INTRODUCTION

Urolithiasis is a very common and complex disease which its incidence is increasing and varying across sex, race and age. Environmental and climatic factors in addition to changes in dietary practices have effect on the urinary tract stone formation ***(Romero et al., 2010).***

Ureterorenoscope (URS), extra corporeal shock wave lithotripsy (ESWL), medical expulsive therapy (MET), open and laparoscopic ureterolithotomy are the treatment modalities for ureteric stones according to stone size and site ***(Argyropoulos et al., 2010).***

Ureterorenoscope is the most used in management of ureteric stones because it is minimally invasive modality**.** It has two types, semirigid and flexible. Semirigid ureteroscope is the best choice for treatment of distal ureteric stones but flexible ureteroscope has a higher stone free rate in the management of proximal ureteric stones, intrarenal stones and calyceal stones ***(Cohen et al., 2013).***

Stones not amenable for retrieval must be disintegrated by lithotripsies then extracted by forceps and baskets. Lithotripsies are many types as Ultrasonic lithotripsy, Laser lithotripsy, electrohydraulic lithotripsy and Pneumatic lithotripsy ***(Rebuck et al., 2011).***

Laser is abbreviation to Light Amplification by Stimulated Emission of Radiation. The theory of stimulated emission was proposed for the first time by Albert Einestein in 1916. That theory depended on that photons of the light could stimulate emission of other photons from excited atoms ***(Hecht et al., 2010)***.

Holmium: yttrium aluminum garnet (Ho: YAG), The frequency-doubled double-pulse Nd:YAG (FREDDY), Pulsed dye and Alexndrite are the types of Laser used for stone disintegration in Urology ***(Bader et al., 2012).***

Ho: YAG laser is the most widespread used laser lithotripsy. It can disintigrate all stones regardless their compositions, hardness and sizes in all sites of urinary tract ***(Kronenberg et al., 2012).***

Ho: YAG laser has advantages such as, it has small fibers which allows it to be used in all ureteroscopes. Also, it has photothermal mechanism of action and its energy is absorbed by water with wavelength 2140 nm ***(Sea et al., 2012).***

Laser fibers are damaged with energy > 1 Joul due to thermal effect. The size of fragment is a problem that challenge the urologists because it affects stone free rates (SFRs), as small fragment size result in high SFRs ***(Sea et al., 2012).***

The different techniques of Laser lithotripsy as dusting and basketing depends on Laser lithotripsy settings. Stone dusting

settings are decreasing energy and increasing frequency resulting in converting a stone to dust or tiny fragments so that to allow these tiny fragments to pass spontaneously**.** Stone basketing is to convert the stone to visible fragments which can be extracted with forceps and baskets by increasing energy and decreasing frequency ***(Chew et al., 2016).***

Dusting has some advantages over basketing such as decreasing ureteral wall injury rate during the procedure. Also, dusting technique is less exhaustive and has a less operative time than basketing. On the contrary basketing takes the lead in stone free rate and there is no need for secondary procedure which is more likely to happen in dusting ***(Schatloff et al., 2010).***

AIM OF THE WORK

Is to compare between the outcome and complications of distal ureteric stone laser disintegration using dusting vs. basketing technique with focussing on stone free rate.

URINARY CALCULI

Epidemiology

Urolithiasis is a complex disease, so an understanding of the epidemiology or pathogenesis helps in management of this disease ***(Romero et al., 2010).***

Prevalence

Stone disease is common with the lifetime risk of stone formation exceeding 12% in men and 6% in women ***(Romero et al., 2010).***

Incidence

Incidence rates defined as the onset of an individual's first kidney stone. And it varies by age, sex, and race. As with prevalence, the incidence rates are highest in males, it begins to rise after age 20, peaks between 40 and 60 years and then declines. For females, incidence rates seem to be higher in the late 20s and then decrease by age 50, then remaining relatively constant. Prevalence and incidence rates were highest for whites followed by Hispanics, blacks and Asians ***(Curhan, 2007).***

Recurrence rates

If left untreated the likelihood of forming another stone is 30–40% at 5 years. The recurrence depends on many factors

including stone type and urinary composition. But, treatment can reduce recurrence rates by 50% or more ***(Trinchieri, 2008).***

Classification of stones

Urinary stones can be classified according to size, location, X-ray characteristics, etiology of formation, composition and risk of recurrence.

1. Stone size

Stone size is usually given in one or two dimensions and stratified into those measuring up to 5, 5-10, 10-20 and > 20 mm in largest diameter ***(Leusmann, 1990)***.

2. Stone location

Stones can be classified according to anatomical position: upper, middle or lower calyx; renal pelvis; upper or distal ureter; urinary bladder and urethra ***(Hesse et al., 2003)***.

