wjpmr, 2017,3(10), 66-92

SJIF Impact Factor: 4.103

WORLD JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.wjpmr.com

Research Article

ISSN 2455-3301

WJPMR

CORRELATION OF THE CHEMICAL COMPOSITION OF KIDNEY STONES WITH THE DIET STYLE, GEZIRA STATE, SUDAN, MARCH 2016, SEPTEMBER 2016

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Article Received on 01/09/2017 **Article Revised on 22/09/2017** **Article Accepted on 13/10/2017**

ABSTRACT

Kidney stones are the most common disease of the urinary tract affecting about 10% of the global population. These unwanted mineral aggregates can result in extreme pain and morbidity, and in some cases may lead to high blood pressure and increase the risk for coronary artery disease and diabetes mellitus. Kidney stone disease may occur from either derangements of urine biochemistries or anatomic abnormalities of kidneys and urinary tract. Genetic, environmental and dietary factors may also cooperate in the patophysiology of nephrolithiasis. The stones cause severe pain and are also associated with morbidity and kidney damage. There is also no clear understanding on the relative metabolic composition of kidney stones. The purpose of this study was to define the relationship between dietary habits and incidence of stone formation and to perform the chemical analysis of stones to know the pattern of chemical composition of stones in Sudanese subjects. Forty eight renal stone samples were analyzed during the period of January – June, 2015. Those patients were referred to Gezira Hospital for Renal Disease and Surgery (GHRDS), Gezira State, Sudan. The stones were analyzed by semi quantitative method in Gezira Central Laboratory. Kits were used for chemical analysis of stones. Calcium, Phosphate, Oxalate, Uric acid, carbonate and cystine were determined. The results of this study revealed that, male to female ratio was 2:1,Majority of the patients were non-vegetarian 89.5% and many of them 56.2% consumed more salted foods. Water consumption amount revealed that, 45.8% of the patients consumed 1.0 liter of water per day. Frequency of consumption of food stuffs revealed that it varied from weekly to rarely for most of the items from various selected categories. Calcium (%72.8), oxalate (%60.4) and uric acid (%27) were the most common, phosphate (10%), Carbonate (6%) and cystine which found only in (2%) of the study samples. The relative frequency of calcium oxalate stone were about (70.8%) out numbers other types which was compatible with the international literature. While there is relative high percentage of uric stones were about (%29.1) which can be explained by the high animal protein consumption in Sudan together with the hot weather which can be a cause of highly concentrated urine. The study recommends analyzing the kidney stones routinely for both sexes for better understanding of the mechanisms involved in lithogenesis.

KEYWORDS: Kidney stones, patophysiology of nephrolithiasis.

1. INTRODITION

Kidney stones are inorganic crystalline aggregates enmeshed in about 5.0% organic matrix (Tang *et al.*, 2006). This disease has tormented humans since the earliestrecords of civilization, as many as 10.0% of men and 3.0% of women have a stone during their adult lives (Coe *et al*., 1992). In majority of kidney stones, calcium oxalate is the main constituent (80.0%) and calcium phosphate is present in amounts ranging from 1.0% to 10.0% ; 10.0% of struvite, 9.0% uric acid(UA) and the remaining 1.0% are composed of cysteine or drug-related stones Coe *et al.*, (1992), Mandel and Mande (1989). Kidney stone is the complex phenomenon and not completely understood till now. No single factor contributed in pathophysiology of stone formation but may be due to, infections, hormonal influences, metabolic disturbances, diet factors or obstructions in the urinary system or increasing excretion of chemical components such as oxalate, calcium, carbonate, magnesium, phosphate, and cystine etc. The prevalence of calcium oxalate stones has been constantly increasing during past fifty years in industrialized as well as in developing countries and varies depending on race, sex and geographic locations (Daudon, 2005). The prevalence of renal stones in men varies from 4.0% to 9.0% and in women it ranges from 1.7 % to 4.0%(Coe *et al*., 2004). Although kidney stones can be traced to the earliest antiquity of human history, the primary causative

factors remain obscure, but renal stones are suspected to have direct relationship to the composition of urine, which is mainly governed by nutrition and environment (Chandrajith *et al.*, 2006).Stone composition, stone location, incidence, age and sex distribution differs according to geographical area. Such differences have been explained in terms of race, diet and climate factors (Lopez and Hoppe, 2010). Sudan is a large country, so it has many differences in factors which had been explained before. So we thought, these factors may affect the chemical composition of the stones. Kidney stones and chronic kidney disease (CKD) are common, affecting 5% and 13% of the adult population, respectively (Lopez *et al.*, 2001). There are several factors that increase the risk of developing kidney stones, such as hypercalcemia, hypercalciuria, hyperuricemia, hyperuricosuria, hyperoxaluria, hypocitraturia, pH of urine (Levy *et al*.,1995; Wagner and Mohebbi, 2010). In most patients, both the stones and biochemical anomalies are caused by the interaction of genetic factors and the exposure to the environment. Genetic factors are responsible for half the risk of developing(Attanasio, 2011).

The occurrence of renal stone disease is related to food habits of individuals. Dietary factors include a high intake of animal proteins and oxalates and a low intake of potassium Containing citrus fruits and fluids (Tur *et al.*, 1991). Inadequate fluid consumption decreases totalurinary volume thereby increasing the concentration of stone forming salts. Intake of sodium is also associated with increased risk of stone formation presumably because of increased urinary calcium excretion (Carbone *et al.*, 2003). Effect urine chemistries: low urine pH, high urine calcium and uric acid excretion and low citrate excretion (Daudon, 2005; William and Chisholm, 1976). As consequences lead to urinary crystals and the renal stone formation. Stones may occur in the kidney, ureter, urinary bladder, prostate, or urethra. Kidney Stones mainly affects adults, predominantly males. The types of stone formed depend mainly on the urine composition, which, in turn, reflects the type of diet consumed. Renal calculi are characterized clinically by renal colic as they pass down along the ureter and manifest as haematuria (Rennke, 1999). In Sudan, Kidney Stones is very frequent, but stone analysis is not routinely performed.

General objective

To detect the most predominant risk factors associated with formation of kidney stones in Sudanese patients presenting to Gezira Hospital for Renal Disease and Surgery in Gezira State, Sudan.

Specific objectives

- To detect the relationship between dietary habits and incidence of stone formation
- To detect the demographic factors of patients
- To determine quantitatively some physical and chemical parameters (pH, EC, TDS, Chloride, Calcium, Sulphate, Carbonate, Bicarbonate, Potassium and Sodium concentrations) in drinking water of tap and well in some location of Wad Medani Town, Gezira state, Sudan.
- To perform the chemical analysis of stones to know the pattern of chemical composition of stones in Sudanese subjects.

2. LITERATURE REVIEW

2.1 Kidney stones

The majority of people are born with two kidneys. Kidneys are bean shaped organs located in the spine. The kidneys form urine which then flows through tubes called ureters to be stored in the bladder their main function is to filter the blood from harmful chemicals known as toxins. These emptied through the urethra (Moe, 2006). Increased incidence of kidney stones, and is strongly associated with race or ethnicity and region of residence (Stamatelou *et al.*, 2003). A seasonal variation is also seen, with high urinary calcium oxalate saturation in men during summer and in women during early winter.

Stone keeping patients suffer a severe pain, blood in urine and fever. The pain is characterized by its severity. Usually pain is centralized in the back or sides and sometimes moves as the stone moves during an attack. Many sufferers experience nausea and vomiting. The size of the stone does not depend on the intensity of the pain but generally larger irregularly shape stone cause intense pain. Much pain in kidney stone patients is due to the muscle contractions as the ureter attempts to force the stone into bladder. If patients have fever with other symptoms it means he/she may have an infection. Urinary stone are of several different compositions such as calcium stones, struvite stones, uric acid stones etc. Bacteria that infect the urinary tract secrete enzymes that increase the level of ammonia in the urine. The ammonium ion thus formed in turn creates the crystals of magnesium ammonium phosphate. The main causes of stones formation are super saturation, lack of inhibitions and matrix (Parks *et al*., 2003).

2.1.1 Definition

Kidney stones also indexed as: Renal Calculi, Urinary Calculi, Urolithiasis (Uro refers to urine), Nephrolithiasis (Nephro refers to the kidney, and -lith means stone) (Parmar, 2004). Kidney stones may be as small as grain of sand or larger than a golf ball. Shape, color, size of calculi depends on their chemical composition; they may be smooth, round, jagged, spiky or asymmetrical. Most stone are yellow to brown in color, although variations in chemical composition can produce stones that are tan, gold or black some stones known as stag horn stones can fill the entire kidney these are usually caused by infection. Most stones are formed of calcium a very common chemicals in dairy products other chemicals such as oxalate, uric acid, and cystine can also cause stones (Khan *et al.*, 1983). Kidney stone disease may occur from either derangements of urinebiochemistries

or anatomic abnormalities of kidneys and urinary tract. Genetic, environmental and dietary factors may also cooperate in the pathophysiology of nephrolithiasis. The stones cause severepain and are also associated with morbidity and renal damage. There is also no clear under standingon the relative metabolic composition of kidney stones (Lopez and Hoppe, 2010).

2.1.2 Distribution of Kidney stone disease

Kidney stone is the 3rd most common clinical problem worldwide (Farooq *et al*., 2007) presenting up to (15%) of population in the western countries. The prevalence of its distribution is variable across the world. Avery high incidence area includes Scandinavian countries, Mediterrenanian, British Isles, Australia, and central Europe, some parts of Malaysia, China, Pakistan, and Western India. In our continent the most common stone belt areas includes Myanmar, Sudan, Thailand, Indonesia, Philippines Saudi Arabia, UAE, Pakistan and India(Abbagani *et al*., 2010).

2.1.3 Types of Kidney stone

There are several varieties of Kidney stones. The most common type is calcium oxalate. Other types include uric acid, struvite and phosphorous. After your stone has been treated or passed, your doctor will check your urine for 24 hours and do blood tests. The results of these may show if excess of certain types of minerals are being spilled into your urine or are circulating in your blood (Bauzá *et al*., 2007).

2.1.3.1 Calcium stones

This is the most common type of stone. These stones cannot be dissolved with medicine. The only treatment is letting the small ones pass and treating the larger ones with ESWL or surgery. After the stones have been passed or removed, then preventing further formation is the priority. In addition to increasing fluid intake, citrate in the form of lemonade or citrate pills (Uro Cit-K, K-dur) should be added. Findings from the 24-hour urine collection or blood work may indicate additional medications to prevent further stone formation (Bauzá *et al*., 2007).

2.1.3.2 Uric acid stones

This type of stone forms in patients with a metabolic urine abnormality such as gout this stone does not show up on regular X-Rays and therefore, CT or ultrasound is needed to find them. Fortunately, uric acid stones can sometimes be dissolved with bicarbonate. If dissolving the stone is unsuccessful, then ESWL or surgery may be necessary (Bauzá *et al*., 2007).

2.1.3.3 Struvite stones

Struvite stones are composed of magnesium ammonium phosphate, are also known as 'infection stones', and account for 15%-20% of all stones. Certain bacteria, such as Proteus mirabilis and Ureaplasma urealyticum, secrete the enzyme urease which hydrolyses urea to carbon dioxide and ammonium ions. This reaction causes the urinary pH to rise. As mentioned previously, *E.coli* was shown to decrease urokinase and increase sialidase activity, causing increased matrix production, thereby leading to crystal adherence to the renal epithelium. This explains how non-urease producing bacteria may be associated with struvite stones. Struvite calculi also account for the majority of staghorn stones was first identified by Ulex, a Swedish geologist, in the 18th century. He created the term 'struvite' in honor of his friend and mentor H.C.G von Struve (1772-1851), a Russian diplomat and natural scientist. Brown suggested that bacteria split urine and thus caused and facilitated stone formation. He also isolated *Proteus vulgaris* from a stone nucleus and this is known today to secrete urease. However it was Hager and Magath, in 1925, who suggested that 'urease' was the cause of hydrolysis of urine. Struvite stones account for 5-20 % of all urinary stones. Data from the 1970s indicated that struvite stones constituted 15% of all stone specimens sent for calculus analysis but Rodman (1999) suggested that this figure over-reported the incidence of infection stones, since many small spontaneously passed calculi are never caught and do not have their chemical composition determined. 15-20 % of urinary stones were infection stones in industrial countries and their clinic results showed that out of 4,400 patients with urinary stones, 510 had infection stones (11.6 %).

2.1.3.4 cystine stones

Cystine kidney stones are due to cystinuria, an inherited (genetic) disorder of the transport of an amino acid (a building block of protein) called cystine that results in an excess of cystine in the urine (cystinuria) and the formation of cystine stones (Bauzá *et al*., 2007).

2.1.4 Classification and pathophysiology

Kidney stones are broadly categorized into calcareous (calcium containing) stones, which are radio-opaque, and non-calcareous stones. Pure uric acid and indinavir stones are radiolucent. Cystine stones are radio-opaque because of the sulphur content. Recent evidence indicates that formation of kidney stones is a result of a nanobacterial disease akin to *Helicobacter pylori* infection and peptic ulcer disease. Nanobacteria are small intracellular bacteria that form a calcium phosphate shell (an appetite nucleus) and are present in the central nidus of most (97%) kidney stones and in mineral plaques (Randall's plaques) in the renal papilla. Further crystallisation and growth of stone are influenced by endogenous and dietary factors. Urine volume, solute concentration, and the ratio of stone inhibitors (citrate, pyrophosphate, and urinary glycoproteins) to promoters are the important factors that influence crystal formation. Crystallisation occurs when the concentration of two ions exceeds their saturation point in the solution(Bauzá *et al*., 2007).On the basis of their composition, stones are classified as shown in (Table 2.1).

