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Combination Therapies: Investigating The Effectiveness Of Combining Different Treatments, Such As Medications And Lifestyle Changes, To Prevent Or Treat Kidney Stones.

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Abstract :

Kidney stones associated with pain and suffering. Kidney stone disease (KSD) (also called nephrolithiasis/or urolithiasis)is a global health issue that progresses affecting people in almost all developed and developing countries. This spread-out disease around the world carries an incidence of 5-15%. Its prevalence is considerably on the higher side with recurrence mostly after treatment. While many methods are available to treat Kidney stone disease, their prevention should be adopted to decrease the suffering and economic burden of Kidney stone disease. The pathogenesis and risk factors leading to stone formation must be known before the control measures for the formation of stones can be instituted. Less urine output and dehydration remain common risks for every type of stone, while hypercalciuria, hyperoxaluria, and hypocitraturia remain major risks for calcium stones. Thus, in this article will be find up-to-date knowledge-based strategies mainly on prevention and Kidney stone disease. Fluid intake particularly is very important (water-2.5-3.0 L/d). Diuresis has also been encouraged. With dietary restrictions, we can minimize the incidence and recurrence of kidney stones. The present review focuses on the pathophysiology and available treatment options for the kidney stones.

KEYWORD

Bioactive compound, citrate, diuresis, natural compound, nephrolithiasis, probiotics, protection, urolithiasis, Renal stones, Colic, Calcium stones, Calculi, Dietary , Lifestyle, western world , disease management , Korean and European company .

INTRODUCTION

Renal calculi essentially arise from deposition of inorganic substances (for instance, crystalline salts) coupled with organic components (like urinary macromolecules) inside renal parenchyma or pelvicalyceal system. The disease process, known as nephrolithiasis or urolithiasis, is common worldwide, with a steadily increasing trend in several regions [[1], [2], [3], [4]]. According to a projection in 2012 from the Urological Diseases in America, the estimated cost may have been up to $10billion, meaning stone diseases have become one of the most expensive urological conditions [5]. More recent analytic data using the National Health and Nutrition Examination Survey (NHANES) report that Kidney stone disease incidence is higher in males than in females among US adult populations [5]. The prevalence over the past decade has remained stable among men but increased in women, from 6.5% during 2007-2008 to 9.4% during 2017-2018 [5]. Kidney (renal) stones are typically formed when urine is significantly concentrated in the kidneys, and in that way, the formed crystals cannot pass through the kidneys. The pain referred to below occurs in the abdomen, called renal colic. The size of kidney stones varies from a grain of sand to as big as a typical tennis ball. A few stones can pass through with urine without intervention due to their size, but most require some help; sometimes minor surgery is necessary.

Schematic diagram of kidney stone :-

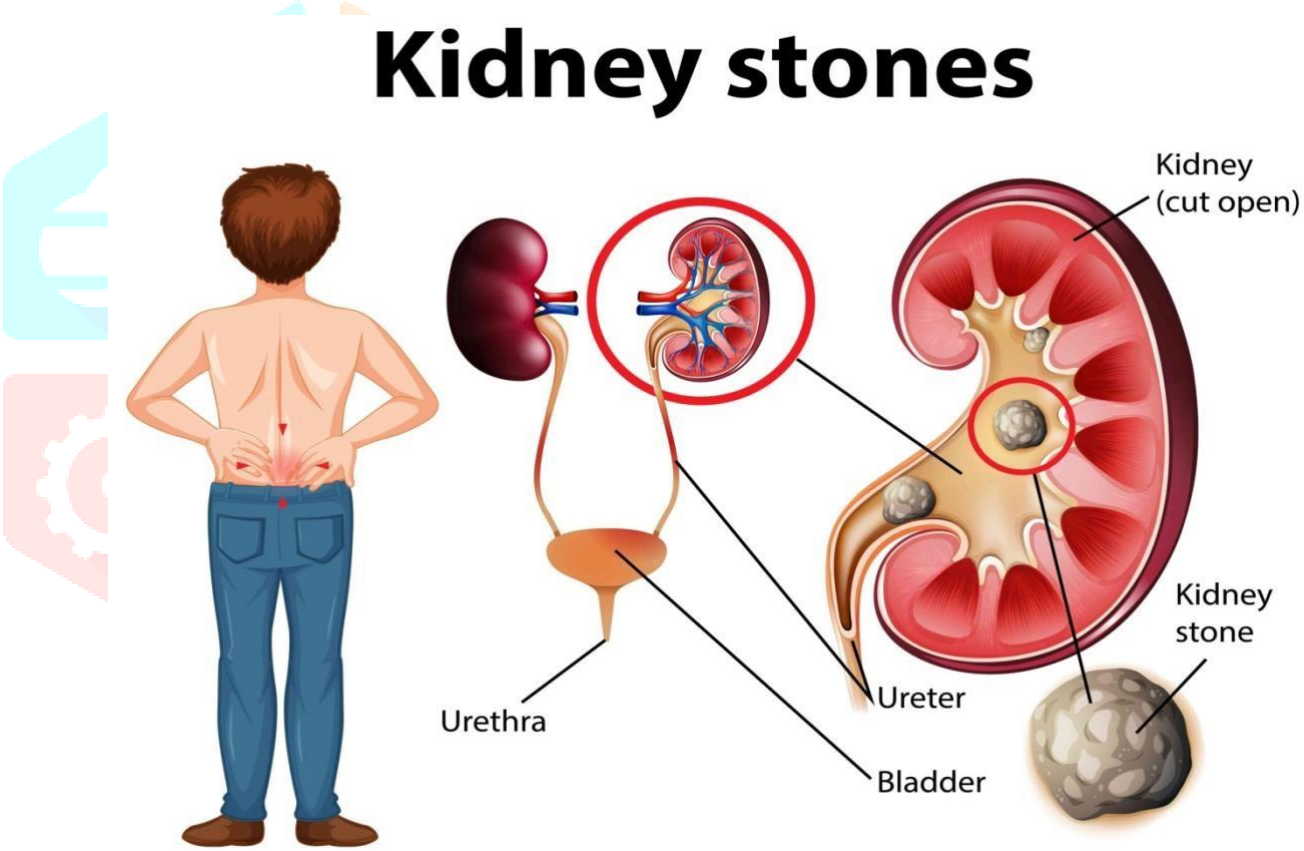


TABLE 1

Composition of Kidney Stones in Developed Countries

Stone type	Children (%)	Adults (%)
Calcium	50 to 90	64 to 92
Calcium oxalate	60 to 90	32 to 46
Calcium phosphate	10 to 20	3 to 5
Both	—	29 to 40
Cystine	1 to 5	1
Struvite (magnesium ammonium phosphate)	1 to 18	2 to 15
Uric acid	1 to 10	3 to 16
Other	4	1

Information from references 3 through 8.

How common kidney stone ?