3. X-ray characteristics

Stones can be classified according to its appearance in the plain X-ray [kidney-ureter-bladder (KUB) radiography] (Table 1). This appearance varies according to composition. Non-contrast-enhanced computer tomography (NCCT) can be used to classify stones according to density (Houns-Field Unit) which can affect treatment decisions ***(Hesse et al., 2009)***.

Radiopaque	Poor radiopacity	Radiolucent
Calcium oxalate	Magnesium ammonium phosphate	Uric acid
Calcium phosphate	Cystine	Ammonium urate
	Apatite	Xanthine
		2,8-dihydroxyadenine
		Drug stones

Table 1: X-ray characteristics of the stones ***(Hesse et al., 2009).***

4. Aetiology of stone formation

A precise causative factor is not identified in most cases. A family history of kidney stones (increases risk by three times), insulin resistant states, a history of hypertension, primary hyperparathyroidism, a history of gout, chronic metabolic acidosis and surgical menopause are all associated with increased risk of kidney stones ***(Mattix et al., 2003).***

i. Low urine volume

Whatever the type of stone, low urine volume is often present. Unselected first time stone formers have lower 24-h urine volume than age and sex-matched controls ***(Borghi et al., 1996)***.

The proportion of individuals with such low urine volumes has been reported to be in the neighborhood of 70%. Low urine volume has also been felt to be an important contributing factor to the formation of uric acid stones in patients with intestinal disorders. Together with low urine volume, Hypercalciuria, hyperoxaluria, or hyperphosphaturia will tend to result in supersaturation of stone forming constituents, thereby promoting nucleation if the upper limit of metastability for that species is exceeded or the continued growth of stones that have already formed ***(Worcester et al., 2002).***

ii. Hypercalciuria

Hypercalciuria, present in 25–60% of stone formers, if not offset by increased urine volume or citrate excretion, will lead to increased supersaturation for calcium oxalate or phosphate. Among these individuals with hypercalciuria is a minority with unrecognized metabolic causes of increased urinary Ca excretion. These include primary hyperparathyroidism, granulomatous diseases, primarily sarcoidosis, Vitamin D intoxication, milk-alkali syndrome and the use of carbonic acid inhibitors ***(Vishal and Jack, 2011).***

Many hypercalciuric stone formers have what has been referred to as idiopathic hypercalciuria. Briefly, hypercalciuria is the most common metabolic abnormality found in patients with recurrent calcium stones. It is most often familial and

idiopathic and is strongly influenced by diet ***(Worcester et al., 2008).***

Patients typically have excessive intestinal calcium absorption and may also have decreased renal tubular calcium reabsorption and decreased bone mineralization. The etiology of this systemic disorder in calcium transport has, in hypercalciuric stone-forming rats and in humans, been linked to an excessive number of receptors for vitamin D by some but not others. There is also evidence for association with base substitutions in a soluble adenylate cyclase on human chromosome 2 in humans with this disorder related to increased intestinal absorption of calcium ***(Reed et al., 2002).***

iii. Hyperoxaluria

A study suggests that a diet characterized by normal calcium, low animal protein, and low salt levels is more effective than the traditional low-calcium diet for the prevention of recurrent stones in men with idiopathic hypercalciuria ***(Borghi et al., 2002).***

This appears to be due to a salutary effect of calcium in the diet on oxalate absorption. Hyperoxaluria promotes stone disease, either by virtue of its pronounced effect on calcium oxalate supersaturation or because of injurious effects of oxalate on the renal epithelium ***(Tungsanga et al., 2005).***

Hyperoxaluria is noted among patients with recurrent calcium stones more often than among those without the condition, possibly due to increased oxalate absorption in the gut. The intake of a high level of protein may increase oxalate production ***(Voss et al., 2006).***

Hyperoxaluria may be due to genetic overproduction or increased absorption due to ingestion of foods high in oxalate or its precursors, intestinal disorders or bowel resection (including gastric bypass surgery) ***(Taylor et al., 2008).***

iv. Phosphaturia

There have been several recent investigations looking at phosphaturia in subjects with urolithiasis. A study has described a mutation in the NHERF1 gene responsible for decreased renal phosphate reabsorption. It has been postulated that the associated hypophosphaturia causes increased 1, 25-dihydroxy-vitamin D production, which causes increased intestinal phosphate and calcium absorption. This combined hypercalciuria and hyperphosphaturia favor the formation of calcium phosphate precipitates that can result in nephrolithiasis ***(Vishal and Jack, 2011).***

v. Hyperuricosuria

Hyperuricosuria is a risk factor both for the development of stones composed of uric acid or its various salts, sodium urate and ammonium urate, as well as the development of

calcium oxalate or calcium phosphate stones. Often from high dietary intake of purines, is thought to promote the formation of calcium stones by reducing the solubility of calcium oxalate ***(Coe et al., 1979).***