2.1.5 Epidemiology

The prevalence of urinary calculi is estimated to be 5 percent in the general population, with an annual

incidence of as much as 1 percent (Delvecchio and Preminger, 2003).Men are twice as likely as women to develop calculi, with the first episode occurring at an average age of 30 years. Women have a bimodal age of onset, with episodes peaking at 35 and 55 years. Without preventive treatment, the recurrence rate of calcium oxalate calculi increases with time and reaches 50 percent at 10 years (Menon and Resnick, 2002).

2.1.6 Pathophysiology

Renal calculi are crystalline mineral deposits that form in the kidney. They develop from microscopic crystals in the loop of Henle, the distal tubule, or the collecting duct, and they can enlarge to form visible fragments(Menon and Resnick, 2002). The process of stone formation depends on urinary volume; concentrations of calcium, phosphate, oxalate, sodium, and uric acid ions; concentrations of natural calculus inhibitors (e.g., citrate, magnesium, Tamm-Horsfall mucoproteins, bikunin); and urinary pH (Mandel, 1996).High ion levels, low urinary volume, low pH, and low citrate levels favor calculus formation. Risk factors and their mechanisms of action are listed in (Table, 2.2).

2.1.7 Epidemiological risk factors

2.1.7.1 Age and Sex

The disease affected all age groups from less than 1 year old to more than 70, with a male to female ratio of 2:1. The incidence of formation first kidney stone between the ages of thirty and seventy vary between approximately 100 – 300 per 100,000 per year in men and 50-100 per 100,000 in women i.e.6%–9% in males and 3%–4% in females (Johnson *et al.*, 1979; Hiatt *et al.*, 1982; Soucie *et al.*, 1994; Curhan *et al.*, 1997; Stamatelou *et al.*,2003). The peak age for the development of calcium oxalate stones was between 50–60 years. However the increased incidence of recurrence in patients in the older age may be attributed to the influence of ageing and diet. The relation between diet and kidney stones may be different in older adults.

Table 2.1: Kidney stones are typically classified by their location and chemical composition.

Kidney Stone type	Population	Circumstances	Color	Sensitivity	Details
Calcium oxalate	80%	when urine is alkaline (ph>5.5)	Black/Dark brown	Radio-opaque	Some of the oxalate in urine is produced by the body. Calcium and oxalate in the diet play a part but are not the only factors that affect the formation of calcium oxalate stones. Dietary oxalate is an organic molecule found in many vegetables, fruits, and nuts. Ca from bone may also play a role in kidney stone formation.
Calcium phosphate	5–10%	when urine is alkaline (high pH)	Dirty white	Radio-opaque	Tends to grow in alkaline urine especially when *Proteus* are present.
Uric acid	5–10%	when urine is persistently acidic	Yellow/ Reddish brown	Radiolucent	Diets rich in animal proteins and purines: substances found naturally in all food but especially in organ meats, fish, and shellfish.
Struvite	10–15%	infections in the kidney	Dirty white	Radio-opaque	Preventing struvite stones depends on staying infection-free. Diet has not been shown to affect struvite stone formation.
Cystine	1–2%	rare genetic disorder	Pink/Yellow	Radio-opaque	Cystine, an amino acid (one of the building blocks of protein), leaks through the kidneys and into the urine to form crystals.
Xanthin		Extremely rare	Brick red	Radio	

Table 2.2: Risk Factors for the Development of kidney stones

Risk factor	Mechanisms
Bowel disease	Promotes low urine volume; acidic urine depletes available citrate; hyperoxaluria
Excess dietary meat (including poultry)	Creates acidic urinary milieu, depletes available citrate; promotes hyperuricosuria
Excess dietary oxalate	Promotes hyperoxaluria
Excess dietary sodium	Promotes hypercalciuria
Family history	Genetic predisposition
Insulin resistance	Ammonia mishandling; alters pH of urine
Gout	Promotes hyperuricosuria
Low urine volume	Allows stone constituents to supersaturate
Obesity	May promote hypercalciuria; other results similar to excess dietary meat
Primary hyperparathyroidism	Creates persistent hypercalciuria
Prolonged immobilization	Bone turnover creates hypercalciuria
Renal tubular acidosis (type 1)	Alkaline urine promotes calcium phosphate supersaturation; loss of citrate

Source :(Miller *et al.*,2007).

The intestinal absorption of many nutrients that influence stone formation, such as calcium, may be reduced in the elderly. In men, the incidence of kidney stones declines markedly after 60 years of age (Hiatt *et al.*, 1982; Curhan *et al.*, 1993; Souice *et al*., 1994), suggesting that the pathophysiology of nephrolithiasis is different in the elderly. Older stone formers excreted less urinary calcium than their younger counterparts and may exhibit defects in urinary inhibitors of crystallization. Increased incidence in males also has been attributed to increased dietary protein intake, which increases urinary excretion of phosphates and magnesium and reduces urinary citrate concentration. The lower risk of stone formation in women was attributed initially to increased urinary citrate concentrations due to the lower urinary saturation of stone forming salts, while later reports indicated that endogenous estrogen and estrogen treatment in postmenopausal women may decrease the risk of stone recurrence by lowering urinary calcium and calcium oxalate saturation. Estrogen may also help to prevent the formation of calcium stones by keeping urine alkaline and raising protective citrate levels. Experiments in animals demonstrated that testosterone promoted crystal growth by suppressing osteopontin expression in the kidney and increasing urinary oxalate excretion while estrogen possibly inhibited stone formation by increasing osteopontin expression in the kidney and decreasing urinary oxalate excretion (Parmar, 2004).

2.1.7.2 Family history

The risk of becoming a stone former is more than 2.5 times greater in individuals with a family history of stone disease(Curhan *et al.*,1997).This higher risk is likely due to a combination of genetic predisposition as well as similar environmental exposures (e.g. diet). A polygenic inheritance has been proposed to account for the tendency to calcium oxalate stone formation in families(Resnick*et al*., 1968).While a number of genetic factors have been clearly associated with rare forms of nephrolithiasis, information is still limited on genes that contribute to risk of the common forms of stone disease.

2.1.7.3 Systemic disorders

Although nephrolithiasis has traditionally been considered a renal disorder, there is overwhelming evidence that it is in fact a systemic disorder. Primary hyperparathyroidism, renal tubular acidosis and Crohn's disease are well-described conditions that increase the risk of formation of calcium containing stones. Primary hyperparathyroidism may be found in 5% of stone formers(D'Angelo*et al*., 1997).

More recently, a number of other common conditions have been convincingly linked to nephrolithiasis. Increasing body size as assessed by weight, body mass index or waistline is associated with an increasing risk of stone formation independent of other risk factors including diet (Taylor*et al*., 2005).

The magnitude of the increase in risk from BMI is higher in women than in men. For example, the risk of stone formation for individuals with a BMI $\geq$ 30 kg/m^2 compared to those with a BMI 21–23 was 30% higher among men but nearly two-fold higher among women. Weight gain also increases the risk of stone formation. A 35 pound weight gain since early adulthood increased risk of stone formation by 40% in men and 80% in women. The mechanism(s) for the increased risk associated with larger body size is unknown at present. A history of gout increases the likelihood of forming kidney stones, both uric acid and calcium oxalate. In a national health survey, individuals with gout were 50% more likely to have a history of stones(Kramer and Curhan, 2002). When examined prospectively, a history of gout was associated with a doubling of the risk of forming a stone, independent of diet, weight and medications (Kramer*et al*., 2003).Although the mechanism for this relation is unknown, possibilities include insulin resistance and acid-base defects. More recently, diabetes mellitus was found to raise the risk of stone formation, independent of diet and body size (Taylor*et al*., 2005).Cross-sectionally, individuals with a history of type II DM were more than 30% more likely also to have a history of nephrolithiasis. Prospectively, a history of type II DM increased the risk of stone formation by 30–50% in women but not in men.

2.1.7.4 Geography

Kidney stone incidence varies in different parts of the world, high incidence areas are Scandinavian countries, Mediterranean countries, British Isles, northern Australia, central Europe, portions of the Malayan Peninsula, China, Pakistan and northern India where as the incidence of kidney stone formation is lower in areas like Central and South America, some parts of Africa. In Asia stone-forming belt has been reported to stretch across Sudan, Saudi Arabia, the United Arab Emirates, the Islamic Republic of Iran, Pakistan, India, Myanmar, Thailand, Indonesia and Philippines.The effect of geography on the incidence of stone formation may be direct, through its effect on temperature; high temperatures increase perspiration, which may result in concentrated urine, which in turn promotes increased urinary crystallization (Taylor*et al*., 2005).

2.1.7.5 Climate/Season

Renal stones are common in hot climates and the incidence was thought to be increased after the peak temperatures during the hot summer months.No significant seasonal variation in calcium stone formation. There was however an increase in uric acid stones formation during summer and autumn and a decrease in infectious stone formation during spring and summer.

Crystalluria was greater during summer months in patients who formed stones and postulated that increased sweating during high temperatures led to concentrated urine and urinary crystal formation. Uric acid and cystine stone-formers are further at risk since concentrated urine

tends to be more acidic, encouraging these compounds to precipitate. Increased vitamin D production during summer, due to increased exposure to sunlight, may lead to increased stone formation due to increased calcium excretion in the urine (Taylor*et al*., 2005).

2.1.7.6 Diet

Some reports have described that vegetarians are at lower risk for stone formation in contrast to non-vegetarians (Robertson *et al.*,1989).The role of animal protein and potassium intake in the etiology of calcium stone formation is paradoxical. Some studies have shown a positive association between animal protein intake and stones whereas others have not (Curhan *et al.*,1997). The consumption of a diet rich in animal protein (from meat, dairy, poultry, or fish), sodium (Muldowney *et al.,* 1982) and refined sugars increases urinary calcium and uric acid concentrations and lowers urinary citrate concentration. Kidney stones formers have been reported to process sugar abnormally by increasing urinary oxalate and urinary calcium as well (Lemann *et al.,* 1969). Dietary potassium restriction increases and potassium supplementation may decrease urinary calcium excretion (Lemann *et al.,* 1991). Calcium intake, particularly through milk and dairy products, may be associated with hypercalciuria and stone formation. However, inverse relationships between dietary calcium and stone formation have been demonstrated, in that groups of men and women with the highest calcium intake have been shown to have nearly one half the rate of stones as groups with the lowest intake (Curhan *et al.*,1993 and 1997). Dietary calcium binds in the intestinal lumen with dietary oxalate, forming an insoluble, non-absorbable complex. The reduction in urinary oxalate levels that occurs with increased intake of dietary calcium is proportionally more important than the increased urinary calcium levels. Like oxalate, some dietary calcium may also be less available (Curhan *et al.,* 1993). Animal protein induces stone formation; reports indicated operation of different mechanisms.Protein ingestion generates renal acid load that gives rise to metabolic acidosis where by theUrinary excretion of citrate is reduced and the excretion of calcium increased by bone resorption. There is also an inhibition of calcium reabsorption in the distal tubules caused by the acidosis. Further excessive intake of animal protein, increases the glomerular filtration rate and this hyperfiltration contributes to an increased urinary excretion of oxalate, calcium and urate (Curhan *et al.*,1997).

2.1.7.7 Body weight

Overweight condition and obesity was found in 59.2% of the men and 43.9% of the women and both these conditions were strongly associated with an elevated risk of stone formation in both genders due to increased urinary excretion of promoters but not inhibitors of calcium oxalate stone formation and further concluded that overweight and obese men are more prone to stone formation than overweight women. Similar study showed that obesity was associated with increased urinary concentrations of sodium, oxalate, uric acid, sulfate and phosphate in men.Excess body weight may be associated with various functional /structural lesions of the kidney and will lead to nephrolithiasis, glomerulomegaly, diabetic nephropathy, carcinoma of the kidney (Taylor *et al*., 2004). Body mass index (BMI) High animal protein intake leads to increased calcium and uric acid excretion as well as decreased urinary citrate (Breslau*et al* 1988). All of which increase the risk of stone formation. An increased risk of stone formation was observed for higher animal protein intake only among men with BMI < 25 kg/m^2 (Taylor *et al*., 2004). A higher intake of sodium (Muldowney e*t al*., 1982) or sucrose (Lemann *et al* 1969) increases calcium excretion independent of calcium intake, whereas potassium supplementation decreases calcium excretion(Lemann *et al.,* 1991)and many potassium-rich foods increase urinary citrate due to their alkali content. Prospective studies demonstrated sucrose was associated with an increased risk in women(Curhan *et al.*,1997 and2004) and higher dietary potassium intake decreased risk in men and older women (Curhan *et al.*,1993 and 1997*;* Taylor *et al.,* 2004). Recently, phytate was also found to reduce substantially the likelihood of stone formation in younger women (Curhan *et al.*,2004).