*Prevalence

- Globally: 1 in 10 people-about 600 million-will ever develop kidney stones in their lives.
- USA: 1 in 11 people-about 29 million-have ever been afflicted with kidney stones.
- Europe: 1 in 15 people-about 50 million-have suffered from kidney stones.

*Incidence**:**

- World: Annually, 5-15% of adults will develop kidney stones.
- USA: Every year, 200,000 to 400,000 new cases are diagnosed.

*Recurrence:

- By 5 to 10 years, 50% of those who have had kidney stones will develop recurrences.
- 30% of those will have multiple recurrences..

*Risk Factors:

- Family history
- Age (Most common in individuals ages 35-60 years old.)
- Gender(more prevalent in males than females)
- Obesity
- Diabetes
- Hypertension
- Kidney disease
- Some medications
- Diet (low fluid intake, high salt and sugar and animal protein)

Types of kidney stone :-

CALCIUM STONE

Traditionally, kidney stones are labelled by the primary crystalline composition(Figure1). Several studies from various regions have consistently reported that scalcium is the most common inorganic constituent among all kidney stones[6,7]. One of the most common types of calcium stones is calcium oxalate(CaOx), either homogeneously or mixed with one another, such as with calcium phosphate (CaP)[6,7].CaOx is said to exist in three different crystalline forms depending upon its hydration. These forms are calcium oxalate monohydrate (COM; CaC2O4-H2O), calcium oxalate dihydrate (COD; CaC2O4-2H2O), and calcium oxalate trihydrate (CaC2O4-3H2O). Of these three forms, COM (also called when we llite) is the most frequently found hydrated mineral in clinical stones, followed in number by COD (also called wed dellite) [6].Moreover, further evidence for the role of dietary calcium in the development of urinary stones is found from prospective cohorts [8,9] that show that individuals in the highest quintile of calcium intake were over 30% less likely to develop a stone than those in the lowest quintile. While there was some variation among the cohorts, in general, the top quintiles represented >1000mg of daily calcium intake. Hence, it has been recommended in the guidelines that calcium stone formers make sure 1000–1200 mg of dietary calcium are taken every day [10].

STRUVITE STONE:

Struvite stones are also called triple phosphate or calcium magnesium ammonium phosphate, and their formation is dependent on the presence of urea-splitting bacteria, with an exceedingly high pH being a prerequisite. Struvite stone is composed of magnesium ammonium phosphate (MgNH4PO4·6H2O) and is often called infection stone.

Struvite commonly combines with CaOx and CaP, especially carbapatite, in the stone matrix[11].As these stones contain bacteria within themselves, aggressive surgical stone removal is required to kill the bacteria and prevent recurrence of infection an stone formation. It is suggested that both culture-specific antibiotic therapy, should begin preoperative and continue for some time post-operative, sterilizes the urine and so helps to avert re-infection. A recent multi- institutional study on the pattern of infection and colonization of struvite stones has shown evidence of change in the bacteriology away from traditional urea-splitting organisms like Proteus to Enterococcus and Escherichia coli [12].

URIC ACID STONE

Risk factors leading to the formation of uric acid stones include low urine pH, low urine volume, and lesser degrees of hyper-uricosuria. The urine pH is frequently thought to be a chief risk factor since urinary uric acid levels generally tend to be comparable between normal patients and uric acid stone formers [13]. The uric acid stone consists of uric acid crystals mostly crystallized in acidic urine, and most uric acid crystals are found in the dihydrateform[14]. This type of stone is particularly common in type two diabetic patients and those with obesity. Uric acid stone prevalence in males seemed to have increased during recent years [15]. However, uric acid crystals are mostly mixed with other types of crystals [15].

CYSTINE STONE

Cystine stones are a rare type of kidney stone associated with the uncommon genetic disease cystinuria.Cystine stone disease arises as a result of defects in the functioning of cystine transporters in the kidney due to mutations of the cystine pathway that are inherited in an autosomal recessive fashion (e.g., SLC3A1).[16]Cystinuria is a very rare inherited disorder where the transport of cystine and dibasic amino acids, like lysine, arginine, and ornithine, across the renal tubular membranes is impaired. Treatment of this disease is primarily centered around aggressive urine hydration and urinary alkalinization to achieve a urine pH of 7 or greater in order to increase the solubility of cystine.When these measures fail to adequately reduce the risk for recurrent stone disease, treatment with a cystine-binding agent may be considered. D- penicillamine and

tiopronin are recommended, although efficacy data are scant. Evidence from uncontrolled or observational trials indicates reductions in stone events of up to 75% [17].

OXALATE STONE

Hyperoxaluria in calcium stone formers occurs in approximately 10%– 15% of individuals .Increased urinary oxalate may stem from dietary intake, endogenous oxalate overproduction, or intestinal oxalate over absorption. Approximately 10% of ingested oxalate is absorbed, while the remainder is eliminated through stools in normal individuals[18,19]. For some unexplained reason, calcium oxalate stone formers are believed to absorb a higher proportion of oxalate than normal subjects[18]. While the amount absorbed is a factor in intestinal absorption of oxalate, it also depends on dietary calcium intake. A high intake of calcium decreases oxalate absorption, whereas a restricted orthophosphate and oxalate diet leads to an increase in oxalate absorption and, consequently, urinary excretion[19,20]. Enzymatic defects in the oxalate biosynthetic pathway cause heavy elevation of urinary oxalate, which gives rise to:

(1) Aggressive calcium oxalate stone formation.

(2) Oxalosis.

