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Urolithiasis, Epidemiology, Etiological factors, and Treatment: An Overview

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Abstract: **Urolithiasis, also referred to as the formation of urinary stones, is a health issue that affects nearly all populations worldwide; it triggers extreme acute neck and back pain and periodically results in more extreme problems, such as pyelonephritis or severe renal failure. The present review primarily aim was to focuses on main three aspects of urolithiasis; Epidemiology, causes and factors contributing in urolithiasis, and finally treatment approaches. A computerized searched was conducted through several databases; PubMed/MIDLINE, Embase, for relevant studies discussing the urolithiasis prevalence, or causes and treatment, all studies that were published in English language with human subjects up to December, 2016. each identified study references list was manually searched for more relevant studies. The urolithiasis patients with a family history revealed greater serum calcium levels than did those without a family history and a propensity for increased excretion of urinary calcium. Lots of aspects of the system underlying kidney stone development stay unclear at present; hence, a much better understanding of this complex system will lead to the development of unique techniques for avoiding this disease, treatment choices for patients with urinary stones advanced considerably over the past couple of decades. Understanding about each treatment method including its benefits and unfavorable results is necessary for physicians to be able to pick the very best choice for the patients.**

Keywords: **Urolithiasis, Epidemiology.**

1. INTRODUCTION

Urolithiasis, also referred to as the formation of urinary stones, is a health issue that affects nearly all populations worldwide; it triggers extreme acute neck and back pain and periodically results in more extreme problems, such as pyelonephritis or severe renal failure [(1,2)]. Kidney stone formation is a typical urological problem with a life time prevalence of approximately 10% in males and 6% in ladies, and its frequency has been increasing in many developed nations [(2,3,4)], with a reoccurrence rate of nearly 60% within 10 years after initial treatment [(5)]. Urolithiasis is a multifactorial disease resulting from complicated interactions in between environmental and hereditary factors. Environmental factors, such as lifestyle, obesity, dietary routines and dehydration, have been linked in urolithiasis advancement, [(6,7)] whereas hormone, genetic or anatomical factors may likewise influence its pathogenesis [(8)]. Appropriate fluid intake and dietary modifications may successfully prevent stone recurrence. Thiazide, potassium alkali, allopurinol and tiopronin have been shown to be reliable in the treatment of different kinds of stones. Couple of novel therapies have actually emerged over the previous years, and treatment of particular types of stones remains tough. Research studies evaluating the pathophysiology and pathogenesis of stone disease are currently ongoing. More than 80% of patients with kidney stones experience urolithiasis caused by calcium oxalate [(1,3)]. Urolithiasis is a disease known from ancient times, even now, many researchers are trying to elucidate the mechanism of calcium oxalate kidney stone formation. The physiochemical systems of stone development through precipitation, growth, aggregation and concretion of various lithogenic salts in urine are still in dispute [(4,8)].

Urolithiasis is usually found in white children, with African American and Asian children just hardly ever affected [(9,10)]. Seventy-five percent of children who have nephrolithiasis have a recognizable predisposition to stone formation [(11,12)].

Metabolic risk factors account for more than 50% of cases [11,12], structural urinary system irregularities represent 32%, and infection accounts for 4% [12]. It is not uncommon to discover more than one predisposing consider the evaluation of a child who has nephrolithiasis [11].

The present review primarily aim was to focuses on main three aspects of urolithiasis; Epidemiology, causes and factors contributing in urolithiasis, and finally treatment approaches.

2. METHODOLOGY

A computerized searched was conducted through several databases; PubMed/MIDLINE, Embase, for relevant studies discussing the urolithiasis prevalence, or causes and treatment, all studies that were published in English language with human subjects up to December, 2016. each identified study references list was manually searched for more relevant studies.

3. RESULTS

Just recently numerous studies evaluated at the pathogenesis of the early phase of urinary stone formation in association with renal tubular cell injury. Calcium oxalate crystals comply with epithelial cells, and NADPH oxidase creates superoxide, which triggers cyclophilin D in mitochondria. Acceleration of the MPT led to mitochondrial collapse, OS, activation of the apoptotic path, and strong expression of OPN in the preliminary process of renal calcium condensation in both in vitro and in vivo experiments (**Figure 1**) [13]. Cyclosporin A and NIM811, a unique selective inhibitor of cyclophilin D activation, successfully blocked the acceleration of MPT, expression of OPN and renal calcification [13,14]. As NIM811 is a 4- replaced cyclosporin that does not bind to cyclophilin A, it lacks immunosuppressive and anti-inflammatory activities. NIM811 and cyclosporin A have similar constitution solutions, however various structural formulas [14].