2.1.7.8Fluid Intake and Beverages

When the urine output is less than 1 L/day, risk of stone formation is markedly higher. Observational studies (Curhan *et al* 1993; 1997 and 2004) and a randomized controlled trial (Borghi*et al*.,1996) have demonstrated the importance of fluid intake in reducing the likelihood of stone formation. Patients with stone disease often ask what they should and should not drink. Despite previous beliefs to the contrary, observational studies have found that coffee, tea, beer, and wine are associated with a reduced risk of stone formation (Curhan *et al.*,1998 and 1999). Although citrus juices theoretically could reduce the risk of stone formation (Wabner and Pak, 1993) orange juice consumption was not associated with stone formation and grapefruit juice intake was associated with a 40% higher risk (Curhan *et al.*,1999 and1998). Grapefruit juice is known to have a number of effects on intestinal enzymes, but the mechanism for the observed increased risk is unknown. Previous studies suggested an increased risk for soda consumption and unadjusted results from observational studies also suggested an increased risk. However, after controlling for other dietary components, consumption of soda (with or without caffeine; diet or sugared) was not associated with the risk of stone formation (Curhan *et al.*,1999 and1998). Although skim and whole milk were not associated with risk in the observational studies probably because these studies adjusted for the intake of dietary calcium, milk intake likely reduces the risk of calcium kidney stone formation.

2.1.7.9 Dietary and non-dietary Factors

The composition of the urine is influenced by dietary intake and several dietary factors have been proposed to

modify the risk of nephrolithiasis. Nutrients that have been implicated include calcium, animal protein (Robertson *et al.*,1979), oxalate(Larsson *et al.*,1987), Sodium(Muldowney e*t al.*, 1982), sucrose(Lemann *et al.*, 1969),magnesium (Johansson *et al.*,1980), and potassium (Lemann *et al.*, 1991). Because patients who develop stones often change their diet, studies that retrospectively assessed diet may be hampered by recall bias. Other studies have examined the relation between diet and changes in the lithogenic composition of the urine, often using calculated relative supersaturation. However, the composition of the urine does not completely predict risk and not all the components that modify risk are included in the calculation of supersaturation (e.g. urine phytate). Thus, prospective studies are best suited for examining the associations between dietary factors and risk of actual stone formation.

2.1.7.9.1Calcium

The first prospective study of dietary factors and the risk of incident stone disease was performed in a cohort of more than 50,000 male health professionals aged 40 to 75 years at baseline (Curhan *et al.*, 1993).Although dietary calcium had been strongly suspected of raising the risk of stone disease, men with a higher intake of dietary calcium actually had a lower risk of incident nephrolithiasis independent of other risk factors. This inverse association has been confirmed in two other prospective studies in women (Curhan *et al.*, 1997 and 2004).And in an updated analysis in men (Taylor *et al*., 2004). Although the mechanism of this effect is unknown, low calcium intake is known to increase oxalate absorption and urinary excretion (Bataille *et al.*,1983).And individuals with lower calcium intake have lower 24 hour urine oxalate excretion. While the reduction in risk due to higher dietary calcium intake may be due to reducing urine oxalate, it is also possible that there is some other protective factor in dairy products (the major source of dietary calcium in the US). A subsequent study showed that low dietary calcium intake may increase the risk of stone formation, even among individuals with a family history of stones (Curhan *et al.*, 1997).The above mentioned observational data were subsequently confirmed in a randomized trial by Borghi and colleagues that compared a low calcium diet (400 mg/d) to a diet containing 1200 mg of calcium along with low sodium and low animal protein intake in men with hypercalciuria and calcium oxalate stones (Borghi *et al.*,2002).The rate of recurrence was reduced by 50% in the higher calcium intake group. While some authorities still question whether a high calcium diet reduces the risk of stone formation, there is overwhelming evidence that calcium restriction is not beneficial and may in fact be harmful, both for stone formation and bone loss. Despite similar bioavailability, the impact of supplemental calcium appears to be different from dietary calcium. In an observational study of older women, calcium supplement users were 20% more likely to form a stone than women who did not take supplements, after adjusting for dietary factors (Curhan *et al.*,1997). In younger women and men, there was no association between calcium supplement use and risk of stone formation (Curhan *et al.*,1993 and 2004).The discrepancy between the risks from dietary calcium and calcium supplements may be due to the timing of calcium intake. In these studies, calcium supplements were often taken in between meals, which would diminish binding of dietary oxalate. The recently published Women's Health Initiative randomized trial also demonstrated a 17% increased risk of stones with calcium supplementation (Jackson *et al*., 2006). However, these results should be interpreted cautiously as the participants were instructed to take their supplements with meals, and the supplements contained both calcium and vitamin D.

2.1.7.9.2 Oxalate

The role of dietary oxalate in the pathogenesis of calcium oxalate nephrolithiasis is unclear. The proportion of urinary oxalate derived from dietary oxalate is controversial; estimates range from 10 to 50%.In addition to the GI absorption of dietary oxalate, urinary oxalate is also derived from the endogenous metabolism of glycine, glycolate, hydroxyproline, and vitamin C. Due to variable and often low bioavailability, much of the oxalate in food may not be readily absorbed. The dietary contribution of urinary oxalate may be higher in stone formers. Up to one-third of patients with calcium oxalate nephrolithiasis may have increased absorption of dietary oxalate, and in some cases a deficiency of oxalate degradation by the bacterium Oxalobacter formigenes in the gut could be the culprit (Holmes and Kennedy, 2000).The impact of dietary oxalate on risk of stone formation has not yet been studied prospectively because of the lack of sufficient and reliable information on the oxalate content of many foods. However, recent reports using modern approaches to measure the oxalate content of food (Siener*et al*., 2006***;*** Holmes and Kennedy, 2000).Have opened the possibility of these studies being completed in the near future.

2.1.7.9.3Magnesium complexes with oxalate

Thereby potentially reducing oxalate absorption in the gastrointestinal tract and decreasing calcium oxalate supersaturation in the urine. A few randomized trials have examined the effect of magnesium supplementation on stone recurrence. However, magnesium was given in combination with other compounds (e.g., thiazide diuretic or potassium citrate) and the dropout rates were high. Currently, it is uncertain whether magnesium supplementation has an independent beneficial effect. In prospective observational studies, higher dietary magnesium was associated with a 30% lower risk of stone formation in men (Taylor*et al*., 2004).But not in women (Curhan *et al.*,1997 and 2004).

2.1.7.9.4 Vitamin C (ascorbic acid)

Vitamin C can be metabolized to oxalate; thus, higher vitamin C intake could increase the risk of calcium oxalate stone formation. A metabolic trial demonstrated

that the consumption of 1000 mg of supplemental vitamin C twice daily increased urinary oxalate excretion by 22% (Traxer *et al*., 2003). An observational study in men found that those who consumed 1000 mg or more per day of vitamin C had a 40% higher risk of stone formation compared to men who consumed less than 90 mg/day (the recommended dietary allowance) (Taylor*et al*., 2004). This relation was observed only after accounting for dietary potassium intake. Although restricting *dietary* vitamin C does not seem appropriate (as foods high in vitamin C are also high in inhibitory factors such as potassium), a calcium oxalate stone former should be encouraged to avoid vitamin C supplements.

2.1.7.9.5 Vitamin B6

Vitamin B6 is a cofactor in oxalate metabolism, and vitamin B6 deficiency increases oxalate production and urine oxalate excretion. Although high doses of supplemental vitamin B6 may be beneficial role in selected patients with type 1 primary hyperoxaluria, the use of vitamin B6 in other settings remains unclear. Based on observational data, high intake of vitamin B6 may reduce the risk of kidney stone formation in women (Curhan *et al.,*1999). Butnot in men (Curhan *et al.,*1996).

2.1.8 Metabolic Causes for Stone Formation

The 24-hour urine chemistries provide important prognostic information and direct therapeutic recommendations for prevention. Traditionally, urine results have been categorized into 'normal' and 'abnormal', but recently there has been a greater appreciation of two important points. First, the urine values are continuous so the dichotomization into 'normal' and 'abnormal' is arbitrary and potentially misleading. Second, stone formation is a disorder of *concentration*, not just the absolute amount excreted. Although terms such as 'hypercalciuria' are often used both clinically and scientifically, the limitations of these terms should be remembered.

2.1.8.1 Hypercalciuria

Hypercalciuriahas been traditionally defined as urine calcium excretion ≥ 300 mg/day in men and ≥ 250 mg/day in women (Hodgkinson and Pyrah, 1958).On a 1000-mg/day calcium diet. Based on these definitions, ~20 to 40% of patients with calcium stone disease will have hypercalciuria. Although a higher cutoff value in males makes sense from a calcium balance perspective, it does not for stone formation, particularly given that 24-hour urine volumes are slightly higher in women (Curhan *et al*., 2001).

Hypercalciuria is the most common metabolic abnormality in patients with calcareous stones and results from various mechanisms. *Absorptive hsypercalciuria*— increased absorption of calcium from the gut results in increased circulating calcium, resulting in increased renal filtered load. The exact mechanism is unknown but seems to be inherited in an autosomal dominant fashion, and the jejunal mucosa is hyper-responsive to vitamin D. Absorptive hypercalciuria is very common, but most patients remain asymptomatic and do not experience stone formation.Anatomical abnormalities that increase the risk of stone disease are: obstruction of the pelviureteral junction, hydronephrotic renal pelvis or calices, calyceal diverticulum, horseshoe kidney, ureterocele, vesicoureteral reflux, ureteral stricture and tubular ectasia (medullary sponge kidney). *Renal hypercalciuria*— increased excretion of calcium in urine results from impaired renal tubular absorption of calcium. This occurs in about 2% of patients with recurrent stone formation. *Resorptive hypercalciuria*— increased resorption of bone occurs as a result of primary hyperparathyroidism. This occurs in about 5% of patients with recurrent stone formation. The risk of renal stones is increased in primary hyperparathyroidism and returns to baseline about 10 years after parathyroidectomy Patients who had stones before undergoing parathyroidectomy have a 27 times greater risk of stone formation after parathyroidectomy than do patients without hyperparathyroidism (Taylor*et al*., 2004).

2.1.8.2 Hyperuricosuria

The relation between uric acid metabolism and calcium stone disease has been intriguing. Uric acid is the end product of purine metabolism and is either derived from exogenous (dietary) sources or produced endogenously during cell turnover. Chronic metabolic acidosis can result in protein metabolism and thus increased excretion of urate and formation of kidney stones. Pure uric acid stones are rare but recur frequently. Low urinary pH (pH < 5.5) is the most common and important factor in uric acid nephrolithiasis; in normouricosuric stone disease the primary defect seems to be in the renal excretion of ammonia and is linked to an insulin resistant state. Hyperuricosuria occurs in 10% of patients with calcium stones, where uric acid crystals form the nidus for deposition of calcium and oxalate. A history of gout doubles the risk of kidney stones in men (Kramer *et al.*, 2003).

2.1.8.3 Hyperoxaluria

Hyperoxaluria is defined as urinary oxalate excretion >45 mg/d. Elevated urinary oxalate excretion may be present in up to 40% of male stone formers and in up to 10% in female stone formers. Although mean urinary oxalate levels may not differ between cases and controls, oxalate does appear to be an important independent risk factor for stone formation (Curhan *et al*., 2001).

On the basis of the mechanism, it is classified as follows: metabolic risk factors for calcareous stones (hypercalciuria (40-60%), hyperuricosuria (25%), hyperoxaluria, hypocitriuria and other (vitamin A deficiency, hot climates, immobilisation, urinary tract anomalies). *Enteric hyperoxaluria*— this results from increased intestinal absorption due to ileal disease (Crohn's disease, ileal bypass) or short bowel syndrome, low calcium intake, or gastrointestinal decolonisation of

Oxalobacter formigenes. Oxalobacter is an intestinal bacterium that degrades dietary oxalate, and decolonization of the gut results in increased absorption of oxalate. Oral administration of *Oxalobacter* has been shown to decrease urinary oxalate concentration in animals and humans. Increased ingestion (oxalate gluttons): Dietary oxalate contributes to about half of the urinary oxalate and is inversely proportional to calcium intake in healthy people without gastrointestinal disease (Holmes and Assimos, 2001). Spinach, rhubarb, beets, chocolate, nuts, tea, wheat bran, strawberries, and soya foods are known to increase urinary oxalate concentrations. Vitamin C supplementation may increase urinary oxalate excretion and the risk of calcium oxalate crystallization in patients who form calcium stones. Ingestion of grapefruit juice increases excretion of both oxalate and citrate in urine with no net change in its lithogenicity. *Primary hyperoxaluria*— this is an inborn error of metabolism (glycolic aciduria). In experimental animals, testosterone promotes stone formation by suppressing osteopontin expression in the kidney and increasing urinary oxalate excretion. Oestrogen seems to inhibit stone formation by increasing osteopontin expression in the kidney and decreasing urinary oxalate excretion (Taylor*et al*., 2004). Drugs that may increase the risk of stone disease are decongestants: ephedrine, guaifenesin[w1-2], diuretics: triamterene, protease inhibitors: indinavir[w3], anticonvulsants: felbamate,[w4] topiramate, and zonisamide[w5]. The non-dissolving carrier of osmotically controlled release oral (OROS) drugs may be misdiagnosed as kidney stones on *x* ray[w6].