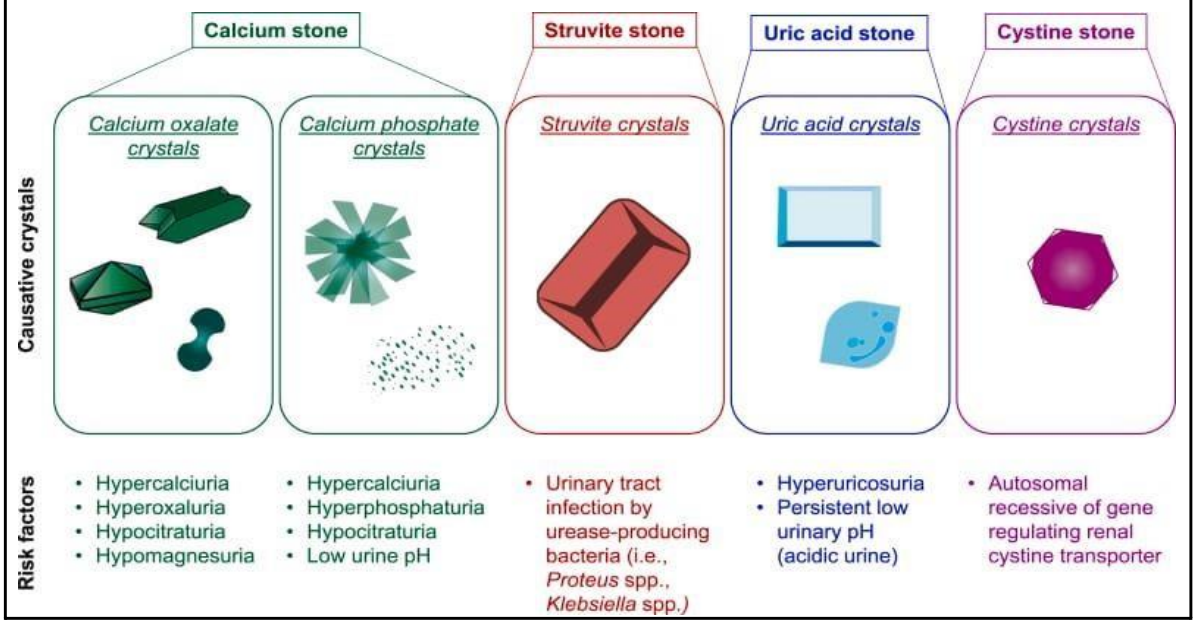
Among the three forms of primary hyperoxaluria, renal failure is usually seen only with primary hyperoxaluria type l. All forms of primary hyperoxaluri are quire strict dietary oxalate restriction[21]. Hyperoxaluric patients are recommended to have a normal calcium diet in association with oxalate restriction. High oxalate foods are spinach, rhubarb, beets, nuts, chocolate, potatoes, bran, legumes, and tea[22]. Some juices, including cranberry[23], grapefruit[24],and carambola juice[25], have been demonstrated to have relatively high oxalate content.

Figure 2

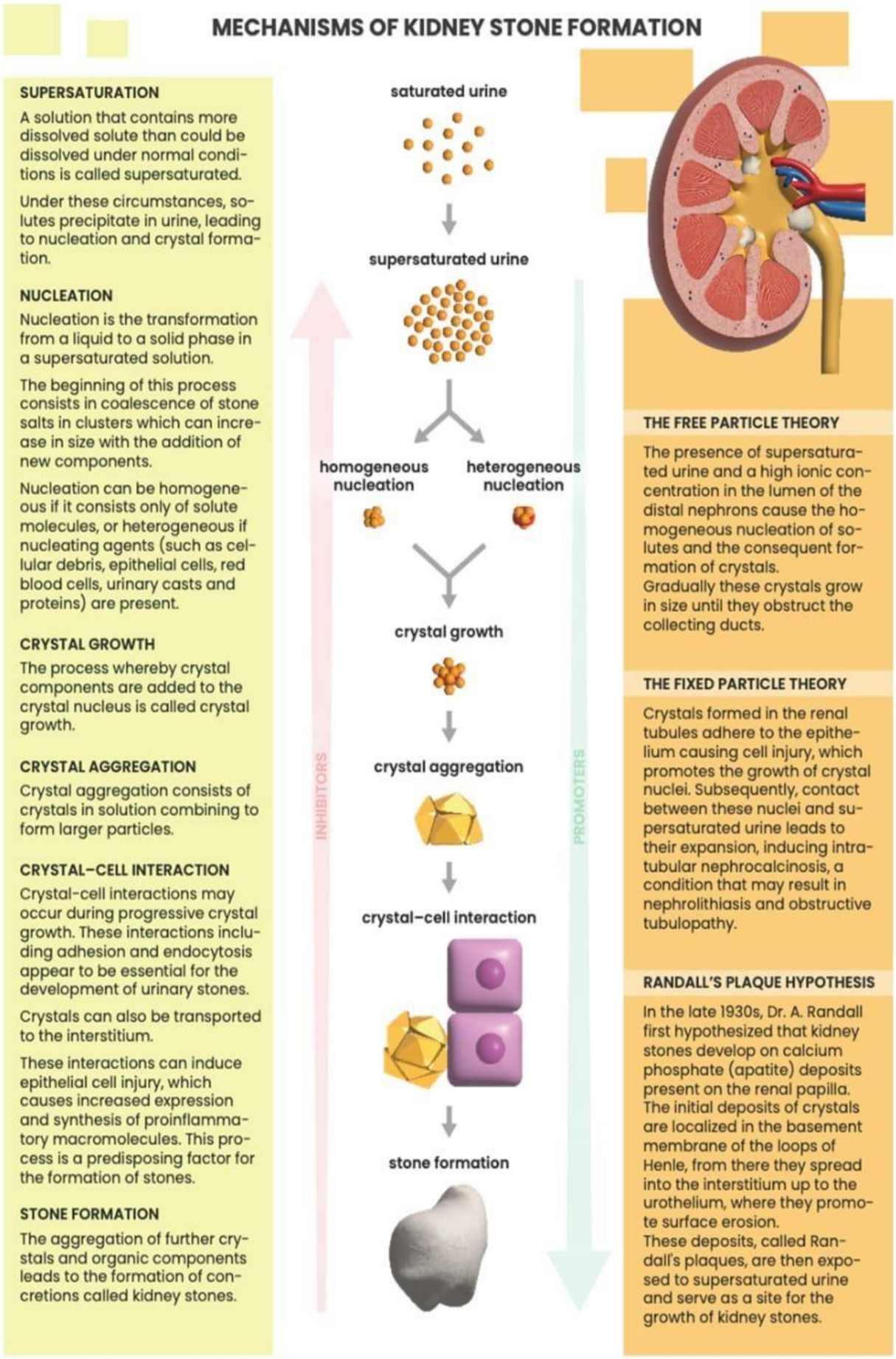
Brief Mechanism of Kidney Stone Formation :

Figure no.03

Search parameters and criteria. The schematic illustration summarizes all keywords used for PubMed search and criteria to retrieve articles for discussion in this review.



A mechanism for stone creation. The first free practice theory occurs initially in the distal nephrons. This takes place in the places that have attained the degree of super-saturation of urine with crystalline salts. The second mechanism - the fixed practice theory, takes place as well as acts in renal tubules through adherence to the epithelium causing cell injury which promote further the growth of the crystal nuclei.“RANDALL' S PLAQUE HYPOTHESIS &quot ;states that accumulation of interstitial calcium phosphate crystal (CaP) commonly leads to inflammation triggering.



