbed calcium is the difference between dietary calcium and faecal calcium and is numerically the same as true absorbed calcium minus endogenous faecal calcium. Many factors influence the availability of calcium for absorption and the absorptive mechanism itself. The former includes substances, which form insoluble complexes with calcium, such as the phosphate ion. The relatively high calcium-phosphate ratio of 2.2 in human milk compared with 0.77 in cow milk may be a factor in the higher absorption of calcium from human milk than cow milk. Intestinal calcium absorption is mainly controlled by the serum concentration of 1,25(OH)2D. The activity of the 1α-hydroxylase, which catalyses 1,25(OH)2D production from 25-hydroxycalciferol (23OHD) in the kidneys, is negatively related to the plasma calcium and phosphate concentrations and positively to plasma parathyroid hormone.
2. The kidneys - Urinary calcium is the fraction of the filtered plasma water calcium, which is not reabsorbed in the renal tubules. At a normal glomerular filtration rate of 120 ml/min and ultrafiltrable calcium of 6.4 mg/100 ml (1.60 mmol/l), the filtered load of calcium is about 8 mg/min (0.20 mmol) or 11.6 g/day (290 mmol/day). Because the usual 24-hour calcium excretion is about 160-200 mg (4-5 mmol), it follows that 98-99% of the filtered calcium is usually absorbed in the renal tubules. However, calcium excretion is extremely sensitive to changes in filtered load. A decrease in plasma water calcium of only 0.17 mg/100 ml (0.043 mmol/l), which is barely detectable, was sufficient to account for a decrease in urinary calcium of 63 mg (1.51 mmol) with low calcium diet. This very sensitive renal response to calcium deprivation combines with the inverse relationship between calcium intake and absorption to stabilise the plasma ionised calcium concentration and to preserve the equilibrium between calcium entering and leaving the ECF over a wide range of calcium intakes. However, there is always a significant obligatory loss of calcium in the urine (as there is in the faeces), even on a low calcium intake, simply because maintenance of the plasma ionised calcium and, therefore, of the filtered load, prevents total elimination of the calcium from the urine.

3. The skeleton - Bone contains a living cellular matrix on which the minerals are deposited. Its chemical composition is approximately water 25-30%, protein 20%, fat 5%, small amounts of mucopolysaccharide and nearly 50% mineral. Most of the mineral is the calcium salts, hydroxyapatite $\text{Ca}_{10}(\text{PO}_4)_6(\text{OH})_2$, but there are small and variable amounts of magnesium, sodium carbonate and
citrate. Calcium salts provide rigidity to the skeleton and the calcium ions plays a role in many, if not most metabolic processes. In the primitive exoskeleton and in shells, rigidity is generally provided by calcium carbonate, but in the vertebrate skeleton it is provided by a form of calcium phosphate. Nearly 200 years ago, John Hunter showed that the architecture of the bones is completely reorganised during the process of growth; no single strut or brace of bone remain fixed and permanent in any part of the body. Two different kinds of cells are constantly altering the structure of the bones, even in adult life. While the osteoblasts lay down fresh calcium salts, especially where new stresses have developed, osteoclasts are constantly eroding redundant calcium deposits. A well-adjusted equilibrium exists between the calcium coming and going from the bones and the calcium present in small but normally constant amounts in the fluids of the body. In an adult man about 700 mg of calcium enter and leave the bones daily (Whedon, 1964). Thus the bones not only provide physical support to the body but also a large reserve of two essential elements - calcium and phosphorus - on which the body can draw in case of need.