2.1.8.4 Hypocitriuria

Hypocitraturia, typically defined as 24 hour excretion ≤ 320 mg/d, increases risk for stone formation (Pak, 1994). And is found in 5–11% of first time stone formers (Curhan *et al*.,2001). At present, there is insufficient evidence to conclude increasing urinary citrate into the high-normal range provides additional protection. It is a common correctable cause of recurrent pure calcium phosphate or brushite stones. Women excrete more citrate and have lower incidence of stone formation than men. Urinary citrate is mainly derived endogenously through the tricarboxylic acid cycle and is excreted by renal tubular cells. Intracellular acidosis, acidic diets (diets rich in animal proteins), and hypokalaemia decrease urinary citrate excretion. Fruits such as oranges and grapefruits are the main exogenous sources of urinary citrate. Hormonal replacement therapy in postmenopausal women results in higher urinary calcium excretion, but it also increases urinary excretion of citrate and leads to net inhibition of crystal precipitation, thereby decreasing the risk of calcium stones. Low urine volume, for which a variety of definitions have been used, is a common and modifiable risk factor. When defined as 24 hour urine volume less than one liter per day, 12–25% of first time stone formers will have this abnormality. Observational studies have demonstrated the risk of stone formation decreases with increasing total urine volume (Curhan *et al*., 2001). And a randomized trial confirmed the value of increasing urine volume (Borghi*et al.*,1996).

2.1.9 Struvite stones

Struvite, a crystalline substance composed of magnesium ammonium phosphate, was first identified by Ulex, a Swedish geologist, in the 18th century. He created the term ‘struvite’ in honor of his friend and mentor H.C.G von Struve (1772-1851), a Russian diplomat and natural scientist. Brown suggested that bacteria split urine and thus caused and facilitated stone formation. He also isolated Proteus vulgar is from a stone nucleus and this is known today to secrete urease. However it was Hager and Magath, in 1925, who suggested that ‘urease’ was the cause of hydrolysis of urine. Struvite stones account for 5-20 % of all urinary stones. Data from the 1970s indicated that struvite stones constituted 15% of the all stone specimens sent for calculus analysis but Rodman (1998) suggested that this figure over-reported the incidence of infection stones, since many small spontaneously passed calculi are never caught and do not have their chemical composition determined. 15-20 % of urinary stones were infection stones in industrial countries and their clinic results showed that out of 4,400 patients with urinary stones, 510 had infection stones (11.6 %). Struvite stones are also referred to as ‘infection stones’ and ‘triple phosphate stones’. The term triple phosphate is derived from the fact that early chemical analyses demonstrated calcium, magnesium, ammonium and phosphate (i.e. three cations and one anion). More recently struvite stones have been shown to be a mixture of struvite and carbonate apatite (Curhan *et al*., 2001).

2.2 Minerals in foods

The term "minerals" may conjure up thoughts of rocks. But to the body minerals are another grout of essential nutrients, needed to both regulate body processes and give the body structure. Not only to bones and teeth, but muscle, blood and other body tissue all contain minerals too. Minerals can't be destroyed by hest or other food handling processes. In fact, if we have completely burned food, perhaps while cooking over afire the little bit of ash left over it is mineral content. Minerals have two categories, major minerals and trace minerals depending on how much the body's need. Regardless of amount, they are all essential (Taylor*et al*., 2004).

2.2.1 Major minerals

Major minerals are needed in greater amounts more than 250 milligrams recommended daily. Calcium, phosphorus, and magnesium fit in this category, along with three electrolytes (sodium, chloride and potassium) (Taylor*et al*., 2004).

2.2.1.1 Potassium

Potassium is a mineral found in many foods. One of its main jobs is to send messages to your muscles so they will work properly. When potassium in the blood is too high it can cause muscle weakness, breathing problems and it can change the heart beat enough to cause serious damage or even death. If potassium is too low, it can

cause muscle weakness, irregular heartbeat, low blood pressure and confusion. When kidneys work well they control potassium you eat. Depending on your blood level of potassium you may be able to eat 2000-4000 mgs of potassium daily. Whether you need a low potassium diet or a high potassium diet the following list should help you make the best choices (Taylor*et al*., 2004).

2.2.1.2 Sodium

Sodium is a mineral element that the body needs to function properly. It is involved in transmitting nerve impulses and in maintaining blood volume and cellular osmotic pressure. The Dietary Reference Intakes for Sodium (Institute of Medicine, National Academies of Science, 2005) established a Tolerable Upper Intake Level (UL), which is the highest daily nutrient intake level that is likely to pose no risk of adverse health effects. As the UL increases, so does the risks of adverse effects. For sodium, the adult UL is 2,300 milligrams (mg). Intakes above this level are associated with increased blood pressure. It is estimated that most people take in an average of 2,300 to 6,900 milligrams (about 1 to 3 teaspoons or 6 to 17 grams of salt) per day. Most dietary sodium is found in the form of sodium chloride, the compound we know as table salt, which is 40 percent sodium and 60 percent chloride. One teaspoon of salt contains about 2,000 milligrams of sodium. A healthy eating pattern limits intake of sodium, solid fats, added sugars, and refined grains and emphasizes nutrient-dense foods and beverages—vegetables, fruits, whole grains, seafood, lean meats, poultry, eggs, beans, peas, nuts, seeds, and fat-free or low-fat milk and milk products (Taylor*et al*., 2004).

2.2.2 Trace minerals

The body needs just small amount, less than 20 milligrams daily of the trace minerals, or trace elements, such as chromium, copper, fluoride, iodine, iron, manganese, molybelanum, selenium, and zinc. Recommended dietary allowances have only been set for a few of them iron, zinc, iodine and selenium, until science learns more, other are presented as arrange of estimated safe and adequate daily intakes (Taylor*et al*., 2004).

2.3 Water

Water is one of the most important chemicals known to man. Without it neither animals nor plants life will exist. Water is essential in processes of digestion, circulation, elimination and the regulation of body temperature. Water is used as a solvent for many substances (Georgel and Schultz,1973). Almost three fourth of the earth's surface is covered by water. Most of this water is not suitable for human use. The demand for fresh water has increased with rapid growth of population, agriculture, and industry (Abdelgafar, 1999).

2.3.1 Water sources

The sources of water supply are divided into two major class, surface and ground water.

2.3.1.1 Surface water

There are many sources of surface water. This includes rivers, streams, lakes and reservoirs. The common characteristics of surface water are that it contains few minerals, t is not very hard. It is usually large in volume and it is easily contaminated, total bacterial contents are high. Therefore, proper treatment is required before human consumption (WHO, 1993).

2.3.1.2 Ground water

Ground water is supplied from rivers, reservoirs and marshes. Ground water comes into contact with various minerals which are soluble in water. The dissolved minerals beyond certain limits may make it unsuitable for irrigation, drinking or industrial purposes.

In many countries ground water is the main source of water for all purposes. This is because rural communities are found close to the ground water resources (WHO, 1993). Ground water can be classified into three classes according to the layer in which it is found (IDC, 1981).

2.3.1.2.1 Ground water

It is that collects above the stable impervious layer of rock. It can move freely and its surface is known as ground water surface. It is supplied mainly by rain water or percolation o rivers or lakes water (WHO, 1993).

2.3.1.2.2 Artesian water

Artesian water is stored in a water containing layer which is sandwiched between and confined by two impervious layers.

2.3.1.2.3 Perched water

Perched waterexists above asectional impervious layer. It is not widely distributed and is seasonal (El Hussien, 2000).

2.3.2 Type of water

2.3.2.1 Distilled water

Distillation uses a boiling process to remove impurities such as bacteria, viruses and pollutants. Although the final result mostly free from harmful elements.

2.3.2.2 Purified water

Water labeled as "purified" usually comes from municipal or city water supply plain tap water. To purify this water, methods such as carbon filters, reverse osmosis, distillation, or demonizing.

2.3.2.3 Tap water

Tap water is non-filtered, with chlorine added to disinfect. Sources are from ground or surface water. Tap water can contain fertilizer, herbicide, insecticide pollutants and pharmaceuticals.

2.3.2.4 Natural spring water

Ground water obtained from springs is similar to water pumped from shallow wells. Spring may be contamination by surface water or other sources on or

below the ground surface. Spring water is susceptible to contamination because the water feeding the spring typically flows through the ground for only a short distance, limiting the amount of natural filtering that can occur.

2.3.4 General properties of water
Are classified into physical and chemical properties

2.3.4.1 Physical properties of water
Physical properties include the following:

2.3.4.1.1 Color
Color is caused by materials in solution or colloidal condition and should be distinguished from which, may cause an apparent may color (Twort *et al*., 1985).

2.3.4.1.2 Turbidity
Water is turbid when it contains visible materials in suspension while turbidity may result from living or dead a large or other organism. Generally turbidity by silts or clay the amount and character of the turbidity will depend upon the type of soils over which the water has run and the velocity of the water (Twort *et al*., 1985).

2.3.4.1.3 Temperature
In general the rate of chemical reaction decrease with decreasing temperature the relative concentrations of reaction and products in chemicals equilibrium can also change with temperature therefore; temperature affects every aspect of the treatment and delivery of potable water. Turbidity and color are indirectly related to temperature, as the efficiency of coagulation is strongly temperature dependant (Twort *et al*., 1985).

2.3.4.1.4 Density
The density of pure water 1g/ml at 4c density decreases with the increase of the temperature unit it reaches the dissociation state (breaking of hydrogen bonds) at the boiling point (Twort *et al*., 1985).

2.3.4.1.5 Surface tension
It's is a property of the surface of a liquid that allows it to resist an external force. Is revealed, for example, in floating of some objects on the surface of water, even though they are denser than water, this property is caused by cohesion of like molecules, and is responsible for many of the behaviors of liquids. Surface tension is responsible for the shape of liquid droplets although easily deformed, droplets of water tend to be pulled into a spherical shape by cohesive forces of the surface layer in the absence of other forces, including gravity, drops of virtually all liquids would be perfectly spherical (Twort *et al*., 1985).

2.3.4.1.6 Water ionization
The degree of ionization of water is weak but increases with the temperature pure water is not considered as electrical conductor although the degree of ionization of water is very poor; but is plays a great role in most chemical reaction(Twort *et al*., 1985).

2.3.4.1.7Electrical conductivity (EC)
Electrical conductivity of a substance is its ability to conduct electrical current and specific electrical conductance defined as the conductance of cubic centimeter of substance compared with the same volume of water (Abdalla, 2001). Pure water has very low electrical conductance. Conductivity of water will increase with the presence of dissolved minerals (Fletcher, 1989). Water with high specific conductance can cause corrosion of iron and steel. Electrical Conductivity is expressed in micro mohs centimeter^{-1} (u mohs/cm) and is directly proportional to the amount of dissolved solid in water. So that it is an excellent indicator of TDS in water (Twort *et al*., 1985).

2.3.4.1.8Total dissolved solids (TDS)
Total dissolved solids in water comprise inorganic salt and small amounts of organic matter that is dissolved in water. (TDS) is expressed in ppm and it is directly related to the conductivity. lt has important effect on the taste of drinking water. Limit of 600 mg/L for Domestic use if based on the taste, so that it is an indicator to study the salinity property of ground water. Water with high TDS concentration is saline water (Table 2.1).Water with extremely low concentration of TDS may be unacceptable because of its fullness (WHO, 1993).

2.3.4.2 Chemical properties of water
Chemical parameters enable us to classify the water, find the degree of pollution and causes of sharp increase of pollution substances. Chemical properties include:

2.3.4.2.1 PH
The pH is negative common logarithm of hydrogen ion activity in moles per liter (WHO, 1984). The pH of the most natural water falls within the range 4 to 9 if the pH value is very low (less than 4) water has a sour or acidic taste. High PH can give taste problems and soapy feel. The majority of water is slightly basic due to presence of carbonate (El Hussien, 2000). Temperature plays a role in the determination of pH at which neutrality occurs (Fletcher, 1989).

2.3.4.2.2 Alkalinity
An alkaline water is one with a pH value more than 7.0, the causes of alkalinity is the presence of carbonates, bicarbonates and hydroxides of calcium, magnesium, potassium and sodium. Among these the calcium bicarbonate is the most abundant substance causing alkaline. The presence of calcium and magnesium is called a temporary hardness. When the alkalinity and hardness are equal, all the hardness is temporary if the total hardness is greater than alkalinity there is permanent hardness, sometimes it happens that, the total hardness indicates the presence of potassium and sodium salts which added to the alkalinity but do not increase the

hardness. The alkalinity of many water is in the range (100 – 200 ppm) (WHO, 1963).

Table 2.3: Health criteria for TDS.

Range (mg/l) TDS	TDS level
Less than 300	Excellent
300 – 600	Good
600 – 900	Accepted
900 – 1200	Bad
Over 1200	Unacceptable

Source:(WHO, 1989).

2.3.4.2.3Sulphate

The concentration of uslphate in natural water can be found in various ranges from a few mg/l to several thousand mg/l. the highest level usually occur in ground water. The sources of sulphate are the solutions of minerals containing sulphates and oxidation of sulphur to sulphates, and thio-sulphates.The presence of sulphate in drinking water causes noticeable taste. The taste depends on associated cation. Taste threshold has been found to range from 250 mg/l for sodium sulphates to 1000 mg/l for calcium sulphate (WHO, 1993).