Strategies to Prevent kidney stone Disease :

1} Modifiable lifestyle factors and habit:

1.1) Coffee and tea consumption: It is well accepted that caffeine consumption is diuretic. However, the relationship between caffeine intake and the incidence of kidney stone disease is disputed. Massey et al. found evidence of a positive correlation between caffeine intake and the development of calcium oxalate stones in a case-control study [26]. On the other hand, in some cross- sectional study, it was shown that there was a dose-response relationship for caffeine consumption in that it increased the risk of recurrent kidney stones For every quartile increase in caffeine intake, the multivariate-adjusted odds ratio (with 95% confidence intervals) for recurrent kidney stones increased by 1.15 (range 1.01-1.31) [27]. How ever, urinary risk of Kidney stone disease was inversely associated with genetically predicted coffee and caffeine intake according to the UK Bio bank study. Relevant single nucleotide polymorphisms (SNPs) that were associated with coffee consumption at genome-wide significance level were derived from a meta-analysis of four genome-wide association studies (GWAS). Out of 17 replicated loci in the study, five of these had been associated with coffee consumption or plasma caffeine metabolites (GCKR, ABCG2, AHR, POR, and CYP1A1/2) [28]. The combined odds ratio for kidney stones was 0.60, with a 95% confidence interval of 0.460.79 and p < 0.001 for every 50% increase in coffee consumption, genetically predicted, and 0.81 with a 95% confidence interval of 0.690.94 and p = 0.005 for every 80 milligrams increase in caffeine consumption, genetically predicted [28]. Caffeine causes significant changes in antidiuretic hormone activity as a result of its inducing increase in urine flow and decrease in the maximal concentration of urine. Caffeine further prevents calcium oxalate stone formation and diminishes the adhesion of calcium oxalate crystals onto the surfaces of renal tubular epithelial cells [29]. But evaluating the relationship between tea consumption and risk of urinary stones represents yet another tough problem. On the one side, tea contains oxalates that could favor the possibility of kidney stone formation. On the opposite side, tea is rich in poly phenols and numerous other phyto chemicals, and the antioxidant activities of these substances potentially help prevent urinary stone formation [30]. Besides, caffeine in tea could reduce calcium oxalate crystals adhesion to the renal tubular epithelial cells [31].

1.2) Vitamin Supplementation : Much of the research exploring the role of vitamins in kidney stone formation has concentrated on the effects of vitamin C and vitamin B6-both involved in oxalate metabolism. While vitamin C can be converted to oxalate, vitamin B6 acts as a cofactor in the metabolism of oxalate. A previous study showed that taking 1000 mg or more of vitamin C per day was associated with a 41% elevated risk (95% CI: 11%-80%) of developing a first kidney stone [32]. In addition, another recent study, with more than 23,000 Swedish men, indicated an increased risk of kidney stones with the use of supplemental vitamin C, returning a multivariable-adjusted hazard ratio of 1.92 (95% CI: 1.33-2.77) compared to nonusers [33]. Zengetal. conducted one of the largest cross-sectional studies to investigate the association of prevalence of kidney stones with nine common vitamins. Using regression analysis, they showed that compared with lower intakes, high intakes of vitamin B6 (OR (95% CI): 0.76 (0.62, 0.93)), vitamin C (OR [95% CI]: 0.73 (0.59, 0.90)), and vitamin D (OR (95% CI): 0.77 (0.64, 0.94)) had a protective influence against kidney stones. Also, a J-shaped curve was evident in the relationship between vitamin C intake and kidney stone prevalence, being initially protective and later repelling [35]. An in vivo study has shown that vitamin C can cause increased urinary oxalate excretion in hyperoxaluric rats induced with hydroxyl -L-proline[34].It has been suggested that the seemingly pro-lithogenic manner observed with vitamin C in relation to kidney stones may beattributable to un-favourable preservation techniques employed during urine preservation, detection, and analysis [35,36].

1.3) Alcohol Consumption Intake: Alcohol is one of the most commonly consumed psychoactive substances around the globe. Previous studies have yielded mixed results regarding the link between alcohol consumption and urolithiasis. The varying effects of different types of alcohol and consumption habits underscore the need to consider both the amount and type of alcohol when assessing the risk of kidney stones. It is believed that the protective effect may stem from increased urine volume due to alcohol's diuretic properties, along with certain phytochemicals found in some beverages that could help prevent crystal formation. On the other hand,

excessive alcohol consumption seems to elevate the risk, likely due to dehydration and metabolic issues. In 2023, a cross-sectional study investigated the connection between alcohol intake and the prevalence of kidney ,stones by analyzing data from the National Health and Nutrition Examination Survey (NHANES).The results indicated no significant link between alcohol consumptionboth lifetime and recent (within the past 12 months) and a history of kidney stone formation. Additionally, the analysis showed that the amount and frequency of alcohol consumption did not significantly correlate with the occurrence of kidney stones, even among heavy drinkers[37]. A study involving over 28,000 Korean patients, utilizing data from the Korean National Health Insurance Service-Health Screening Cohort, found that alcohol consumption was linked to a lower likelihood of kidney stone disease (KSD) (adjusted odds ratio (aOR)= 0.89, 95% confidence interval (CI): 0.86-0.92,p<0.001).This inverse relationship was especially notable in individuals under 55 years old (aOR = 0.82, 95% CI: 0.78-0.87) and in males (aOR = 0.86, 95% CI: 0.84-0.89)[38]. Similar patterns were observed in a prospective study involving over half a million participants from China, which examined the relationship between alcohol consumption and the risk of kidney stones through a multivariable analysis[39,40,41].

1.4)Physical Activity : Recent studies have investigated the potential protective role of physical activity against Kidney stone disease, but the findings remain inconclusive. A comprehensive cohort study involving nearly 90,000 postmenopausal women indicated that physical activity, irrespective of intensity, might reduce the risk of Kidney stone disease [42]. This association was further supported by a meta analysis encompassing over 200,000 participants, which linked higher levels of physical activity with a lower incidence of kidney stones in women [43]. In another study reported by Fenget al., physical activity shows an inverse relationship with the prevalence of kidney stones, exhibiting a dose–response effect that plateaus beyond a certain level of activity. Specifically, as physical activity increases, the prevalence of kidney stones decreases until reaching a plateau at about 2480 MET-minutes per week, where the odds ratio (OR) stabilizes at 0.75 (95% CI: 0.63–0.91), indicating no further reduction in kidney stone prevalence with additional physical activity [44].However, results from subsequent meta-analyses have failed to establish a significant connection between physical activity and the risk of developing kidney stones, suggesting that earlier positive findings may have been affected by various confounding factors [45]. Given the mixed evidence and the presence of confounding variables in these studies, the actual impact of physical activity on Kidney stone disease risk remains controversial. This underscores the needfor further research, particularly studies that adjust for multiple variables to clarify the true effects of physical activity on kidney stone prevention. Such investigations are essential to establish

2} Effect of Diet on Urinary Stone Event :

The second participant attended the meet. Effect of Diet on the Stone Formation in Urine: Dietary factors are extremely important in preventing stone formation. The available studies, albeit studying other individual risk factors, have focused on one variable at a time. However, it must be remembered that humans do not consume nutrients alone-they eat a composite of nutrients, and the interaction may be significant. For instance, Taylor et al. Examined the effect of a DASH-style diet (high consumption of fruits, vegetables, lowfat dairy products, and nuts, low sodium and processed meats) prospectively in more than 240,000 patients in three large cohorts. The highest DASH scores in three cohorts were linked to a 40-45% decreased incidence of stone formation over 50 years of monitoring, with more than 5600 beginnings of kidneys stones. High dietary calcium competence promotes oxalate-binding and less absorption of oxalate from the distal tract, with therefore less urinary excretion.