Minor contribution cover in favour of mammary glands, placenta and sweat glands. During growth and pregnancy a tissue calcium balance must be maintained. Various hormones regulate homeostasis of calcium in health. Important are parathyroid hormones and 1,25 hydroxycholecalciferol derived from renal metabolism of vitamin D3. Calcitonin plays an important role in regulating the process. Thyroid hormone, growth hormone, adrenal glucocorticoids and gonadal steroids also play some role in calcium
The excretion of calcium takes place by sweat, faecal and urine. In sweat, 15-100 mg per day can loose. The major net lose of calcium is urinary excretion which accounts for 50-200 mg or more each day. Urinary calcium excretion is enhanced by hypercalcemia, phosphate deprivation, acidosis, and glucoexcretion. Urinary calcium also rises very slowly with intake (slope of 5-10%) and the risk of kidney stones from dietary hypercalciuria must therefore be negligible. In fact, it has been suggested that dietary calcium may protect against renal calculi because it binds dietary oxalate and reduces oxalate excretion.

The crystals of Calcium Oxalate grow abnormally fast in the urines of patients with recurrent renal stone formation. This was found to be due to the urine containing reduced amounts of an inhibitor presents in all urines (Dent and Sutor, 1971). Although urinary calcium must rise with the growth-related rise in glomerular filtration rate. Urinary and endogenous faecal calcium are not only the forms of excreted calcium; losses through skin, hair and nails need to be taken into account. These are not easily measured, but a combined balance and isotope procedure has yielded estimates of daily insensible losses in the range of 40-80 mg (1-2 mmol) which are unrelated to calcium intake.

Urinary calcium excretion is decreased by parathyroid hormone, certain diuretics and vitamin D.

**MANIFESTATIONS OF STONES:**

As stones grow on the surfaces of the renal papillae or within the collecting system, they need not produce symptoms.
Asymtomatic stones may be discovered during the course of radiographic studies undertaken for unrelated reasons. Stones rank, along with benign and malignant neoplasms, renal crystals and genito-urinary tuberculosis, among the common causes of isolated hematuria. Much of the time, however stones break loose and enter the ureter or occlude the ureteropelvic function, causing pain and obstruction.

STONE PASSING

A stone can transverse the ureter without symptoms, but passage usually produces pain and bleeding. The pain begins gradually, usually in the flank, but increases over the next 20 to 60 min. to become so severe that narcotic drugs, may be needed for its control. The pain may remain in the flank or spread downward and anteriorly toward the ipsilateral loin, testis or vulva that migrates downward indicates that the stone has passed to the lower third of the ureter, but if the pain does not migrate, the position of the stone cannot be predicted. A stone in the portion of the ureter within the bladder wall causes frequently urgency and dysuria that may be confused with urinary tract infection. The vast majority of ureteral stones less than 0.5 cm in diameter will pass spontaneously. An impacted stone may lead to hydroureter, pyelonephritis and in case of bilateral renal stone features of obstructive uropathy.

PATHOGENESIS OF STONES:

Urinary stones usually arise because of the breakdown of a delicate balance. The kidneys must conserve water, but they must
excrete materials that have a low solubility. These two opposing requirements must be balanced during adaptation to diet, climate and activity.

The problem is mitigated to some extent by the fact that urine contains substances that inhibit crystallization of calcium salts and other that bind calcium in soluble complexes. These protective mechanisms are less than perfect. When the urine becomes supersaturated with insoluble materials, because excretion rates are excessive and / or because water conservation is extreme, crystals form and may grow and aggregate to form a stone.