2.3.4.2.4 Chloride

Chloride is one of the major anions in water. Chlorides are present as sodium Chloride (NaCl, common salt) and to a lesser extent as calcium and magnesium chlorides (Abdelgafar, 1999). The main problem caused by excessive chloride in water concerns the acceptability of the supply. Concentration above 250 mg/l can impart a distinctly salty taste to water (Twort, 1985). For people suffering from heart and kidney diseases, high chloride water usage has to be restricted (WHO, 1993). Excessive chloride Concentration increases rates of corrosion of metals in the distribution system.

2.3.4.2.5 Carbonate and bicarbonate

Carbonate and bicarbonate are two of the three forms of the components of the carbonates equilibrium. The variation of pH of water affects the concentration of carbonate ions, if the pH is less than 4.5, water is free from carbonic acid, when pH is above 8.3 then carbonic acid content is disregarded. High concentration of carbonic acid in water makes the water corrosive to metals concrete (Abdalla, 2001).

2.3.4.2.6 Calcium (Ca^{2+})

Calcium is necessary in plant and animal and is essential component of bones, shells, and plant structures. The presence of calcium in water supplies results from passage over deposits of limestone, dolomite gypsum; and pypsiferous shale. Small concentrations of calcium carbonate combat corrosions of metal pipes by laying down a protective coating. The amount of calcium in domestic waters and industrial is often controlled by water softening (e.g. ion exchange) (APHA, 1971).

Calcium contributes to the total hardness of water. Chemical softening treatment reverse osmosis, electro dialysis or ion exchange are used to reduce calcium and the associated of water (Chapman, 1996).

2.3.4.2.7 Sodium

The sodium ion is a major constituent of natural water. it has been estimated that food accounts for approximately 90% of the daily intake of Na^+ where drinking water contributes the remaining 10% (Elnour,2004).

2.3.4.2.8 Potassium

Potassium as k^+ is usually found in low concentration in natural water. Since rocks which contain potassium are relatively resistant to weathering. K^+ concentration in natural water is usually less than 10 mg/l. where concentration as high as 1000 mg/l, 2500 mg/l can occur in hot springs and brines respectively (Chapman, 1996).

2.3.4.2.9 Hardness

Hardness in water is caused by metallic ions which are capable of precipitating soap. The hardness of water is due to the presence of bicarbonate, sulphate and chloride of calcium and magnesium. There are two types of hardness temporary hardness which is due to the presence of bicarbonates. This type is removed by boiling the water and precipitation of $CaCo_3$and $MgCo_3$. Permanent hardness which is due to the presence of chloride and sulphate this hardness cannot be eliminated by boiling of water (Abdelgafar, 1999). The degree of hardness in water (Table 2.2) may affect it's acceptability to the consumers in terms of taste. The taste threshold for the calcium ion is in the range 100 to 300 mg/l, depending on the associated anion. The magnesium taste threshold is less than that for calcium. Water hardness in excess of 500 mg/l is not tolerated by consumers.Water with hardness above approximately 200 mg/l may cause scale deposition in the distributive system. Also large amount of soap are required to produce lather in hard water. on the other hand soft water with a hardness less than 100 mg/l may affect human health(Twort *et al*., 1985).

2.3.4.2.10 Water quality standard

International standards for drinking water were first published by WHO in 1989, then have been revised and reissued in a new form in 1993 and entitled as (Guideless for drinking water quality (Table 2.3).

Table 2.4: Classification of water on the basis of hardness.

Range (mg/l) $CaCo_3$	Hardness level
0 – 50	Soft
50 – 100	Moderately soft
100 – 150	Slightly hard
150 – 200	Moderately hard
200 – 250	Hard
250 – 300	Very hard

Source (Twort *et al*., 1985

Table 2.5: WHO maximum permissible values for drinking water.

Characteristic	Units
Hardness	500 mg/l as $CaCo_3$
PH	6.5 – 8.5
Potassium	10 mg/l
Sodium	200 mg/l
Calcium	200 mg/l
Magnesium	150 mg/l
TDS	1000 mg/l
Sulphate	400 mg/l
Chloride	250 mg/l

Source: WHO(1993)

3. MATERIALS AND METHODS

3.1 Study design
This is a cross-sectional descriptive, hospital based study aiming to correlate the chemical composition of kidney stones with the diet style of some patients.

3.2 Study area
The study was conducted in Gezira Hospital for Renal Disease and Surgery (GHRDS). GHRDS is the only specialized hospital outside Khartoum (serving a large population from Gezira State and other States), and University of Gezira, Department of Food Science and Technology in the period from January-November 2015.

3.3 Study population
3.3.1 Stones samples
Each patient was subjected to full clinical history and complete examination. The appropriate investigations necessary for diagnosis were performed (i.e. urine analysis, ultra-sound, IVU, and CT). The proper management of patients was done according to the European Association of Urology (EAU) Guidelines (Block *et al.*, 2009). These stones were either removed surgically by extracorporeal shock wave lithotripsy (ESWL) or using endoscopy and some of these stones were passed spontaneously. The stones were transferred to Gezira Central Laboratory for chemical analysis by semi-quantitative method as was recommended by Beeler *et al.*, (1964). Kits were purchased from Mascia Brunell S.P.A (Italy) and were used for chemical analysis of stones as described before.

3.3.1.1 Inclusion criteria
All patient withrenal stones.

3.3.2 Foods samples
Peanut, Lentil, Wheat, Egg plant, Okra, Sweet potato, Tomato, Banana, Lemon, Dates, Milk, Cheese, Yoghurt, Egg, Fish, Chicken, Tea, Coffee. All the samples were obtained from Wad Medani local market, Central Sudan. All samples were collected in the plastic sack and transported to the food Laboratory, University of Gezira Department of Food Science and Technology.

3.3.3 Water samples
The sample of drinking water were taken from three locations in Wed Medani City in plastic container (500 ml) for chemical analyses and stored at temperature.

3.4 Samples Size
3.4.1 Stones samples
Involved 48 consecutive Sudanese patients (32 men and 16 women). All of them with kidney stones diseases.

3.4.2 Foods samples
Involved 18 foods samples

3.4.3 Water samples
The nine samples were collected from each location, tap water, zeer water and well.

3.5 Data collection tools
Structured questionnaire and direct interviews.The data taken included gender, age, Residency, height, weight, history of gout, diabetes, chemotherapy, urinary obstruction, and hyperparathyroidism. Collect information regarding the detailed dietary history with added information about his/her food likes/dislikes, preferences/intolerances (if any), eating habits, general meal pattern and dietary intake. Added information about dietary modifications (if any) in relation to the kidney stone conditions such as foods especially taken or avoided, consumption of salt and amount of water consumed were also collected. Information on food preferences of the patients in terms of frequency of consumption was collected. In addition, detailed list of foodstuffs selected with special reference to their mineral content (calcium, phosphorus magnesium, oxalates), stone forming constituents and foodstuffs helping in reducing the risk factors of stones was collected from the patients. Questions used to collect information for the present study are given in Appendix.

3.6 Sample preparation
3.6.1 Stones samples
The stones were washed in distilled water grinded. The stones were first examined for physical characteristics. Since the most stones are mixtures and may consist of several layers, they were cut into two halves and were powdered for analysis of chemical compositions.

3.6.1.1 Test for Carbonate
By treat some powder in test tube with cold molar nitric acid boil then cool, positive result appear as air bubbles.

3.6.1.2 Test for calcium
By adding few drops of ammonium oxalate solution, adjust alkaline pH of solution by addition of ammonia solution follow by adding acetic acid until being acid again, positive result appear as white precipitate.

3.6.1.3 Test for phosphate
By adding ammonium molybdate then adding powder of reducing agent, positive result appears as blue color.

3.6.1.4 Test for oxalate
By adding few of calcium chloride, adjust pH, results appear as white precipitate.

3.6.1.5 Test for uric acid
By adding few sodium carbonates to few powders then add phosphotungestic acid, positive result appear as blue color.

3.6.1.6 Test for Cystine
Kidney stone been a little powder placed in a white tile and then added to drop of sodium cyanide solution and then a drop of fresh sodium solutionNatrobroseid result appear as magenta color.

3.7 Foods samples
3.7.1Proximate analysis of foods sample
Analysis of proximate composition was carried out on the sample consisting of foods powder. The proximate composition include ash.

3.7.1.1Ash determination
The method of AOAC (1984) was used to determine the ash content of the samples analyzed in this study; 5 grams of sample were weighed into a clean pre-dried and weighed porcelain dish. The dish containing the sample was placed in a muffle furnace at 550^0c and left burning for 5 hours at this temperature. Then, the dish with its content was weighed again after cooling in dessicator to the room temperature.

Ash contents were calculated as follows:

$$\textbf{Ash\%} = \frac{\text{Weight of ash} \times 100}{\text{Weight of sample}}$$

3.7.2 Determination of minerals
Potassium (K), sodium (Na) and calcium (Ca) determination were accomplished by means of flame photometer model (Corning 400) according to the AOAC (1984) in which different concentration (5,10,15,20,25ppm) were prepared from stock solutions of Ca, Na and K using the flame photometer the readings were taken and a graph was made.

❖ **Reagent**
- **Sodium stock solution**

Weight of 2.5 g of Na CL were dissolved in distilled H_2O and diluted to one liter "1000ppm Na/ml" then 10 ml of the solution were taken and diluted to 100ppm Na/ml.

- **Potassium stock solution**

Weight of 1.91 g of KCL were dissolved in distilled H_2O and diluted to one liter "1000ppm Na/ml" then 10 ml of the solution were taken and diluted to100ml with H_2O to make 100ppm K/ml.

- **Calcium stock solution**

Weight of 1.77 g of $CaCL_2$ were dissolved in distilled H_2O and diluted to one liter "1000ppm Na/ml" then 10 ml of the solution were taken and diluted to100 ml with H_2O to make 100ppm Ca/ml.

Aliquot containing 10,20,30 and 40ppm for Na, 4,8,12,16 and 20ppm for K and 100, 200, 300, 400 and 500ppm for Ca were taken for contracting the standard curve for each element.

❖ **Procedure**
The sample was prepared by weighing 5 g of sample were weighed into a clean pre-dried and weighed porcelain dish. The dish containing the sample was placed in a muffle furnace at 550^0C and left burning for 5 hours at this temperature. Then, the dish with its content was weighed again after cooling in desecrator to the room temperature and ash content was determined then ash was dissolved in distilled water and adding 10 ml HCL to make 100 ml. Then the absorption of the sample was measured and the concentration determined from the calibration, curve.

3.8 Water samples
3.8.1 Determination of Total Dissolved Solids (TDS)
Total dissolved solids were determined by evaporating a known weight of the water sample to dryness.

❖ **Procedure**
25ml of water samples were transferred to a weighted evaporating dish and evaporated to dryness by heating at 100 ^{0}C in an oven to constant weight.

❖ **Calculation**

$$\textbf{Mg/L of TDS} = \frac{\text{Mg residue} \times 100}{\text{Volume of sample}}$$

3.8.2 Measurement of Electrical conductivity (EC)
❖ **Reagents**
- Potassium chloride 0.01 M
- Distilled water

0.0745 mg of AR potassium chloride was dissolved and diluted to 100 ml distilled water to make standard reference solution.

❖ **Procedure**
- EC was measured by electrical conductivity meter

A measuring set was used with specific conductance cell and it was calibrated with (0.01m) KCL. The EC of the sample were then determined in Mmoh's/cm.

3.8.3 Determination of pH value
❖ **Reagent**
Standard buffer solution of pH (3.7,10) were used. The tablets of pH (3.7 and 10) were dissolved in 100 ml distilled water to make the buffer solution specified.

3.8.4 Determination of Hardness

EDTA titrimetric was used as described in standard methods.

❖ **Reagents**

○ **Ammonia buffer solution (pH=10)**

About 16.9 g ammonium chloride were added to 143 ml concentrated ammonium hydroxide, and diluted to 250 ml with distilled water.

○ **Standard EDTA Titrant (0.01 m)**

About 3.723 g of graded Na_2 EDTA ($Na_2H_2C_{10}H_{14}O_8N_2.2H_2O$) were dissolved in distilled water and diluted to 1000 ml.

○ **Eriochrome black T indicator**

About 1 g Eriochrome black T was added to 10 g potassium nitrate and mixed together.

❖ **Procedure**

One ml ammonium buffer solution and about 30 mg of eriochrome black T indicator were added to 50 ml water sample in a 250r ml conical flask, and then the solution was titrated with the 0.01m EDTA solution until the color changed from wine red to blue end point.

❖ **Calculation**

$$\textbf{Hardness as mg } CaCo_3\textbf{/L} = \frac{V \times 0.01 \times 100 \times 1000}{50 \text{ ml of sample}}$$

V= Volume of EDTA required for titration.

3.8.5 Determination of Chloride (CL^-)

❖ **Reagents**

○ **Potassium chromate indicator 5%**

Five mg of K_2CrO_4 was dissolved in distilled water, few drops of standard $AgNO_3$ were added till red precipitate was formed; the solution was filtered and diluted to 1000 ml with distilled water.