2.1)Sodium: The first link between sodium intake and urinary calcium excretion was reported by Kleemanetal. [46].An increase of sodium from 19 to 419 mEq/d resulted in an 82% increase in urinary calcium in healthy volunteers. Later salt-loading studies in normal humans revealed a calcium excretion pathophysiologic relationship with sodium: roughly 0.8 mm ol calcium for each 100 mm ol of sodium ingested daily (40 mg calcium for every 6 g salt consumed)[47]. Experimental data indicate that idiopathic calcium stone formers may be more sensitive than most to the effect of sodium intake on calcium levels in the urine, implying that those with reduction in dietary sodium might have a more pronounced metabolic effect. For instance, in

a study of 14 hyper calciuric stone formers, raising sodium intake from 213 to 276 mm ol /d augmented calcium excretion by 11.1-13.3 mm ol/d (a calcium-to-sodium ratio of 3.5 mmol calcium for every extra 100 mm ol sodium ingested daily)[48]. Two other studies found an increase in calcium/sodium ratios in hypecalciuric stone formers of 1.9[49] and 2.1[50]. Besides its detrimental effect on calcium excretion, Sakhaeeetal.[51] pointed out that increasing sodium from 50 to 300 mmol/d decreased urinary citrate, which protects against crystallization and stone disease, by about 20% (with a concomitant rise in urinary calcium by 44%). In the case of 210 hyper calciuric calcium stone formers randomized to either low sodium or an ad libitum diet (both groups consuming 2-3 L of fluids and maintaining a calcium intake of 800-1000 mg each), Nouvenne et al.[52] showed a reduction in urine calcium from 432±96 to 271±86 mg/d with the low sodium diet, whereupon urinary sodium dropped from 228±57 to 68±43 mm ol/d at 3 months. Because of the data presented here, reducing dietary sodium to below 100 mm ol /d is a goal that makes a good deal of sense and is supported by national guidelines[53] .Targeting a dietary sodium intake <100 mm ol/d is a reasonable goal, on the basis of the data herein, and issupported by national guidelines[54].

2.2)Citrate Intake: Hypocitraturia is another key risk factor for KSD. Evaluation of the above knowledge shows that for hypo citraturia various factors can be cited which lead to its formation; the use of some drugs, carbonic anhydrase inhibitors (like acetazolamide) used for other diseases, is one of the most important of such factors [55]. The AUA, EAU, CUA, and UAA have all encouraged patients with kidney stones to eat more fruit and vegetables in order to increase fiber and urinary citrate concentrations, particularly patients with hypocitratria[56]. Citrus fruits are the main dietary source of citrate, with grapefruit and lemon juices containing the highest and second highest citrate concentrations, respectively, compared to other citrus fruit juices [57]. Intake of orange soda, another beverage rich in citrate, has also been shown to increase urinary citrate excretion [58]. Additionally, a recent study found that lemonade use could be a low-cost method for prevention of further calcium stone formation through the increase of urine output and urine citrate concentration and the decrease of CaOx and CaP oversaturation [59]. Supplementing the diet with fresh lemon juice may prevent the recurrence of CaOx stone disease [60]. In addition to this, lime powder inhibits COM crystal growth and Over production of reactive oxygen species during COM crystal- induced tubular cell injury[61].The urinary excretion of citrate is increased, urine pH is increased, and urinary CaOxsupersaturation[62] is decreased with the administration of lime powder in healthy volunteers in a clinical trial. In vitro cell culture, mouse model in vivo, and healthy subjects were found not to have any toxic effects due to lime powder ingestion-this indicates that it would be safe to consume lime powder daily. Furthermore, lime powder resulted in significant decreases in the excretion of 24-h urinary (total) protein yet increases in urinary uromodulin concentrations, when compared to placebo were done in stone formers[63,64]. Uromodulin has both stimulatory and inhibitive effect in kidney stone formation. Under some conditions, uromodulin has been shown to promote crystal aggregation. However, in vitro and in vivo studies agree that uromodulin is a powerful inhibitor of calcium stone formation. This leads to lime powder preventing Kidney stone disease by increasing urinary concentrations of citrate and uromodulin-both strong inhibitors of calcium stone formation[65,66,67].

2.3)Calcium Intake and vitamin D Supplementation:- There is, however, a point of concern with daily calcium intake since hypercalciuria is one of the main risk factors for calcium stone disease. The AUA, EAU, CUA, and UAA have uniformly recommended dietary calcium intake between 1000 and 1200 mg/d, respectively .[68,69].There is evidence that calcium supplement may increase the risk of kidney stone disease[70].On the other hand, high dietary calcium reduces the symptomatic kidney stone disease because of the binding of dietary calcium with dietary oxalate in the gut and its subsequent lesser absorption of oxalate in the gut[70,71].Hypercalciuria has several contributing factors, including renal acid load diets[72], metabolic bone disease[73], and hyperparathyroidism[74].Calcium absorption in the gut can be increased through vitamin D supplementation.Dietary calcium with vitamin D ends up synergistically enhancing kidney stone disease formation in rat studies[75].Such in vivo investigations assessed, and coadministration of calcium and vitamin D could aggravate Randall plaque formation[76].A retrospective study has indicated that vitamin D supplement increases urinary calcium excretion among calcium stone formers[77].However, a meta-analysis noted that long-term supplementation of vitamin D increases the risk of hyper calciuria but does not increase the risk of kidney stone disease[78].In another prospective analysis involving nearly 200,000 participants, it has been reported that the association between vitamin D intake and risk of kidney stone disease does not reach

statistical significance[79].Equally, a randomized controlled trial has shown that monthly vitamin D supplementation does not increase the risk of kidney stone disease or serum calcium concentration [80].It is also important to consider the calcium stone subtype and patient background to understand the discordant results for certain findings.Thus one should consider patient risk factors along with calcium and vitamin D levels before deciding on dietary calcium intake and vitamin D supplementation.

2.4)Oxalate : oxalate-rich foods, whereas endogenous oxalate is produced by liver and erythrocytes [81,82]. Hyperoxaluria can be classified as primary and secondary hyperoxaluria. Defects in liver enzymes involved in glyoxylate Metabolism, which results in oxalate overproduction and), are the leading cause of. Primary hyperoxaluria [81,82]. Hence, liver injury is associated with primary hyperoxaluria [83,84]. Secondary hyperoxaluria is caused by many factors, such When high oxalate and oxalate precursor–rich food is ingested and increased incidence of .intestinal absorption of oxalate due to mal absorption syndrome [81,82]. The urinary oxalate level is primarily affected by intake of foodstuffs, such as, respectively. oxalate and its precursor [85]. Although patients with calcium oxalate stone Diseases are frequently advised to restrict dietary oxalate intake, unless there is an alternative. Hyperoxaluria they receive, it has not been shown that this is useful to prevent stone formation. recurrence. When hyperoxaluria is observed, it may be advisable to restrict the. Intake of single foods high in oxalate, especially in the light of evidence. That stone formers have increased fecal oxalate excretion [86].Notably, an Calcium intake sufficient to >1000 mg/day can compensate the impact of dietary oxalate.