STONE FORMATION AND ITS RELATIONSHIP WITH DIET

Most stones (about 75%) are calcium containing, composed largely of calcium oxalate or calcium oxalate mixed with calcium phosphate and Calcium carbonate. 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 in concert. A supersaturation of urine with the stone forming salt (s) is of fundamental importance and a prerequisite for the necessary precipitation. The solubility of the different stone components depends on the urinary pH and the excretion of other urine constituents. Accordingly, a saturation of urine above the solubility product (SP) and the associated risk of crystallization are determined by the urinary concentration (mmol/l) of the solutes taking part in the crystal formation and the pH. When the SP has been exceeded, the supersaturation is metastable. At this level of supersaturation the crystals can grow and aggregate
(agglomerate) but new crystal formation is not possible. In order to start the formation of new crystals, the supersaturation has to be further increased to a level termed the formation product (FP). The isolated findings of increased concentrations are not diagnostic for stone disease but might reflect only the concentration capacity of the kidney. Normal values of urine constituents are usually expressed as the total excretion during a 24h period or a fraction of a 24h period. Mostly stones are formed readily in infected urine in which bacteria have converted urea into ammonia, so raising the pH and thereby making the phosphates less soluble. Calcium Oxalate is often present in mixed stones and about 75% of stones are pure Calcium Oxalate. Only about 10% of stones do not contain Calcium and these are nearly all composed of uric acid. Crystals of Calcium Oxalate are present in all urines and are probably the result of precipitation from a supersaturated solutions of these ions.

The crystals are small and normally washed out when the urine is voided. Various factors in the urine may inhibit the precipitation and prevent the rapid growth of crystals. Citric acid is present in urine in molar concentration comparable to that of Calcium. As it binds Calcium ions to form a soluble complex, citrate might allow more free Calcium ions to pass into solution. Crystals of Calcium Oxalate grow abnormally fast in the urines of patients with recurrent renal stone formation. This was found to be due to the urines containing reduced amounts of an inhibitor present in all urines (Dent and Sutor, 1971).

Although there are many causes for the initiation and propagation of stones, the most important determinant is an increased urinary
concentration of stones constituents, such that is exceeds their solubility (defined as solubility product SP) in urine. A low urinary volume in some metabolical normal patients may also favour supersaturation, due to increased urinary concentration of stone constituents.

Calcium oxalate and calcium phosphate form many stable soluble complexes among themselves and with other substances in urine, such as citrate. As a result their free ion activities are below their chemical concentration and can be measured only by indirect method. Reduction in concentration of ligands such as citrate can increase ion activities without changing total urinary calcium. Secondly urine supersaturation can be increased by dehydration or by overexcretion of calcium oxalate and phosphate. Urine pH is also important as various salts differ in their solubility over a range of pH. Calcium stones are favoured by very low urinary pH. Calcium oxalate stones are association in about 33.4% patients with hypercalciuria and about 15% hypercalcemia, another 55% patients have hypercalciuria without hypercalcemia, this is caused by several factors including hyperabsorption of calcium from GIT (absorptive hypercalciuria) an intrinsic defect in renal reabsorption of calcium in renal tubules (Renal Hypercalciuria) or idiopathic fasting hypercalciuria in presence of normal parathyroid function. Hypercalciuria implies that 24 hour urinary calcium excretion on a free diet is higher than normal. The normal range of urinary calcium, however, varies according to geographical region and a particular definition of normality therefore applies only to specific population groups.
An accepted definition of hypercalciuria is the excretion of >300mg/day in women and 400 mg/day in men or 4mg/kg/day of calcium in either sex when the patient is screened on a defined 2000 mg/day calcium intake. Idiopathic hypercalciuria is present in about 5% of the healthy normal population. Idiopathic hypercalciuria tends to run in families, and the absorptive type has been classified into two forms. In both there is a normal or low parathormone level, fasting urinary calcium levels below 0.11mg/dl creatinine clearance and excess calcium absorption with a rise in the urinary value to over 0.2mg/dl after a 1g oral calcium load in synthetic meal.