○ **Silver nitrate titrant (0.02 M)**

About 3.4 mg $AgNO_3$ was dissolved in distilled water and diluted to 1000 ml, the solution was then stored in dark bottle.

❖ **Procedure**

About 50 ml of water sample were placed in 250 ml conical flask, 2 ml potassium chromate indicator were added, then the solution was titrated with 0.02 M $AgNO_3$ solution to pinkish yellow end point. Distilled water being used as a blank, was treated in the same mentioned above (Vogel's, 1978).

❖ **Calculation**

$$\textbf{Mg } CL^-\textbf{/L} = \frac{(A-B) \times M \times 35.45 \times 1000}{\text{Ml of sample}}$$

Where:

A=ml of $AgNO_3$ titrated with 50 ml sample

B=ml of $AgNO_3$ titrated with blank

M=Molarities of silver nitrate titrant

3.8.6 Determination of Sulphate

Sulphate (SO_4^{-2}) was determined as $BaSO_4$

❖ **Reagents**

○ **Hydrochloric acid HCL (1:1)**

10 mg of concentrated HCL were diluted with 10 ml distilled water.

○ **Barium chloride solution (10%)**

10 mg of AR $BaCL_2.2H_2O$ was dissolved and diluted to 100 ml with distilled water.

❖ **Procedure**

50 ml of water sample were placed in a 400 ml beaker. The pH was adjusted with HCL (1:1) to pH (4 - 5) by using a pH- meter. The solution was then heated to boiling. Warm barium chloride ($BaCL_2$) solution was then added while stirring until complete precipitation. The solution was overnight, filtered through filter paper the residue was cooled in a disicatorand weighted.

❖ **Calculation**

$$\textbf{Mg of } SO_4^{-2}\textbf{/L} = \frac{\text{Mg of } BaSO_4 \times 0.412 \times 1000}{\text{Ml of sample}}$$

$$\textbf{0.412 = a gravimetric factor} = \frac{\text{Molecular weight of } SO_4^{-2}}{\text{Molecular weight of } BaSO_4}$$

3.8.7 Determination of Carbonate and Bicarbonate

❖ **Reagents**

○ **Hydrochloric acid0.05 M**

About 4.5 ml of concentrated HCL was diluted to 1000 ml with distilled water.

○ **Phenolphthalein indicator solution**

About 0.5 mg phenolphthalein was dissolved in a 50 ml 95% ethanol and diluted to 100 ml with distilled water.

○ **Methyl orange indicator solution**

About 0.5 mg methyl orange powder was dissolved in dissolved in distilled water and diluted to 100 ml.

❖ **Procedure**

About 50 ml of water sample were placed in 250 conical flasks few drops of phenolphthalein solution were added, and then the solution was titrated with 0.05 M HCL. Until the color changed from purple to color lees the volume of acid (say x) was recorded. Then few drop of methyl orange indicator were added and the titration with HCL continued until the color changes from yellow to red, the volume acid (say y) was recorded.

❖ **Calculation**

X = Volume of acid = 1/2 carbonate

○ **Therefore**

2X= volume of acid =all carbonate in 50 ml sample

Y= volume of acid =all carbonate + all bicarbonate

The result were expressed as mg/L

$$\textbf{Mg of } CO_3^{-2}\textbf{/L} = \frac{0.05\times (2\times) \times \text{M.WT} \times 1000}{\text{Ml of sample} \times 2}$$

(Y-2X) =Volume of acid required to titrate all bicarbonate in 50 ml sample.
M.WT = molecular weight of HCO_3^{-2}

3.8.8 Determination of Calcium (Ca^{2+})

❖ **Reagents**

○ **Sodium hydroxide (I m)**

About 4 gm of A.R Na OH was dissolved in distilled water and diluted to 100 ml.

○ **Murexide (Ammonium purpurate) indicator (0.05gm).**

○ **standard EDTA(0.01)**

Na_2EDTA disodium EDTA $Na_2H_2C_{10}O_8N_2 2H_2O$as in (2.6).

❖ **Procedure**

About 50 ml of water sample were placed in 250 ml conical flask, 1ml of Na OH solution was added to get a pH of (12-13) and the solution was stirred; (0.05 mg) of Murexide indicator was added while stirring, the solution was then titrated with (0.01m) EDTA solution, until the color change from purple to blue (Vogel's, 1978).

❖ **Calculation**

$$\textbf{Mg } Ca^{2+} = \frac{M\times V\times A.Wt\times 1000}{\text{Ml of sample}}$$

Where
M= molarity of EDTA required for titration
V= volume of EDTA required for titration
A.Wt =Atomic weight of calcium

3.8.9 Determination of Sodium and Potassium

Sodium and potassium were determined by flame photometry.

❖ **Reagents**

○ **Stock sodium solution**

About 2.542 g of dried Na CL were dissolved in distilled water and diluted to 1000 ml, to give 1000 ppm Na^+.

○ **Standard sodium solutions**

Stock solution was diluted with distilled water to give standard sodium solutions ranging from (20 to 100ppm).

○ **Stock potassium solutions**

About 1.907 g of A.R KCL dissolved in distilled water diluted to 1000 ml, to give 1000 ppm m K^+.

○ **Standard potassium solutions**

About 20 ml from the stock solution was diluted with distilled water in 100 ml flask to give 200 ppm potassium solution take 2.4.6.8 and 10 from the above solution in 100 ml flask and the volume were complete to 100 ml to give 4, 8,12,16 and 20 ppm potassium.

❖ **Procedure**

Flame photometry the calibration cure was made using standard solution of sodium and potassium, and then the values of sodium and potassium concentrations of the samples were then recorded.

3.9 Statistical analysis

The obtained data were subjected to an appropriate statistical tool (descriptive statistics, a nova analysis and correlations) so as to detect the significance of any observed difference.

The data set was cleaned and edited for inconsistencies. Missing data were not statistically computed. Statistical analyses were performed using the Statistical Package for Social Sciences (version 16.0, SPSS, Inc) software. Descriptive statistics such as means and standard deviations were calculated for the continuous variables and frequencies for qualitative data.

4. RESULTS AND DISCUSSION

4.1 Study sample

He overall male (32 out of 48) to female (16 out of 48) ratio was 2:1. More than half (62.5%) the study samples referred to the hospital were from the Gezira State; (26 out of 48), while the rest (22 out of 48) were from Other States. The age distribution of both sexes ranged from (21-30 to 75-80) years (Table, 4.1) and Figure (4.1, 4.2).

4.2 Demographic and Anthropometric Profile of the Study Population

A total of 48 patients suffering from kidney stones were analyzed. Out of them both males and females were in the ratio 2:1Age of the sample ranged from 21 to 75 years. The mean age was 64.22 with SD 16.24 years. The study population had a total mean age ± SD was 64.22 ±16.24years (range 21 - 75) and the mean BMI ± SD was25.65 ±5.16 (Table 4.2). The distribution of BMI groups in the study population suggests that 14.5% were underweight, 33.3% were with normal weight while 31.2% had overweight and 22.9% were obese.

4.3 Clinical data of the patients referred to the Gezira Hospital for Kidney Disease and Surgery

Thirty five percent of the patients (most of them are males 12/17) underwent extracorporeal shock wavelithotripsy (ESWL), while 30% of the stones (most of them are males 4/14)) had been passed spontaneously, 20% (mot of them are males 7/10) were treated with open surgery after ESWL failure while 15% of the patients (most of them are males 4/7) were sub mitted to endoscopic treatment (Table, 4.3 and Figure, 4.3). The majority (28 out of 48) were over 50 years, while the rest (20 out of 48) were equal bellow 50. A nova proved anon- significant differences in the columns level (males & females; f =3.53, f-crit = 34.12) and in the rows level (type of clinical treatment; f= 3.53, f-crit= 29.46), i.e. the

observed differences in the clinical treatments of kidney stones among males and females can statistically be ignored.

Table 4.1: Age and sex distribution of patient.

Age	Sex		
	Female	Male	Number
21 – 30	4	8	12
31 – 40	4	4	8
41 – 50	5	5	10
51 – 60	2	5	7
61 - 70	1	4	5
71 – 80	0	6	6
Total	**16**	**32**	**48**
Residency			
Gezira State	20	6	26
Other States	12	10	22

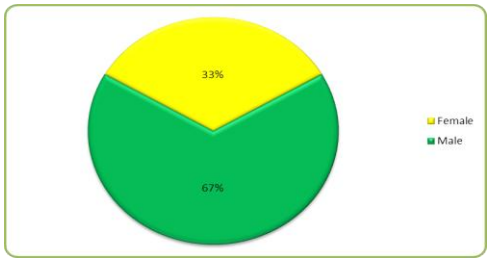

Figure 4.1: Distribution by sex of the patients with kidney stones.

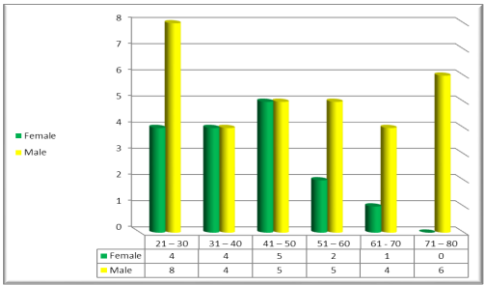

Figure 4.2: Distribution of Male and Female within the age interval.

Table 4.2: Demographic and Anthropometric Profile of the Study Population.

Variables	Minimum	Maximum	Mean	Std. Deviation
Age (years)	21	75	64.22	**16.24**
Height (cm)	150	187	166.47	**9.25**
Weight (kg)	38	108	71.9	**17**
BMI (kg/m^2)	15.42	36.29	25.65	**5.16**

Table 4.3: Clinical data of the patients referred to the Gezira Hospital for Kidney Disease and Surgery.

Characteristics	Male	Females	Total	%
ESWL	12	5	17	**35**
Open surgery	7	3	10	**20**
Endoscopic	4	3	7	**15**
Spontaneous passage	9	5	14	**30**
Total	32	16	48	**100**

ESWL: extracorporeal shock wave lithotripsy

SUMMARY		Count	Sum	Average	Variance
ESWL		2	17	8.5	24.5
Open surgery		2	10	5	8
Endoscopic		2	7	3.5	0.5
Spontaneouspassage		2	14	7	8
Total		2	48	24	128
Females		5	32	6.4	29.8
Male		5	64	12.8	123.7
ANOVA					
Source of Variation	**SS**	***Df***	**MS**	***F***	***P-value***	***F crit***
Rows	547.4	4	136.85	8.21	0.03	6.38
Columns	102.4	1	102.4	6.15	0.06	7.70
Error	66.6	4	16.65	8.21	0.03	6.38
Total	716.4	9				

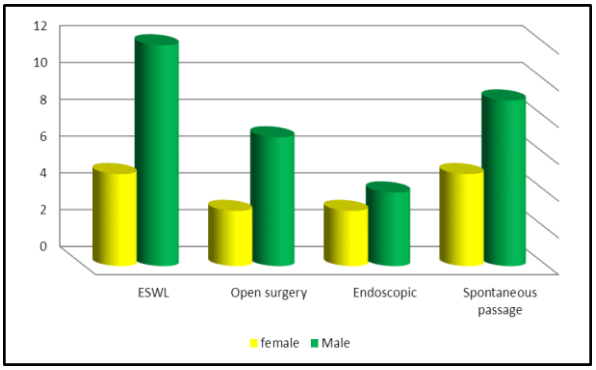

Figure 4.3: Clinical data of the patients referred to the Gezira Hospital for renal Disease and Surgery.

4.4 Chemical composition of kidney stones and their valid percentage

Among the total number of patients (48), the majority of their kidney stones containing calcium (35/48=72.9%), followed by oxalate (29/48=60.4%), uric acid (13/48=27%), phosphate (9/48=18.7%), carbonate (7/48=14.5%) and at last cystine (1/48=2%) as was presented in Table (4.4)and Figure (4.4).

It important to mention that, the single stone can give positive reaction to more than one chemical group.

It was clear that, calcium (23/35 in males), oxalate (19/35 in males) and uric (9/35 in males) were about 2:1 in respect to females (12/13, 10/13 and 4/13, follow the same order).

It was also observed that carbonate was more than 2 times in females (5/13) than males (2/13), while cystine found only as a single case in males.

A nova analysis proved a significant difference at the rows level (chemical composition; f=6.80, f-crit= 5.05), and anon-significant differences at the columns level (gerder; f=4.97, f-crit=6.60), i.e. the chemical groups of the kidney stones distributed at similar ratio between males and females, while that, the chemical composition did not.

4.5. Chemical constituents of the kidney stones

About 70.8% of these stones composed of calcium oxalate alone (34/48= 70.85), composed of calcium phosphate (10/48), while that 12.5% (6/48) composed of calcium carbonate. It was also observed that, 31.1% of the stones composed of both calcium oxalate and uric acid (15/48), while 16.6% of it composed of both calcium oxalate and calcium phosphate (8/48), and similar ratio of calcium oxalate, calcium phosphate and uric acid (Table (4.5)and Figure (4.5).

The statistical analysis showed that, the calcium oxalate constituent in all stone was about 2.5 times more than the average (mean), while calcium carbonate was the least constituent among all stones (about 2 time less), than the average.