2.5)Animal Protein :- The relationship between dietary protein intake and the risk of kidney stone disease (KSD) varies depending on the type of protein consumed[87]. Specifically, the consumption of animal proteins, including fish, beef, and chicken, is linked to a higher risk of developing Kidney stone disease, particularly calcium and uric acid stones[87,88]. Consequently, the European Association of Urology (EAU) and the Urological Association of Asia (UUA) guidelines recommend limiting animal protein intake to 0.8– 1.0 g/kg of body weight per day for patients with calcium stones and those with hyperuricosuria. This is due to the fact that animal proteins are associated with lower urinary pH and citrate levels, as well as higher concentrations of urinary oxalate and uric acid[89,90]. While the Canadian Urological Association (CUA) does not specify exact amounts, it also advises moderate consumption of animal proteins[91]. In contrast, the American Urological Association (AUA) suggests restricting nondairy animal protein intake for patients with calcium and uric acid stones[92]. Additionally, the dietary acid load from animal-derived foods impacts calcium and citrate metabolism, resulting in increased urinary calcium excretion and decreased urinary citrate excretion[93,94]. On the other hand, plant protein intake appears to have protective effects against Kidney stone disease [95]. Therefore, it is advisable to increase the ratio of plant to animal proteins in the diet to help prevent kidney stone disease.

Table 1 .Dietry recommendation to prevent kidney stone recurrence.

Dietary component	Recommendation
Fluid	• Maintain fluid intake that achieves urine volume ≥2.5 L daily • limit sugar –sweetened soft drinks • Consider intake of orange juice with no added sugar to prevent calcium nephrolithiasis
Calcium	• Avoid severe dietary calcium restriction • Maintain calcium intake of 1000-1200mg/day
Oxalate	• Avoid oxalate rich foods (nuts, chocolate, brewed tea, spinach, rhubarb, beets, potatoes, peanut butter, wheat bran, beans) • Avoid juices with cranberry, grapefruit, starfruit
Protein	

Carbohydrate Sodium Citrate Calcium supplement Vitamin B6 supplement Vitamin c supplement Vitamin D supplement Low carbohydrate / high protein diet (atkins) DASH diet Mediterranean diet	• Maintain normal calcium intake • Modestly restrict animal protein (red meat, fish, poultry, pork, shellfish) to no more than 6–8 ounces daily • Restrict refined carbohydrate <20g/day • Limit sodium intake to ≤100mEq/day (2300mg/day) • Increase intake of the fruit and vegetable , orange juice is beneficial. • Consider for enteric hyperoxaluria (take with the two largest meals) but avoid for idiopathic calcium stone disease if dietary calcium intake is sufficient. • Consider for primary hyperoxaluria type :1 but not proven for idiopathic causes • Limit intake vitamin C to <2g/day • Should not be withheld solely on the basis of stone disease. If deficient and repletion is indicated, monitor with 24 h urine analysis • Avoid (increase in net acid; hypocitraturia;hypercalciuria, hyperuricosuria). • Likely protective against stone disease . • Likely protective against stone disease (inferred from similarities to DASH diet).
* DASH, dietary approach to stop hypertension *	

3}Natural Bioactive Compound :

Over the last ten years, researchers have extensively explored the positive effects of dietary plants on kidney stone disease (KSD)[96,97,98,99,100] and the anti litho-genic properties of various plant extracts[101,102,103,104,105,106,107,108]and bioactive compounds.[109,110,111,112,113,114] Recent reviews have compiled evidence regarding dietary and medicinal plants, along with their bioactive components, in the prevention and management [115,116]of kidney stones. In this section, we will discuss some of these anti litho-genic bioactive compounds, focusing on recent studies that examine their molecular mechanisms.

3.1)Caffine : Caffeine is a natural xanthine alkaloid and a key bioactive component found in coffee beans. It is well established that caffeine has a diuretic effect. However, the relationship between caffeine intake and the prevalence of KSD has been a topic of debate. A study involving 39 individuals with calcium stones and 39 control participants found that caffeine consumption increased the calcium oxalate precipitation index, thereby raising the risk of Kidney stone disease [117]. Conversely, several large metaanalyses[118] have consistently indicated an inverse relationship between the intake of caffeinated beverages and the risk of Kidney stone disease [119,120,121], suggesting that caffeine may actually lower the risk of developing kidney stones]. Supporting this, a recent prospective cohort study confirmed that coffe consumption[122] is linked to a reduced risk of Kidney stone disease. Additionally, an in vitro study demonstrated that caffeine has protective effects against kidney stone formation[123]. The findings suggest that caffeine exerts an anti lithogenic effect by decreasing the ability of renal epithelial cells to bind calcium oxalate monohydrate (COM). This protective mechanism is facilitated by the translocation of annexin A1, a protein that binds to COM, from the apical surfaces to the cytosolic compartment of renal epithelial cells in the distal nephron[123].

3.2)Epigallocatechingallate : Epigallocatechingallate (EGCG) is a key bioactive compound primarily found in green tea. While tea is recognized as a significant source of oxalate, studies show that green tea consumption is linked to a lower risk of kidney stone disease (KSD) [124]. This negative correlation appears to be stronger in males compared to females [124]. Research indicates that green tea supplements enhance superoxide dismutase (an antioxidant) activity, reduce urinary oxalate excretion, and lower calcium oxalate (CaOx) deposition in rats with kidney stones induced by EG [125]. Additional in vivo studies have supported the protective effects of green tea, demonstrating that it decreases urinary oxalate excretion and the renal deposition of CaOx crystals in rats treated with sodium oxalate [126]. Furthermore, EGCG has been shown to protect against oxalate-induced damage to renal cells and the overproduction of reactive oxygen species [126]. To explore how EGCG inhibits kidney stone formation, an in vitro study found that EGCG blocks the oxalate-induced movement of αenolase, a protein that binds to calcium oxalate, thereby reducing the ability of renal epithelial cells to bind to calcium oxalate [127].

3.4)Diosmin: Diosmin is a bioflavonoid that can be found in various plants and fruits, particularly in citrus fruits. Research has shown that diosmin has an antilithogenic effect, helping to prevent calcium oxalate (CaOx) deposition in the kidneys of rats with induced nephrolithiasis [128]. Additionally, diosmin has been found to mitigate degenerative changes in both the renal cortex and medulla [128]. A more recent in vitro study has thoroughly investigated the dual effects of diosmin on CaOx crystals [129]. During the crystallization process, diosmin increases both the number and mass of CaOx crystals. Conversely, it reduces the size and growth of these crystals. In other processes, diosmin promotes the self-aggregation of CaOx and its invasion into the extracellular matrix, while simultaneously decreasing the interactions between CaOx and cells [129].