Cyp D activation caused the velocity of MPT, thus altering the mitochondrial transmembrane capacity and causing mitochondrial collapse. Cytochrome c is an essential protein that induces apoptosis. On mitochondrial collapse, cytochrome c is released into the cytosol and binds to cytosolic apoptotic protease-activating factor 1, consequently triggering caspase-9 and caspase-3, and starting apoptosis During this procedure, ROS are likewise released from the intramembranous compartment into the cytosol, more hurting kidney tubular cells [15]. Inhibiting Cyp D activation avoided renal calcium formation brought on by mitochondrial collapse, OS and activation of the apoptosis pathway. Targeting Cyp D activation through genetic and/or pharmacological methods may use new therapeutic strategies for kidney stone disease [15].

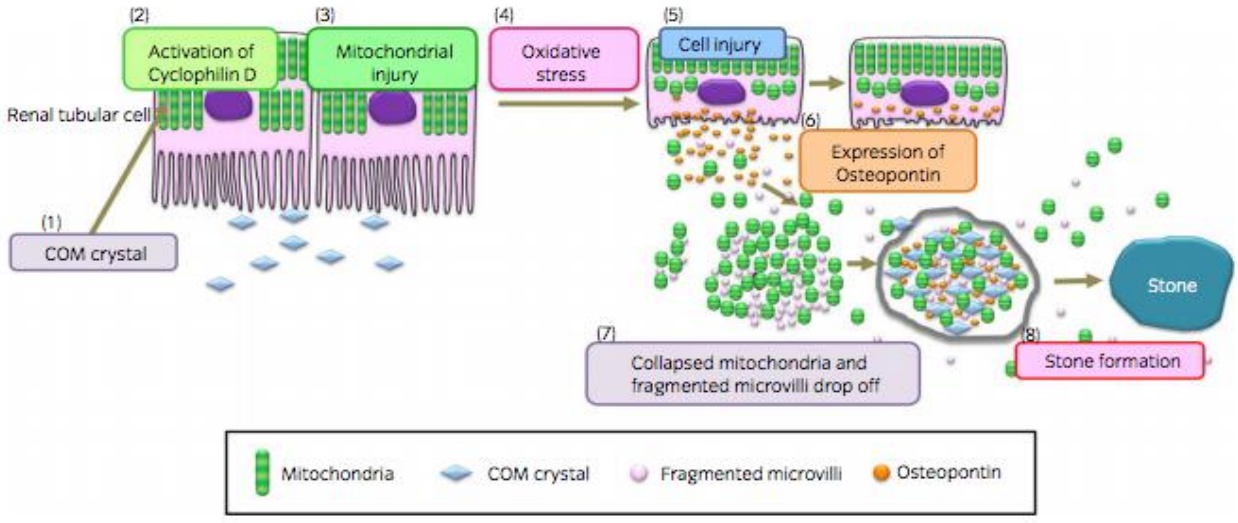

Figure1: Proposed pathway of kidney stone formation. In the proposed pathway, calcium oxalate monohydrate (COM) crystals attach to renal tubular cells (1), leading to activation of cyclophilin D (2). Subsequently, mitochondrial collapse (3) and oxidative stress occur (4). These events activate apoptosis, cell injury (5) and osteopontin expression (6). Thereafter, collapsed mitochondria and fragmented microvilli drop off into the urine (7) and condense into kidney stones (8). [13]

- ***Epidemiology of urolithiasis in general population worldwide:***

Recent epidemiological studies have actually shown an increased occurrence of kidney stones in patients with lifestyle-related diseases, such as obesity [7], type 2 diabetes [16] and high blood pressure [17,18]. Collectively, these medical conditions are now called metabolic syndrome, which has received a great deal of attention in the last few years as a risk factor for heart disease development [19]. Metabolic syndrome has likewise been related to kidney stone disease [20,21], and numerous research studies have discovered increased urinary oxalate and calcium excretion, and reduced citrate excretion in patients with obesity or diabetes [22,23]. Other research studies reported that high blood pressure was connected with hypercalciuria and hypocitraturia [24,25]. In Germany [26] a more increase in the prevalence and incidence of urolithiasis has been explained. Frequency has actually risen from 4% to 4.7% from 1979 to 2001. In the year 2000, the incidence of urolithiasis in Germany was found to be 1.47% (1979: 0.54%). Data from the United States (United States) are inconsistent. Inning accordance with the data from the National Health and Nutrition Examination Survey II and III, renal stone occurrence among 20 to 74 old US locals was greater in 1988 to 1994 than in 1976 to 1980 (5.2% vs 3.8%) and it was greater in males than females [27]. On the contrary data for the Rochester population for many years 1970-2000 demonstrated an age-adjusted incidence of new start symptomatic stone disease for males of 155.1 and 105.0 per 100,000 per year in 1970 and 2000, respectively, and for females of 43.2 and 68.4 per 100,000 per year, respectively [28]. During the 30 years, rates for ladies increased by about 1.9% annually, whereas rates for men declined by 1.7% annually.