**DIETARY CALCIUM:**

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 to 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. However, among patients who took calcium supplements there was actually 5% increased risk of developing kidney stones. It may be that dietary calcium in whole foods usually reduces oxalate absorption but calcium supplements may not always reduce oxalic acid absorption. Calcium supplements that are not taken with oxalic acid rich foods and not be expected to decrease oxalic acid absorption and excretion. It seems discourage that the use of high-dose calcium supplementation in nearly all patients with recurrent 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. Patients who do not consume sufficient dietary calcium from foods, one may be forced to weigh the possible reduction of osteoporosis risk against a possible increased risk of kidney stone formation from calcium supplementation. Calcium supplement is deemed more beneficial than risky, it seems prudent to use one that contains calcium citrate and as the citric acid helps to keep the calcium oxalate crystals from forming in the urine. Avoidance of an excessively high-calcium diet is an obvious recommendation for calcium stone formers. Stone formers as a group are much more sensitive to dietary calcium than non-stone formers. Calcium from food sources is absorbed during digestion in the intestines. The body uses this mineral for many important functions. Any excess that has been absorbed is excreted or passed through the kidneys. The biggest portion of calcium in the diet comes from milk and foods made from large amounts of milk, such as cheeses and yogurt. The calcium in these foods is usually easily absorbed. Other foods, such as dark green leafy vegetables, contain significant amounts of calcium.

However, they also contain other substances which prevent the body from readily absorbing the calcium. So, the amount of available calcium in green leafy vegetables is less than in milk. Certain antacids and over-the-counter medications also contain calcium that may or may not be in a form the body can absorb. Usually the body does
not absorb more calcium than is needed. However, certain conditions
can cause too much calcium to be absorbed, or too much to be
passed into the kidneys. Too much calcium in the urine is medically
known as hypercalciuria. Only in certain cases of hypercalciuria may
calcium stones be prevented controlling the amount of calcium in
the diet.

Relationship with hypercalciuria to dietary calcium intake is
unsettled. Many researcher's has claimed a positive relationship
with calcium intake and hypercalciuria and other researcher's
founded no relationship between the calcium intake.

**OXALIC ACID OR OXALATE:**

It is found mostly in foods from plants. Calcium combines with
oxalate in the intestines. This reduces calcium's ability to be
absorbed. Sometimes oxalate or calcium oxalate stones form
because there is not enough calcium in the intestines. Then, too
much oxalate goes to the kidneys to be excreted. The medical term
for too much oxalate in the urine is hyperoxaluria. In certain cases
of oxalate or calcium oxalate stones, the recommend reducing
oxalate intake along with a slight increase in calcium. It is
recommended that these patients have no more than 50 mg of
oxalate per day in the diet. To do this, foods 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,
eight foods have been shown to be most at fault for raising urine
oxalate levels. They are rhubarb, spinach, strawberries, chocolate,
wheat bran, nuts, beets, and tea.

Avoiding a diet that is too severely limited in calcium also is
important because of the risk of a reactive hyperoxaluria and the creation of a negative calcium balance with subsequent osteopenia or actual osteoporosis. In 2 large population studies involving both men and women, patients with the highest daily calcium intake were demonstrated to have significantly fewer stones (within reasonable limits) than those with the lowest dietary calcium levels.

Proportionately, oxalate is 15 times stronger than calcium in promoting nephrolithiasis. The net stone formation rate may actually increase if dietary oxalate intake and hyperoxaluria are not controlled. Calcium citrate is recommended if calcium supplements are needed. This combination has been shown to be the most effective in limiting the new stone formation rate for those who require calcium supplements.

**Roman Smith H et al (1993)** Since calcium oxalate is responsible for most kidney stones, avoiding foods containing oxalate may be helpful.

**SODIUM:**

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. In healthy adults, a high sodium intake has been associated with higher fractional intestinal calcium absorption as well as increased PTH and vitamin D-3 levels. 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 density in calcium stone formers.

Most experts recommend limiting dietary sodium (salt) in calcium stone formers to about 100 mEq/d, but this is difficult because salt (sodium) enhances the taste of food to many people. Patients should be aware that most restaurant meals and fast food items, such as pizza, contain a considerable amount of sodium. Many prepared foods have low-sodium varieties available. Ketchup, mustard, teriyaki, and soy sauces all have large amounts of sodium, as well as canned soups, cold cuts, vegetables, and TV dinners.