4} Bacterial E-radiation : Bacterial eradication of UTIs caused by urease- producing bacteria is linked to the formation of struvite stones. However, the impact of UTIs on other types of kidney stones has also been documented [130]. Bacterial cultures from the urine of stone formers and stone matrices (both the nidus and periphery) have identified Escherichia coli as the most prevalent organism across all these clinical samples, suggesting that E. coli may play a role in kidney stone formation [130]. Notably, only intact, viable bacteria an facilitate the growth and aggregation of CaOx crystals, while dead or fragmented E. coli do not have this effect [131]. A subsequent study indicated that outer membrane vesicles (OMVs) from E. coli isolated from the urine of stone formers enhance CaOx crystallization, crystal growth, and aggregation [132]. The promoting effects of OMVs from E. coli are mediated by the Elongation factor (EF)-Tu present on the OMVs' surface, and neutralizing this factor with a specific antibody significantly diminishes these promoting activities [167]. Interestingly, EF-Tu is found in greater quantities in E. coli from the urine of stone formers compared to those from non-stone formers with UTIs [131]. Furthermore, recent research has shown that flagella from viable E. coli also contribute to the promotion of CaOx crystallization, growth, and aggregation [133]. Besides E. coli, other bacteria such as Klebsiella pneumoniae, Pseudomonas aeruginosa, and Staphylococcus aureus have also been detected in the stone nidus, cortex, and urine of stone formers [130]. These bacteria similarly promote the growth and aggregation of CaOx [131]. Traditionally, increasing water intake is one of the strategies to prevent UTIs. Thus, higher water consumption and diuresis can reduce the risk of kidney stone disease in various ways. The removal of infection sources (such as stone removal) should be considered for effective bacterial eradication, especially in patients with recurrent UTIs [134]. Additionally , the use of antibiotic based on bacterial culture from the urine and stone matrices may be able to prevent recurrent formation . However ,bacterial eradication by antibiotics should be carefully considered because multidrug resistance has been detected in a bacteria isolated form the urine and stone matrices[130].Some classes of antibiotics are associated with the increased risk of KSD or crystal- induced nephropathy [135].The use antibiotics for >2 mo in early adulthood and middle age is also associated with high risk of KSD in later life[136]. Moreover , a recent study has found that the use of antibiotics may suppress oxalobacter formigenes in the gut of mircobiome [137].Therefore , bacterial eradication by using antibiotics can be performed but with serious caution and close monitoring .

5} Prebiotic:

Probiotics have become better known for their inhibitive roles against Kidney stone disease, with oxalate-degrading bacteria (e.g., Oxalobacter spp., Lactobacillus spp., and Bifido bacterium spp.) especially taking the lead in this effort due to increasing evidence that seems to favor such insinuation [138,139,140,141,142,143,144,145].O.formigenes is an anaerobic, oxalate- degrading bacterium; as such, these probiotics are thought to reside in the intestinal tract. A recent study has shown that subjects suffering from urinary stone disease possess less abundant lower-relative of O. formigenes in their intestinal tracts than do healthy subjects [146].Interestingly, O. formigenes abundance was shown to inversely correlate with urinary oxalate concentration [146]. It should be noted that oxalate homeostasis in the gastrointestinal tract is a consequence of the combined action solobrist O. formigenes and the numerous other microbiota [147]. Besides O. formigenes, the intestinal oxalate can also be efficiently degraded by other probiotics such as Lactobacillus spp. and Bifido bacterium spp. and reduce urinary oxalate excretion [148,149]. More recently, a study in the Drosophila melanogaster model of urolithiasis reported that Bacillus subtilis reduces CaOx stone formation [150]. Also, the gut diversity and abundance of microbiota have been noted to be significantly influenced by dietary patterns/styles from recent investigations [151]. Thus, dietary habits could affect the action of gut probiotics against KSD prevention.

6}Increse fluid Intake :

It is well recognized that dehydration is a universal risk in KSD. Increasing one's fluid intake in an effort to manage hydration status and reduce urinary concentration has been a long-standing recognized recommendation for KSD prevention. Diuresis refers to the process of increasing urinary volume achieved either by perforation of normal equilibrium of urine output or by drugs that increase urine flow. Increased urine flow rate and/or reduced water re-absorption by diuretic process then relates to increased urine volume and decreased urine osmolarity. Note that the AUA, EAU, CUA, and UAA uniformly recommend maintaining

urine output >2.0-2.5 L/day with fluid intake at 2.5-3.0 L/day [[92][89][91][69]]. Clinical trials have demonstrated the association between increased urinary volume with lower risk of recurrent calcium nephrolithiasis [152]. Therefore, a strategy for KSD prevention is to consume large volumes of water [152,153]. Over the last decade, increased fluid intake has uniformly been validated as a method for preventing KSD [154,155,156].

***Pharmacological Management :-**

When dietary measures are ineffective or inappropriate for patients with unique metabolic conditions, pharmacotherapy may assist in the reduction of stone recurrence. A systematic review and meta-analysis involving 21 randomized controlled trials and 2168 participants highlighted the greater efficacy of pharmacological intervention over dietary measures in patients with recurrent idiopathic calcium stones [157].Among others, the authors noted that the benefit derived from pharmacotherapy was only apparent for those with two or more previous stone episodes .Even though multiple randomized controlled trials have Shown that pharmacotherapy woks against stone recurrence, compliance toward prescribed drug therapy has often been less than optimal. In a study using medical claims data, Dauw and colleagues [158] revealed that adherence to the drug regimen for stone prophylaxis was only 30.2% among those prescribed medications for stone prevention. Other combinations of drugs, female gender, having lower quality health insurance and residing in the southern or northeastern United States were independently associated with lower like lihoods of adherence. However, older age and salaried employment appeared to favorably predict adherence. Adherence rates, on the other hand, largely varied according to the prescribed medication, with only 13% of patients adhering to citrate therapy as opposed to thiazides or allopurinol,42.5% and 40%, respectively.