Kidney stones affect about 10% of the worldwide population and are increasing in prevalence. Urine stone formation is known to be regulated by many factors, such as urinary stone-forming constituents, urinary pH, inhibitor and promoter of crystallization and crystal aggregation. The presence of stones may be asymptomatic, symptomatic, or discovered when the patient suffers complications. Recurrences are common in 30-50% of men, with the formation of another stone occurring within 5 years of first stone incidence. To decrease the likelihood of stone recurrence, patients are routinely advised to increase their urine volume by increasing their fluid intake (Straub and Hautmann, 2005).

Table 4.4: Chemical constituents of the kidney stones.

Type	Number	Percentage
Calcium oxalate	**34**	70.8
Calcium oxalate + Uric acid	**15**	31.1
Calcium phosphate	**10**	20.8
Calcium oxalate + Calcium phosphate	**8**	16.6
Calcium oxalate + Calcium phosphate + uric acid	**8**	16.6
Calcium carbonate	**6**	12.5
Mean	13.5	**28.07**
SE	4.29	**8.93**
Range	28	**58.3**
Min	6	**12.5**
Max	34	**70.8**
Sum	81	**168.4**
Count	6	**6**

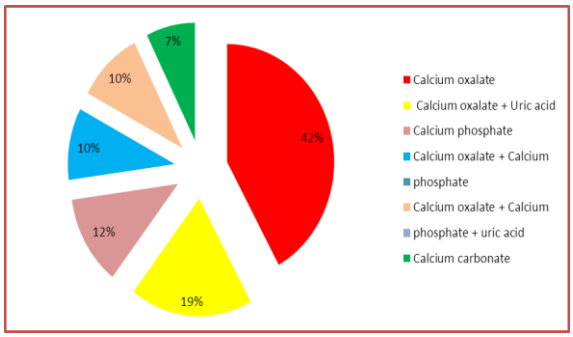

Figure 4.4: Chemical constituents of the kidney stones.

Table 4.5: Chemical composition of kidney stones and their valid percentage.

Sex	Males	Females	Total
Frequency	**32**	**16**	**48**
Calcium	**23**	**12**	**35**
Oxalate	**19**	**10**	**29**
Uric acid	**9**	**4**	**13**
Phosphate	7	**2**	**9**
Carbonate	**2**	**5**	**7**
Cystine	**1**	**0**	**1**

SUMMARY	Count	Sum	Average	Variance
Calcium	2	35	17.5	60.5
Oxalate	2	29	14.5	40.5
Uric acid	2	13	6.5	12.5
Phosphate	2	9	4.5	12.5
Carbonate	2	7	3.5	4.5
Cystine	2	1	0.5	0.5
Females	6	33	5.5	21.5
Males	6	61	10.16	80.96

ANOVA						
Source of Variation	SS	*Df*	MS	*F*	*P-value*	*F crit*
Rows	446.66	5	89.33	6.80	0.02	5.05
Columns	65.33	1	65.33	4.97	0.07	6.60
Error	65.66	5	13.13			
Total	577.66	11				

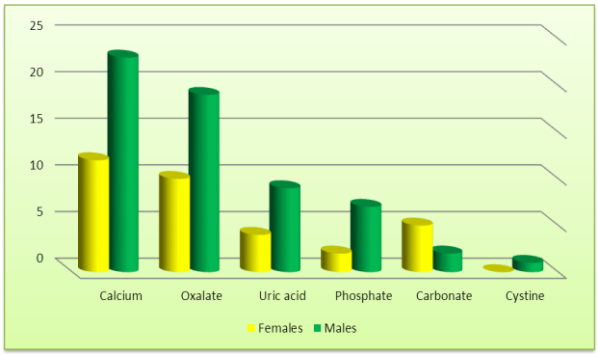

Figure 4.5: Distribution of kidney stones according to their chemical composition.

The aim of this study is to show the relationship between dietary habits and incidence of stone formation and analyze the components of Kidney stones in order to obtain data about stone formation and the possible ways of prevention and selecting treatment. In this study it was found that calcium, oxalate and uric acid stones were the most common, and less phosphate, carbonate and cystine. In Sudan, uric acid and uric acid dehydrate stones were more often seen in adults, while ammonium urate is often common in Sudanese. Almost similar finding were reported by (Khan *et al*., 2004) who found that the majority of the stones were composed of calcium followed by uric acid and phosphate, however, they didn't find cysine stones. In another study done byChou *et al.*, (2007),fewer cystine stone were found and a similar incidence of uric acid and calcium phosphate. The exact pathogenesis of kidney stone is unknown; however the nutritional and environmental factors may contribute to the development of stone formation. The choice of management of ureteric stones depends on the size of the stone and laterality of the stone. Open surgery for kidney stones was required for 20% of cases, and extracorporeal shock wave lithotripy was done in 35% of cases and 15% underwent endoscopy. The presence of minimally invasive procedures for stone retrieval (ESWL, endoscopy) which comprise about 50% of the surgical intervention indicates a good advance towards a better management of stone removal, with less dependence on open surgical procedures.

4.6. Demographic characteristic of clinical history of Diseases

Obesity was found in 28 patients (58.3%),gout was found in 25 patients (52%), diabetes mellitus was found in 30 patients (62.5%), hypertension was found in 20 patients (41.6%), cancerwas found in 20 patients (41.6%),Patients with history ofHistory of Chemotherapy was found in 20 patients (41.6%),Urinary obstructionwas found in 22 patients (45.8%),history ofkidney stoneswas found in 10 patients (20.8%).

Diabetic and Hypertension were the other risk factors closely associated with renal calculi in the study population. Those with hypertension are more likely to develop kidney stones, especially when they are overweight(Batmanghelidj and Kohlstadt, 2006). Obesity is another risk factor which contributes to kidney stones. Since body fat is hydrophobic, the proportion of body water decreases with increasing obesity, which can lead to dehydration(Breslau, 1988).The associated changes in body composition pose biophysical challenges associated with disturbed thermo genesis and dehydration. In addition, if the fluid intake is not balanced with dehydration it will lead to concentrated urine resulting in stone formation. But in our study trends are when overweight and obesity are considered as the risk factors(Table, 4.6 and Figure, 4.6).

4.7. Eating habits of kidney stone patients

The eating habits of kidney stone patients are presented in Table (4.7)and Figure (4.7). Data in the table revealed that 66.6% of total male patientswere non-vegetarian while 62.5% were vegetarian 4.16%.In case of female kidney stone patients, 33.3% were non-vegetarian, 27% were vegetarian 6.2%. Thehigh non-vegetarian diet may be a reason ofstones in the patients suffering with problems ofuric acid and cystine stones. Similar results were obtained by Vasanthamani and Sushmitha (1997)who reported that about 43% kidney stonepatients were non- vegetarian while only 5% were vegetarian. Sinha *et al*., (2010) also reported more incidences of kidney stones in nonvegetarians.

All kidney stone patients were asked for theirliking for salt in food. No standards were specified and results are based on the terms of taste.Salt consumption by kidney stone patients wasalso reported. The data obtained indicated that 56.2% of the total kidney stone patientswere taking high salt in their diet. In males, 56.2% were taking high salted food while 31.2% and 12.5% were taking respectively low and moderate amounts of salt in theirdiet. Among female patients, the consumption ofhigh salt was observed in 47.3%31.2%, were taking moderate amount of salt while 12.5% were consuming low amountof salt in their diet. High intake of salt amongkidney stone formers may also be a reason forrecurrence of stone. The common salt (sodium chloride) isbeing consumed in varying amounts by patientsas observed in the present investigation. Asreported by Carbone *et al*. (2003), sodium mayincrease urinary calcium excretion with whichchances of stone formation also increase.

4.10.Frequency of consumption of special foodstuffs by selected kidney stone patients

Data in the Table (4.8) reveals that egg was consumed daily by only 9.3% of male patients and 6.2% of female patients. Maximum males were taking egg weekly 31.2% and females on every alternate day 25%. Chicken was preferred regularly bymaximum male patients 68.7% and rarelyby female patients 56.2%. Fish was consumed rarely by male 62.5% and regularly by majority of female 62.5% patients. Sinha *et al*. (2010) reported that intake ofanimal foods being rich in urines is directly associated with risk of stone formation in cases ofkidney stone patients.

Table 4.6: Demographic characteristic of clinical history of Diseases.

Demographic data	Number	%
Obesity	28	58.3
Gout	25	52
Diabetes	30	62.5
Hypertension	20	41.6
Cancer	8	16.6
Urinary obstruction	22	45.8
Hyperparathyroidism	12	25
Family history of kidney stones	10	20.8

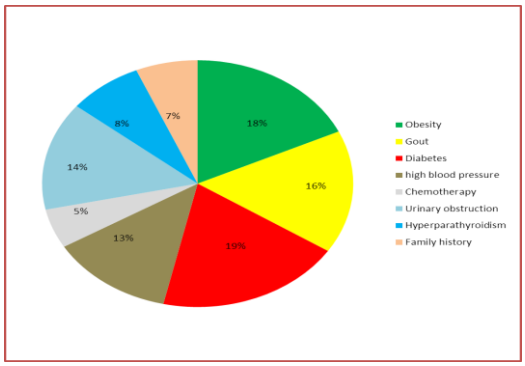

Figure 4.6: Demographic characteristic of the clinical history for the patients.

Table 4.7: Eating habits of kidney stone patients.

Particular	Male (n=32)	Female (n=16)	Total (N=48)
Eating Habits			
Vegetarian	2	3	**5**
Non –Vegetarian	30	13	**43**
Salt Consumption			
Low	10	2	**12**
Normal	4	5	**9**
More	18	9	**27**

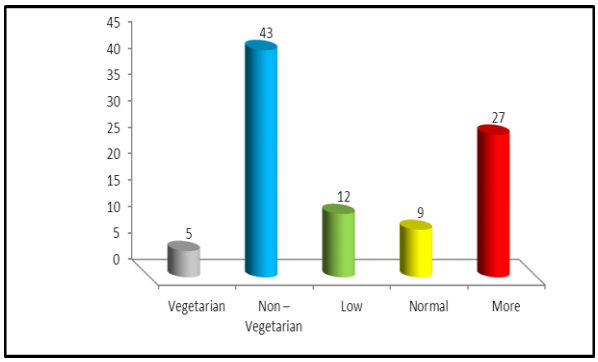

Figure 4.7: Eating habits and Salt Consumption of kidney stone patients.

Table 4.8: Minerals content (mg/100g) of some Sudanese popular foods.

Food type	Ca	K	Na
Peanut	92	70	**18**
Lentil	90	60	**15**
Wheat	41	90	**2**
Egg plant	9	30	**15**
Okra	20	30	**17**
Sweet potato	30	24	**28**
Tomato	13	44	**33**
Banana	5	58	**30**
Lemon	20	38	**36**
Dates	39	65	**2**
Milk	89	43	**77**
Cheese	90	29	**75**
Yoghurt	85	19	**22**
Egg	50	26	**37**
Fish	54	27	**39**
Chicken	16	79	**45**
Tea	43	34	**19**
Coffee	30	35	**22**

Similar observation wasmade in the present investigation. All the kidney stone patients were taking tea daily, which may be a risk factor fordevelopment of kidney stone as tea and coffeeare rich sources of oxalates. Coffee was preferred daily by 6.25% of males and on alternative days by 12.5% of female patients. 56.2% male and 43.7% female kidney stone patientswere taking coffee regularly. Gasinska and Gajeswka, (2007) investigated the feeding habitsof 22 adult (12 men and 10 women) patients withkidney stones from Poland to

determine the mainfood sources of oxalate and reported that themain dietary sources of oxalates were tea and coffee (80-85 %) in these patients. It wasconcluded that frequent consumption of oxalaterich foods such as tea and coffee is a significantrisk factor for kidney stones. About 15.6% of male patients and 12.5% of femalepatients were taking Soda water on alternativedays which may be a reason of stone formation in them.Drinking two or more colas per day is associated withdiabetes, hypertension and kidney stones.

Data of consumption of various foodstuffsby the kidney stone patients with special reference to their calcium, phosphorous, urine and oxalate content are presented on the basis of availability and seasonal consumption of different food stuffs.

Cereal Products and Pulses Among Pulses peanut and lentil were consumed rarely by majority of male kidney stone patients (43.7% and 43.7%, respectively) while respectively 50%and 56.2% of female kidney stone patients were regularly consuming these food items, however, peanutwas preferred by 28.1% of male kidney stone patients. There was only one male kidney stone patient who was taking Wheat daily and its consumption were rare among male and female kidney stone patients.Corn and wheat, which act as inhibitor of stone formation(Curhan *et al*., 2004). As evident from data such seasonal and rare consumption of some foods may be a cause of kidney stone formation in these patients.