Even stones that are composed primarily of uric acid frequently have components of the Ca salts. Low urine volume has been implicated in the uric acid stones that occur in chronic diarrhea and that result from excessive exercise or exposure to very warm ambient conditions ***(Worcester et al., 2002).***

vi. Alkaline urine pH

Urine pH that is alkaline more of the time is likely responsible for the minority of calcium stone formers whose stone are composed predominantly of calcium phosphate ***(Pak et al., 2004).***

Other conditions where acidification of the urine is compromised, such as in medullary sponge kidney, hyperparathyroidism, use of carbonic anhydrase inhibitors or carbonic anhydrase deficiency and in hereditary and acquired forms of renal tubular acidosis ***(Hildebrandt et al., 2001).***

Of course, some of these conditions induce nephrocalcinosis as well as urolithiasis, and the highest urinary pH values are associated with struvite, magnesium ammonium phosphate, rather than predominantly calcium stone formation,

although carbonate apatite may be formed as well ***(Griffith et al., 1976).***

vii. Hypocitraturia

Hypocitraturia has a wide range in reported prevalence which may be due to differences in the populations studied, differences in dietary background, and differences in the laboratory definition of hypocitraturia. Generally, it is between 30–40% of stone formers, but values as low as 8% to almost 70% have been reported ***(Domrongkitchaiporn et al., 2006).***

In only a small proportion of patients can this abnormality be ascribed to renal tubular acidosis and chronic diarrhea syndromes. Most appear to be dietary in origin; the different proportions among stone patients likely being explained by ethnic variations in food intake, specifically fruit. Hypocitraturia as an isolated abnormality is not common among stone formers but is often accompanied by other defects such as hypercalciuria and hyperoxaluria ***(Levy et al., 1995).***

In general Hypocitraturia is found in conditions that acidify the proximal tubule cell by one means or other, perhaps related to high protein diets ***(Amanzadeh et al., 2003).*** These diets, while not resulting in frank metabolic acidosis, may lower the serum bicarbonate to a small degree and, thus, induce hypocitraturia. The transporter responsible for proximal tubule citrate reabsorption is stimulated by metabolic acidosis, which may be the proximate cause of hypocitraturia ***(Aruga et al.,***

2000). Occasionally, no underlying defect can be uncovered and the patient is diagnosed to have idiopathic hypocitraturia ***(Levy et al., 1995).***

Stones can be classified into: infectious, non-infectious, genetic, or drug stones

A. Non-infection stones

- Calcium oxalate
- Calcium phosphate (including brushite and carbonate apatite)
- Uric acid

B. Infection stones

- Magnesium ammonium phosphate
- Ammonium urate

C. Genetic causes

- Cysteine
- Xanthine
- 2, 8-dihydroxyadenine

D. Drug stones

- Indinavir stones

(Leusmann, 1990)

Mechanisms of stone formation

We can say that urine is saturated, for example with, calcium and oxalate, when their concentrations exceed the solubility product. Below the solubility product, crystals will not form as the urine is under saturated ***(Kok, 1990)***.

Above the solubility product, crystals of calcium and oxalate logically should form, but because of the inhibitors of crystal formation they don't. However, inhibitors of crystallization become ineffective, and crystals start to form above certain concentration. The concentration at which crystallization starts is known as the formation product and the urine is said to be supersaturated with the substance ***(Kok, 1990)***.

Inhibitors of crystallization like citrate, magnesium, glycosaminoglycan and Tamm Horsfall protein increase the ability of urine to hold more solute in solution than pure water. On the contrary, dehydration causes super saturation of urine which leads to crystallization. Periods of intermittent super saturation of urine can occur as a consequence of dehydration and following meals ***(Borghi, 1996).***

Nucleation is the earliest phase of crystal formation. Nuclei usually form on the epithelial cells or on other crystals. Then nuclei form into clumps a process called aggregation ***(Borghi, 1996).***

5. Stone composition

Metabolic factors are important in stone formation. Stone analysis is the basis for further diagnostic and management decisions (Table 2) ***(Leusmann, 2000)***.

Chemical composition	Mineral
Calcium oxalate monohydrate	Whewellite
Calcium oxalate dehydrate	Wheddelite
Basic calcium phosphate	Apatite
Calcium hydroxyl phosphate	Hydroxylapatite
b-tricalcium phosphate	Whitlockite
Uric acid dehydrate	Uricite
Ammonium urate	
Sodium acid urate monohydrate	
Magnesium ammonium phosphate	Struvite
Magnesium acid phosphate trihydrate	Newberyite
Carbonate apatite (phosphate)	Dahllite
Calcium hydrogen phosphate	Brushite
Calcium carbonate	Aragonite
Octacalcium phosphate	
Cysteine	
Xanthine	
2,8 dihydroxyadenine	
Drug stones	
Foreign body calculi	

Table 2: Stone composition ***(Keoghane et al., 2010)***

Risk groups for stone formation

The risk status of stone formation is important because it defines the probability of recurrence, and for pharmacological