Medication	Rationale	Dose	Specifics/ sideEffects	Monitoring
Calcium oxalate stones:				
Thiazide	Hypercalciuria	Hydrochlorothiazide 25-50mg BID , Chlorthalidone 2550mg/day, Indapamide 1.255mg/day	Hypokalemia, hyperlipidemia, hyperuricemia, hyperglycemia, hypocitraturia, hyperuricosuria, fatigue,erectile dysfunction .	BMP, uric acid , lipid profile
Potassium citrate (oral)	Hypocitraturia, low urine pH	10-30Eq BID	GI side effect	Serum creator & potassium
Potassium citrate (liquid)	Enteric hyperoxaluria, chronic diarrhea	15–30 mEq TID– QID (titrate to reduce oxalate)	GI side effect take with two largest meals	Serum creatinine& potassium
Allopurinol	Hyperuricosuria	100-300mg/day	Hypertransami—nasemiastevens – Johnson syndrome .	Liver enzyme
Uric acid stone :				
Potassium citrate (oral)	Alkalinization	10-30mEq BID (titrate dose to pH 66.5)	GI side effects	Serum creatinine& potassium
Sodium bicarbonate	Alkalinization	650mg BID-QID	Increased sodium load may increase risk of calcium stone	BMP
Allopurinol	Hyperuricosuria 2nd line therapy when alkalinization not successful	10-300mg/day	Hypertransami—nasemiastevens – Johnson syndrome	Liver enzyme
Cystine Stone				

Tiopronin (α-MPG) Potassium citrate (oral)	Increase cystine solubility Alkalinization	Initial 400mg/day titrate to effect 10-30mEq BID (titrate	Hematologic effects, tachyphylaxis, proteinuria, nausea,diarrhea, vitamin B6 deficiency (longterm use) GI side effects	CBC, BMP, urine protein serum creatinine& potassium
		dose to pH 7–7.5)		
Struvite stones				
Acetohydroxamic acid	Urease inhibitor	250mg BID-TID.	Headache, anemia, thrombophlebitis, rash, tremulousness	CBC
BMP- basic metabolic profile; CBC- complete blood count; GI- gastrointestinal; MPG-mercaptopropionyl glycine. ® A First line therapy				

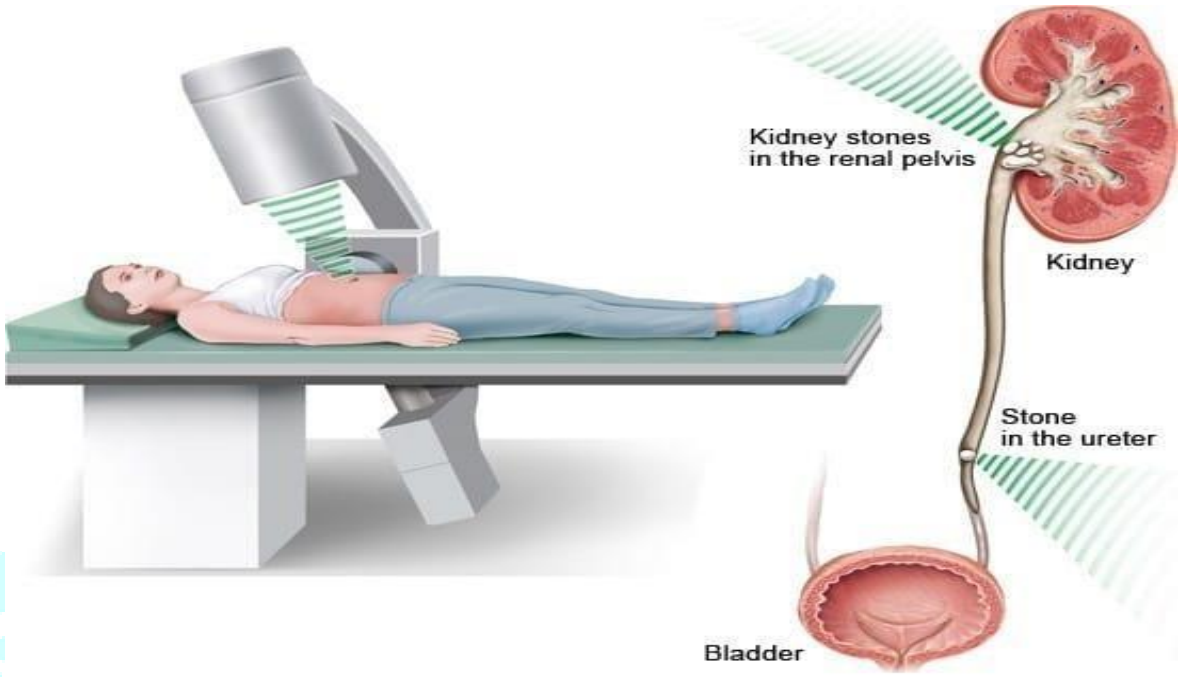
Ultrasound shock wave therapy :

In ultrasound shock wave therapy, sound waves are used to break up the stones. The stone fragments are then flushed away in the urine. This treatment is also referred to as extracorporeal shock wave lithotripsy (ESWL). A machine is used to send sound waves from outside of the body through the tissue to the stones. Shock wave therapy typically takes about 30 to 60 minutes when Treating simple kidney stones without complications. It is often possible to do so without having to spend the night in hospital. The outcome the treatment can be checked by means of ultrasound or x-ray scans.Shock wave therapy is especially suitable for kidney stones smaller than 20millimeters in diameter. If the stones are in the upper third of the u reter, they should not be any bigger than 10 millimeters,.

The removing stone through an Endoscopic procedure:

The extracting stone by an Endoscopic procedure:

There are two traditional techniques for the extraction of stones by surgical procedures: ureterorenoscopy (URS) and percutaneous nephrolithotripsy(PCNL or PNL).



- Ureterorenoscopy (URS)

Here, extremely small instruments are passed through the urethra (the passageway that urine exits from) and bladder with an endoscope and then nudged up into the ureter where the stone is located. There the stone is either mechanically pulverized or using a laser so that the pieces can be flushed out in the urine or removed using the endoscope. URS is used for stones that are bigger than 10 millimeters in diameter and are in the middle or lower third of the ureter. Kidney stones up to 20 millimeters in diameter are often removed using URS.

- Percutaneous nephrolithotripsy (PCNL):

In this method, an endoscope is moved into the renal pelvis or the kidney through a small cut made on your back. There the stones can also be either broken up mechanically or with a laser. Tiny forceps are used to remove the pieces of the kidney stones. This method is mainly used to treat kidney stones greater than 10 millimeters in diameter. PCNL is now often performed as mini percutaneous nephrolithotripsy (mini PCNL). The procedure then uses a Much smaller endoscope and a much smaller passageway for the instruments. The stones are not removed by forceps but are pulverized with a laser and then flushed out. Both these procedures require general anesthesia and an almost negligible hospital stay.

Conclusion :

Kidneystone disease KSD continuous to be a significant and costly health issue around the globe .Although there are successful treatment, the high recurrence rate of stone indicates the need for prevention. The etiology of KDS is multifactorial , and an approach must include lifestyle and dietary change , drug therapy , and investigation of natural bioactive compound . higher fluid intake, stone specific diet , and innovation in significantly , pharmacotherapy can also reduce the burden of KSD . emerging evidence on probiotic, bioactive compound , and targeted therapies is also brining promising avenues toward reducing recurrence and improving patient outcomes. Further research should aim at focusing personalized strategies that consider

the individual and AMP ;#39; risk factor and metabolic profiles so that prevention and management of kidney stone may ultimately improve .

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