Kenney JJ (1986) - reported that excessive salt intake increases the risk of kidney stones, osteoporosis, hypertension, stroke, kidney failure and heart disease and also may promote stomach and kidney cancer.

**POTASSIUM:**

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 higher sodium-to-potassium ratio than non-stone formers.

Potassium decreases urinary calcium excretion due to an induced transient sodium diuresis resulting in a temporary contraction of the extracellular fluid volume and an increase in renal tubular calcium reabsorption. Potassium also increases renal phosphate absorption, raising serum phosphate levels, which reduces serum vitamin D-3, resulting in decreased intestinal calcium absorption.

Ettenger B et al (1997) showed that potassium magnesium citrate may be more effective than potassium citrate alone.

**ANIMAL PROTEIN:**

High dietary protein intakes are known to increase urinary calcium excretion and, if maintained, will result in sustained hypercalciuria. A diet high in animal protein affects certain minerals in the urine that may promote the formation of kidney stones. Therefore, people who tend to develop kidney stones should avoid eating more protein than the body needs each day. The possible link between high animal protein intake and kidney stones has been known since at least 1973. This link has been found in epidemiological studies first in India, then in England, Germany, Austria, Japan, Italy, and, finally, in the United States. A large prospective study in the United States found a significantly increased risk of stones in the group with the highest animal protein intake. Additionally, known stone formers appear to be more sensitive to the stone-enhancing effects of high-animal protein diets than non-stone forming control populations.

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. Animal protein also increases the body's acid load directly. Methionine and cystine are more common in animal protein than plant protein. Both methionine and cystine contain relatively high levels of sulfur. When the sulfur is oxidized to sulfate, additional acid is generated. Sulfate also can form a soluble complex with calcium in the renal tubules, which can reduce calcium reabsorption and contribute to hypercalciuria. Urinary sulfate levels can be used as a general marker of oral animal protein intake. Normal levels generally are 40 mg/d or less, while optimal levels in calcium stone formers would be below 25-30 mg daily.

Dietary animal protein intake should be less than 1.7 g/kg of body weight per day. High protein intake has been judged second only to vitamin D ingestion in its ability to increase intestinal calcium absorption. Other effects of a high-animal protein diet include increased urinary oxalate and uric acid, as well as reduced urinary citrate.

Robertson WG et al (1979) reported that both an increase in oxalate and calcium in the urine can result from increasing dietary protein intake will usually increase the TSI. As a result, increased dietary protein will likely contribute to kidney stone formation. The risk of kidney stone formation seems greater from animal than vegetable proteins.

An intriguing 1996 randomized study compared the stone production rates in about 100 known calcium oxalate stone formers who differed in their dietary protein and fiber intakes (Hiatt, 1996).
The first group was instructed just to increase fluid intake, while the second group was told to increase fluid intake and consume a high-fiber, low-animal protein diet. These groups were observed for 4.5 years. The researchers found significantly fewer stones in the group with the high-fiber, low-animal protein diet. Of course, the possibility exists that the fiber or just the combination of the high fiber and animal protein restriction was effective.

Finally, a link between high animal protein ingestion and increased oxalate excretion may exist. For example, glycolate is an oxalate precursor whose generation is highly linked to animal protein intake. While some investigators have found a link between high animal protein intake and increased urinary oxalate, others have not.

**CARBOHYDRATES (REFINED):**

Several large population studies have investigated the issue of the potential contribution of a high-carbohydrate diet to stone production. For example, Curhan found that carbohydrates were not a significant risk factor for stone formation in men but were associated with an increased stone production in women.

Good evidence indicates that a high-carbohydrate diet causes an increase in urinary calcium excretion due to decreased distal renal tubular calcium reabsorption and an increase in intestinal calcium absorption. 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. Glyoxalate not only is involved in the metabolism of endogenous oxalate but also is involved in urea and the gluconeogenesis
pathway.)