Other population-based studies examined prevalence and incidence rates of urolithiasis in different nations. A surprisingly high 15% occurrence of urolithiasis was observed in the rural population of Thebes in Greece [29]. In Iceland [30] the age-standardized occurrence for the 30-79 years' age group was 4.3% for men and 3.0% for females, with no significant increase in time. The occurrence was 562 per 100 000 per year amongst men and among ladies was 197 per 100 000 annually. Information from some developing nations showed frequency figures much like those previously explained in Western nations. In Iran [31] the prevalence was approximated as 5.7%, a little more frequent in males (6.1%) than women (5.3%) whereas the yearly incidence of urolithiasis in 2005 was 145.1. In establishing countries, the prevalence of stones is most likely underestimated considering that quiet and not yet found kidney stones were diagnosed by renal sonography in 3% of non-symptomatic subjects [32].

- ***Incidence or urolithiasis in Children population:***

Urolithiasis in children is traditionally characterised by a pattern of either endemic bladder stones in young kids in establishing countries or relatively rare calcium-based stones in upper system stones in Western countries. In children upper urinary tract were generally related to urinary system anomalies and infection rather than with metabolic disruptions. A shift in the public health of paediatric kidney stone disease in the United Kingdom (UK) was observed over the past 30 years [33]. Hidden metabolic causes are now the most typical however can be masked by existing side-by-side urinary system infection. In Croatia [34] stone structure, location and etiology in children resembled those in developed Western nations, calculi being mainly located in the upper urinary tract (90%) with primary constituent of calcium oxalate followed by struvite. The occurrence of kidney stones in Icelandic children [35] is high compared to other Western populations, affecting women more than males, with an annual occurrence of renal stones of 5.6 and 6.3 per 100,000 children less than 18 and 16 years of age, respectively. Underlying metabolic risk factors were determined in many patients. In Iraqi children primary endemic bladder calculi are now less regular than in the past. Metabolic disorders were the major causes of urinary stones, but can be masked by associated urinary system infections [36]. Staghorn calculi associated with frequent urinary system infection accounted for 15% of the cases. In other nations endemic childhood bladder stones are still regular. In Lao bladder stone occurrence was associated with an history of regular episodes of diarrhea and early intro of white rice into diet as early as the first week of life [37]. A strange kind of stone disease was described in Aborigenal children living in tropical and desert areas of Australia who are at risk of establishing urate stones in their upper urinary system not related to structural anomalies [38]. A report from the Goldfields area of Western Australia [38] described urate kidney stones as a typical finding in Aborigenal children. Carbohydrate intolerance might be an etiological factor together with chronic diarrhea and intraluminal breakdown of sugars by enteric germs resulting in a condition of chronic metabolic acidosis [38].

- ***Etiological factors for urolithiasis:***

Hypercalciuria is the most crucial risk consider calcium stone formation, happening in 35% to 65% of cases. Urinary calcium raises the ionic calcium concentration and increases the urinary saturation of stone-forming calcium salts (calcium-phosphate or calcium-oxalate) [39,40]. In addition, complexation of calcium with urinary inhibitors such as citrate

and glycosaminoglycans decreases urinary repressive activity, therefore increasing stone risk [41,42]. Hypercalciuria can be accompanied by diseases causing a hypercalcemic state such as primary hyperparathyroidism, myelo-proliferative disease, vitamin D intoxication, or Cushing's syndrome, many hypercalciuria is idiopathic and results from hyper-absorption of calcium in the intestinal tract or failure of calcium reabsorption in the kidney tubule [39]. In addition, hypercalciuria may have a hereditary predisposition, and about half of patients who have hypercalciuria have a family history of stone disease [43].

Recent research studies have recommended a close relation between urolithiasis and family history [44,45]. Urolithiasis develops more frequently in people with a family history of kidney stones than in those without a family history, however little details is readily available relating to whether the increased risk is attributable to hereditary factors, environmental direct exposures, or some mix. A favorable family history of stones has actually been reported in 16% to 37% of patients who have actually formed a kidney stone, compared to 4% to 22% in healthy control subjects [46]. Formerly, Curhan et al analyzed the association between family history and risk of kidney stone formation in a friend of 37,999 male participants in the Health Professionals Follow-up Study [44]. In their study, kidney stone formers with family history had significantly higher urinary calcium excretion than did those without a family history. They likewise showed that a family history of kidney stones considerably increased the risk of stone formation. Of interest, Lerolle et al reported a significant dose-effect association between calciuria and stone disease in patients with familial hypercalciuria [45].