Among vegetables, Okra was consumed weekly by majority of patients (40.6 and 56.2% males and female, respectively).Maximum male and female kidney stone patients (46.8% and 56.2%, respectively) were consuming Sweet potato. Brinjal was consumed regularly by both male 46.8% and female 62.5% kidney stone patients. This causes hyper-oxaluria which is a causative factor in formation ofkidney stones. Tomato was consumed daily by maximum kidney stone patients (43.7% males and 43.7% females). Excessive consumption of tomato may be one of the causes of stone formation. Banana was consumed on every alternateday by majority of patients (37.5% and 50% male and female, respectively). Lemon was consumed regularly by maximum male patients 56.2% The generalprecaution in dietary treatment of kidney stonesis to avoid foods which irritate the kidneys tocontrol the acidity or alkalinity of the urine. banana and lemon areknown as stone inhibitors as they are rich in Bcomplex vitamins and citric acid (Daudon, 2005).Milk and milk products are good source ofcalcium. Majority of kidney stone patients wereConsuming milk and curd daily (respectively, 62.5% and 56.2% males and 62.5% and 50% females). Cheese was taken regularly by maximum male 62.5% and female 56.2% patients. Consumption of milk and milkproducts in this may be a reason for formation ofstones in kidney stone patients as they are richin calcium. High blood level of calcium increasesexcretion of calcium in the urine. An abnormallyhigh intake of milk, alkalis or vitamin D may result in the formation of calcium phosphate stones(Vasanthamani and Sushmitha, 1997).

4.11 Water consumption by kidney stone patients

The amount of foodstuff and water consumed by kidneystone patients and the results are presented in Table (4.9 and 4.10)and Figure (4.8).This table revealed thatmajority of the male kidney stone patients 46.8% were consuming 1litter of water per day, followed by 25% consuming 1.5 litter, 21.8% consuming 0.5 litter and 6.25% consuming 2 litter. In case of females, majority 43.7% was consuming 1 litter of water per day, followed by 31.2% consuming 0.5 litters, 18.7 % consuming 1.5 litter and 6.2% consuming 2 litters.

4.12 Sodium, potassium and calcium

The results of the two cations (K^+ and Na^+) in all samples show that, their concentrations in water pot are higher than tap and cooler water samples, this may be due to the fact that the clay the water pot made of contain K^+ and Na^+ ions, all samples contain potassium concentration between (1.6 – 2.9) ppm, and sodium concentration between (11 – 2.8) ppm the concentration are within the permissible level reported by WHO (1993). The concentration of calcium in water pot in all samples except Gezira port locations are less than tap and cooler water (Table, 4.11and Figure, 4.9, 4.10 and 4.11). All samples values between (29 - 43) ppm which are within the permissible level reported by WHO (1993).

4.11 The pH, electrical conductivity (EC) and total dissolved solids (TDS)

The pH values between (7.3-7.65) that mean the samples fall in alkaline side and almost the values of pH in tap, pot and cooler water are the same within the permissible level reported by WHO. The results of total dissolved solid show all results of tap and cooler water are higher than water pot, may be due to clay of the water pot absorb solids, all results of TDS between (206-242) ppm within permissible level compared with that stated by WHO.The results presented in Table (4.12) show that, EC results of tap and cooler water are higher than water pot, may be due to high concentration of total dissolved solids in tap and cooler water, also all results within permissible level.

Table 4.9: Frequency of consumption of special foodstuffs by selected kidney stone patients.

Food Stuffs	Frequency of consumption									
	Male (n=32)					Female (n=16)				
	Daily	Alternate	Weekly	Regular	Rarely	Daily	Alternate	Weekly	Regular	Rarely
Cereal Products and Pulses										
Peanut	3	3	5	9	14	1	-	3	8	**4**
Lentil	-	-	5	13	14	-	-	2	9	**5**
Wheat	1	-	-	-	31	-	-	-	-	**16**
Vegetables										
Egg plant	-	6	9	15	2	-	-	4	10	**2**
Okra	-	7	13	9	4	-	5	9	1	**4**
Sweet potato	-	2	9	15	6	-	-	9	2	**5**
Fruits										
Tomato	14	6	-	2	8	7	3	2	-	**4**
Banana	5	12	7	7	1	5	8	3	1	**-**
Lemon	-	4	9	18	1	4	7	3	2	**-**
Dates	-	-	2	15	15	-	-	3	5	**8**
Milk And Milk Products										
Milk	20	4	8	-	-	10	4	2	-	**-**
Cheese	-	-	9	20	3	-	-	5	9	**2**
Yoghurt	18	10	4	-	-	8	5	2	1	**-**
Animal Foods										
Egg	3	5	10	8	6	1	4	3	2	**6**
Fish	-	-	3	9	20	-	-	1	10	**5**
Chicken	-	-	-	22	10	-	-	3	4	**9**
Nuts and Beverages										
Tea	32	-	-	-	-	16	-	-	-	**-**
Coffee	2	1	4	7	18	-	3	2	4	**7**
Soda	-	5	9	3	15	-	2	4	7	**3**

Table 4.10: Water consumption by kidney stone patients.

Particular	Male (n=32)	Female (n=16)	Total (N=48)
Amount of Water Consumed/ Day **(liter)**			
0.5	7	5	**12**
1	15	7	**22**
1.5	8	3	**11**
2	2	1	**3**

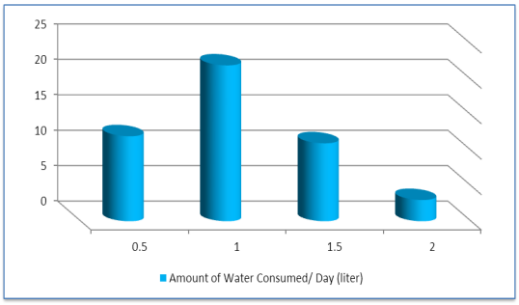

Figure 4.8: Water consumption by kidney stone patients.

Table 4.11: Sodium, potassium and calcium in some samples of water.

Location and sample types	Ca^{+2}			K^{+}			Na^{+}		
	min	max	average	Min	max	average	min	max	Average
Nishasheiba Tap water	34	38	36	2.0	2.2	2.1	13.2	13.4	13.3
Water pot	30	32	31	2.4	2.8	2.6	14	14.6	14.3
Cooler water	33	35	34	2.1	2.2	2.15	13.0	13.6	13.3
Aedadia Tap water	30	34	32	2.2	2.2	2.2	17.6	17.7	17.65
Water pot	29	31	30	2.7	2.9	2.8	18.0	18.2	18.1
Cooler water	33	35	34	2.2	2.4	2.3	17.6	17.7	17.65
Gezira port Tap water	35	37	36	1.6	1.9	1.75	11.0	11.3	11.15
Water pot	37	40	38.5	2.1	2.3	2.2	11.2	11.6	11.4
Cooler water	40	43	41.5	1.8	1.9	1.85	11.1	11.4	11.25

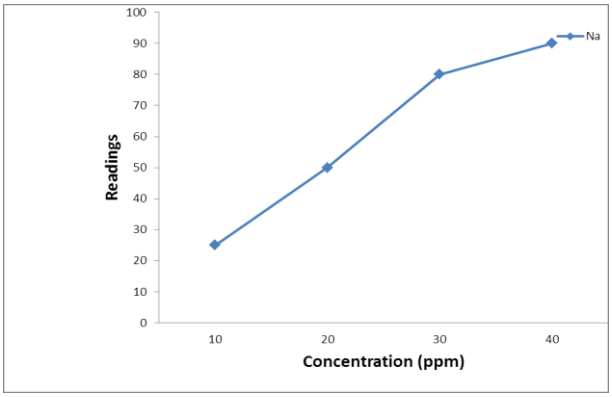

Figure 4.9: The sodium standard curve.

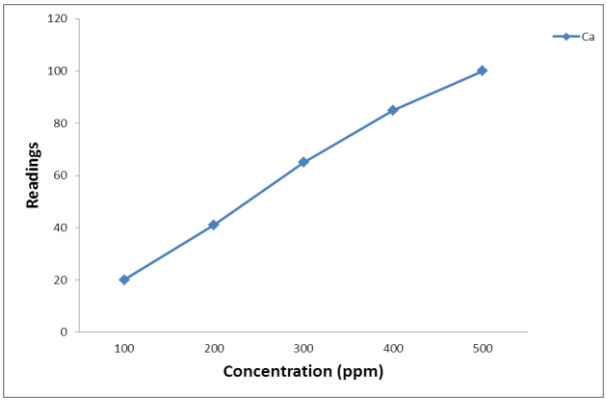

Figure 4.11: The calcium standard curve.

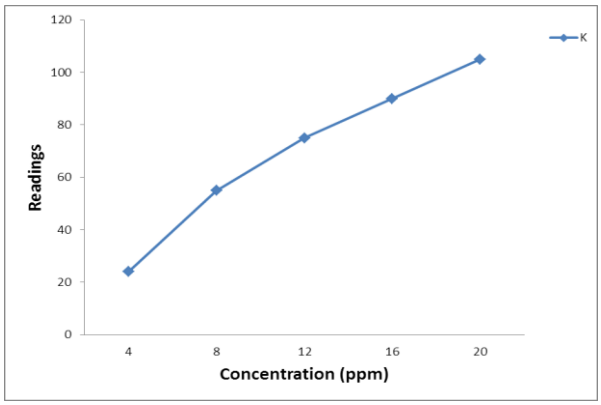

Figure 4.10: The potassium standard curve.

Table 4.12: The pH, electrical conductivity (EC) and total dissolved solids (TDS) of some samples of water collected from some locations.

Location and sample types	Ph			TDS			EC		
	min	max	Average	min	Max	Average	min	max	Average
Nishasheiba Tap water	7.2	7.4	7.3	218	220	219	364	367	365.5
Water pot	7.3	7.4	7.35	218	218	218	363	363	363
Cooler water	7.4	7.5	7.45	222	224	223	368	372	370
Aedadia Tap water	7.3	7.6	7.45	231	234	232.5	388	390	389
Water pot	7.5	7.8	7.65	230	233	231.5	383	385	384
Cooler water	7.5	7.6	7.55	238	242	240	400	404	402
Gezira port Tap water	7.4	7.4	7.4	214	216	215	359	363	261
Water pot	7.2	7.5	7.35	206	209	207.5	348	352	350
Cooler water	7.4	7.6	7.5	213	215	214	357	360	358.5

4.12 Chloride, sulphate, carbonate, bicarbonate concentrations (ppm)

As an observation carbonate and bicarbonate results the rat and the tap water carbonate values (Table, 4.13) were low and obtain bicarbonate were high, may be due to conversion of carbonate to bicarbonate as the in equation below:

$CO^{-2}_3 + H^+ \longleftrightarrow HCO_3$

The results of chloride show that all samples of water pot have chloride concentration higher than tap and well water may be due to clay of water pot contain chloride ions, all samples between (0.89 - 8.7) ppm within permissible level reported by WHO.

The sulphate values between (3.4 - 4.7) and almost the values of the sample in top, pot and cooler water are same values within the permissible level reported by WHO.

Table 4.13: The Chloride, sulphate, carbonate, bicarbonate concentrations (ppm)of some samples of water collected from some locations.

Location and sample types	CL^-			SO_4^{-2}			CO_3^{-2}			HCO_3^-		
	min	max	Aver.	min	max	Aver.	Min	max	Aver.	min	Max	Aver.
Nishasheiba Tap water	5.1	5.1	1.5	4.1	4.1	1.4	11	11	11	109	110	109.5
Water pot	8.6	8.7	8.65	4.3	4.3	4.3	13	14	13.5	103	103	103
Cooler water	5.0	5.1	5.05	4.3	4.4		13	15	14	100	105	102.5
Aedadia Tap water	3.0	3.3	3.15	4.2	4.4	4.35	9	9	9	100	103	101.5
Water pot	6.0	6.4	6.2	4.5	4.7	4.3	12	12	12	110	112	111
Cooler water	3.3	3.3	3.3	4.4	4.7	4.55	13	13	13	116	119	117.5
Gezira part Tap water	1.0	1.1	1.0	3.4	3.4	3.4	7	9	8	108	111	109.5
Water pot	1.6	1.7	1.65	3.5	3.7	3.6	8	8	8	95	99	97
Cooler water	0.89	0.89	0.89	3.5	3.7	3.6	9	10	9.5	98	101	99.5

5. CONCLUSIONS

1- Calcium oxalate stones remain the most frequent components followed by uric acid.
2- Gender differences are reported with males having higher prevalence of uric acid stones while females suffering from calcium oxalate stones.
3- Low fluid intake is significantly associated with stones prevalence.
4- The majority of patients were non-vegetarian 89.5% and many of them were consuming more salted foods 56.2%. Majority 65% of the patients were consuming only 1litter of water per day which may be cause of stone formation in them as less water consumption increases crystallization
5- Food preferences in terms of overall frequency of consumption of special foodstuffs revealed that frequency varied from weekly to rarely for most of the foodstuffs from various selected categories. Tea, coffee, milk products and vegetables were most consumed by the patients which may be a cause of stone formation in them.

6. RECOMMENDATIONS

- Drink plenty of fluids (2-2.5 litter per day) to prevent stone formation. Take banana, carrots, bitter guard andwhich are rich in stone inhibitors.
- Take more citrus fruits such as, lemon and orange to dissolve the stones.
- Avoid foods rich in oxalates such as tomato and cucumber.
- Avoid excessive use of animal foods such as chicken, egg, fish and meat to prevent uric acid stone formation.
- Restrict cauliflower, egg plant and pumpkin in diet as they form uric acid stones.
- Analyzing the kidney stones routinely for both sexes may be better for understanding of the mechanisms involved in lithogenesis.
- Health education should be directed towards changing people lifestyle such as to avoid overweight.
- People awareness should be increased about the disease and its complications.
- Treatment of such diseases should be as fast as possible.

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