**FIBER:**

Fiber is the indigestible part of plants. There are two types of fiber: soluble (dissolves in water) and insoluble. Both provide important functions in the body, but it is insoluble fiber (found in wheat, rye, barley, and rice) that may help to reduce calcium in the urine. It combines with calcium in the intestines, so the calcium is excreted with the stool instead of through the kidneys.

Insoluble fiber also speeds up movement of substances through the intestine, so there will be less time for calcium to be absorbed. Calcium stone formers as a group have a lower intake of dietary fiber than healthy control population. Dietary fiber, including oat, wheat, and rice bran, can reduce hypercalciuria and lower intestinal calcium absorption by 20-30%. As much as 24 g of dietary fiber per day may be necessary. Wheat bran, for example, is rich in oxalate, which accounts in part for its ability to bind and absorb free intestinal calcium. Although no reports of significant problems with increased dietary fiber have been made, some potential risks exist.

Dietary fiber may reduce intestinal magnesium, resulting in a deficit. Patients on a very high-fiber diet should be checked periodically for magnesium deficiency. A magnesium supplement, such as magnesium oxide, can be added if necessary.

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<tr>
<th>Foods Rich in Calcium, Phosphates, Oxalates &amp; Purines</th>
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<tr>
<td><strong>Calcium</strong></td>
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<td>Leafy vegetables</td>
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<td>Milk and Milk Products</td>
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<td>Sesame seed</td>
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EFFECT OF FLUID, BEVERAGES & ALCOHOLIC BEVERAGES:

FLUIDS:

This is the most important preventive measure for all patients who develop kidney stones. It hinders the formation of stones by diluting the urine. For example, more sugar can be dissolved in a full glass of iced tea than in a half glass. Patients should drink enough fluid to produce two quarts or more of urine each day. As a guideline, drink 1-2 glasses of fluid every hour while awake, and 1-2 glasses once during the night if awakened for some reason. 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:

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.

Hollinberry PW Massey LK (1986) Drinking coffee or other caffeine-containing beverages increases urinary calcium.

Curhan GC, Willet WC, et.al. (1996) - have found that 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.

**ALCOHOL:**

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.
CITRUS:

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.

Ettinger B Pak CY et al (1997) suggested that lemon juice may be preferable, as it has almost five times the citrate of orange juice. A small study found that drinking 2 liters of lemonade a day doubled urinary citrate in people with decreased urinary citrate.

KETOGENIC DIET:

The ketogenic diet sometimes is used to treat intractable seizure disorders in children. It involves an initial period of fluid restriction and starvation until ketone bodies appear in the urine. This is followed by a low-protein, low-carbohydrate, and fluid-restricted diet. This tends to cause chronic metabolic acidosis with hypocitraturia and relatively low urinary volumes, which induce kidney stone formation. Elevated uric acid levels also have been reported. The average time from initiation of the diet until stone presentation is about 15 months, so patients who are started on this diet should be checked for stone formation at about 12 months after diet application. Fluid liberalization and citrate supplements can be used to prevent. Curhan GC. et al.,(2004) - fortunately, now suggests that dietary calcium does not promote kidney stones, but those consuming more foods rich in calcium and potassium, and drinking lots of fluids, have already been shown to have a lower risk of forming
who ate the least. While taking supplemental calcium did not appear to increase risk, it didn't lower it. Other dietary factors that lowered kidney stone formation risk were eating foods high in phytates—a chemical in high fibre foods such as whole grains that binds minerals (37% risk reduction), drinking lots of fluids (32% risk reduction), and eating animal protein (16% risk reduction). Eating foods rich in sugar (sucrose) raised risk of kidney stone formation by 31%.

AIMS AND OBJECTIVE: "Prevalence of hypercalciuria in renal stone formers and its relationship with diet".

(1) To study the prevalence 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 and compare with normal population.

(6) To study hypercalciuria, its effect on calcium stone formers and normal population.