Uric acid is a final result of purine metabolic process and works as the nucleus of urinary formation to induce the formation of calcium oxalate stones. It is likewise responsible for urinary stone formation by lowering the activity of inhibitors in the urine [47]. Hyperuricosuria may also be connected to a family history. This relation can be supported by the fact that the metabolic process and excretion of uric acid might be influenced by inherited factors and that men with gouty diathesis are at increased risk of stone development [47]. Curhan et al discovered increased urinary excretion of uric acid in a group with a family history of kidney stones, however statistical significance was not reached [44]. Urinary oxalate excretion has actually been thought to be essential for urinary stone development due to the fact that a small change in urinary oxalate can trigger an excellent modification in urinary calcium-oxalate saturation. There are recognized hereditary conditions resulting in an overproduction of oxalate, such disorders are really rare and the association in between hyperoxaluria and a family history is still not apparent [44].

- ***Treatment of urolithiasis:***

Medical expulsive therapy (MET) is a watchful waiting technique for dealing with urethral calculi and can be used successfully for a considerable number of patients [48,49]. About 70% of ureteric stones are found in the lower third of the ureter at the time of presentation [48]. Stones located in the distal portion of the ureter will have a successful spontaneous stone passage in about 50% of cases [48]. The stone expulsion time depends upon many factors including stone size, location, and associated blockage [50]. A watchful method can result in a number of complications such as urinary tract infections, hydronephrosis, and colic events [51]. Inning accordance with American Urological Association (AUA) standards [52], the estimated spontaneous passage rate for stones <5mm is ranging from 71% to 98%, and for those measured 5 to 10 mm, stone passage rate is 25% to 53%. It has actually been estimated that the passage time for stones less than 2 mm is 8 days and for stones 4-6 mm, it might take 22 days for the passage of stones, respectively [50]. It is not recommended to extend this conservative technique beyond 6 weeks, due to its prospective risk of issues [51,52].

Extracorporeal shock wave lithotripsy (ESWL) for treatment of urolithiasis:

ESWL was developed in Germany by Chaussy et al. and have transformed the treatment of both kidney and urinary lithiasis. Because its introduction in early 1980s, ESWL has become the first line treatment for kidney stones, proximal stones, and midureteral stones because of its noninvasive nature, low costs, high performance of stone disintegration, less exposure of patients to anesthesia, shorter hospitalization and fewer complications [44].

ESWL is consisted of shattering forces produced by an external source of power called lithotriptor, which produces high strength and radio frequency acoustic waves. All lithotripsy devices consist of 4 elements: an energy source, a focusing system, a localization unit, and a coupling machine. The shock waves are concentrated straight onto kidney or ureteral stone. The system of fragmentation counts on cavitation, spalling, and shear [53]. Cavitation is thought about to be the most important force responsible for fragmentation of the stones into smaller sized pieces which can then be easily gone through the ureters [53].

Inning accordance with AUA Ureteral Stone Clinical Guidelines [54], ESWL is thought about as the very first line treatment method for calculi less than 1 cm. When stone is located in the lower pole [55], the success rate of ESWL reduces. Lingeman et al. reported stone-free rates of roughly 30% for patients with lower pole calculi of 11 - 20 mm and 20% for patients with calculi > 20 mm [55].

Current evidence has suggested the utility of ESWL for proximal ureteral stones which can be broadened to stones as much as 15mm [56]. Shafi et al. reported the success rate of 78.6% after 3 months of follow-up and also most of patients prefer ESWL over other treatments [56]. Contraindications for ESWL treatment consist of pregnancy, unrestrained urinary system infections and blockage, decompensated coagulopathy, arrhythmia, unchecked high blood pressure and kidney artery or abdominal aortic aneurysm [57].

4. CONCLUSION

The urolithiasis patients with a family history revealed greater serum calcium levels than did those without a family history and a propensity for increased excretion of urinary calcium. This outcome recommends that a family history of urolithiasis might be associated with irregularities in calcium metabolism. Lots of aspects of the system underlying kidney stone development stay unclear at present; hence, a much better understanding of this complex system will lead to the development of unique techniques for avoiding this disease

While surgical techniques are still considered the pillar of treatment for urolithiasis, medical expulsion therapy has actually just recently emerged as an alternative treatment method for the management of distal ureteric stones. treatment choices for patients with urinary stones advanced considerably over the past couple of decades. Understanding about each treatment method including its benefits and unfavorable results is necessary for physicians to be able to pick the very best choice for the patients.

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