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Uric Acid Nephrolithiasis: An Updated Review

Haya Muhammad Al-Qahtani[1], Noura Saad Aldosari[2], Turki Mohammad Hasssan Almalki[3], Sarah Bhni Mohamed Al Haili[4], Amal Jadah Abu Al-Qasim Hakimi[5], Khalid Ayed Aoudh Alharthi[6], Marwah Mohammed Nabush[7], Adel Ali Hakami[8], Fahad nigiri Aldawsari[9], Naya Shujaa Aldalbahy[10], Asmaa Mohammed Nasser Al matrad[11], Mohammed Ali Abdul Hadi Al-Ghamdi[12], Sultan Abdullah Alhuwaymil[13], Jazaa Mohsen Al-Mutairi[14], Mohammed Ahmed Hassan Maslof[15]

Abstract

Uric acid nephrolithiasis is a prevalent condition marked by the formation of kidney stones from uric acid crystal deposition. This disorder is often linked to hyperuricemia and acidic urine, with multiple risk factors, including obesity, diabetes, hypertension, and metabolic syndrome. Uric acid stones, which account for 5-40% of urinary stones globally, have historically caused significant pain and require advanced treatments.This review aims to update the understanding of uric acid nephrolithiasis, focusing on its etiology, epidemiology, pathophysiology, and current treatment strategies.The review synthesizes data from numerous studies on the biochemical formation, risk factors, and management of uric acid stones. It emphasizes the role of urinary pH, hyperuricosuria, low urine volume, and genetic predispositions in stone formation. The impact of diet, genetic disorders, and environmental factors is also discussed.Uric acid stones primarily form under conditions of low urinary pH, which impairs the solubility of uric acid. Diets high in purines, dehydration, and metabolic disorders significantly increase the risk of stone formation. Key risk factors include hyperuricosuria, obesity, and acidic urine. Advances in treatment now allow the dissolution of many uric acid stones by increasing urinary pH and volume while managing hyperuricosuria. However, recurrence remains common, and the condition is associated with high healthcare costs.Uric acid nephrolithiasis remains a significant clinical challenge with diverse etiology and risk factors. Early diagnosis and intervention, including dietary modifications, adequate hydration, and medical management to increase urinary pH and reduce uric acid levels, are crucial in preventing recurrence. Continued research into the molecular mechanisms of stone formation and novel treatments is essential for improving patient outcomes.

Keywords: *Uric Acid Nephrolithiasis, Kidney Stones, Hyperuricemia, Urinary Ph, Metabolic Syndrome, Hyperuricosuria, Stone Prevention.*

Introduction

Uric acid nephrolithiasis, a prevalent form of kidney stone disease, arises from the deposition of uric acid crystals within the kidneys. These crystals may aggregate to form stones, which can result in painful urinary tract obstructions that are prone to recurrence. This condition is frequently associated with elevated levels of uric acid in the bloodstream, a condition known as hyperuricemia. Notably, nearly two-thirds of uric acid kidney stones can be dissolved by increasing urinary pH and volume while simultaneously reducing

[1] Ksa, Ministry Of Health, Al Quwayiyah Health Center
[2] Ksa, Ministry Of Health, Ad Diriyah Hospital.
[3] Ksa, Ministry Of Health, Tabuk First Ambulance Center.
[4] Ksa, Ministry Of Health, First Riyadh Cluster
[5] Ksa, Ministry Of Health, Abu Arish General Hospital
[6] Ksa, Ministry Of Health, Sabtalalaya Hospital
[7] Ksa, Ministry Of Health, King Fahd Central Hospital Jazan
[8] Ksa, Ministry Of Health, King Fahad Hospital
[9] Ksa, Ministry Of Health, Shaqra General Hospital
[10] Ksa, Ministry Of Health, Methnab General Hospital
[11] Ministry of Health Al-Yamamah Health Center
[12] Ksa, Ministry Of Health, Al-Makhwah General Hospital
[13] Ksa, Ministry Of Health, Tebrak Phc
[14] Ksa, Ministry Of Health, Prince Salman Bin Mohammed Hospital In Dalam
[15] Ksa, Ministry Of Health, Al Gharb Dental Complex,Riyadh

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hyperuricosuria. Nephrolithiasis represents a common health issue in developed nations, affecting approximately 2 to 5% of individuals at least once in their lifetime globally. Several factors contribute to an individual's susceptibility to nephrolithiasis, including genetic predispositions, underlying metabolic abnormalities, as well as dietary and environmental influences [1][2][3][4]. Medical conditions such as metabolic syndrome, obesity, diabetes mellitus, gout, hypertension, and chronic renal failure are also frequently associated with the development of uric acid nephrolithiasis [5][6]. The identification of uric acid as the primary acidic component of bladder stones is credited to Swedish pharmacist C. Scheele, who first observed this in 1776 [7]. Historically, uric acid kidney and bladder stones have been a source of significant pain and suffering, with the only available treatment in the pre-modern era being high-risk surgery, which carried a considerable risk of mortality [8]. Notably, prominent historical figures, including Sir Isaac Newton and Michelangelo, are known to have suffered from recurrent uric acid kidney stones.

Etiology

Uric acid is the end product of purine metabolism in humans, with purines originating from three main sources: cellular RNA from cell turnover, hepatic metabolic synthesis, and the dietary intake of high-purine foods. The digestion of purines results in the production of xanthine, which is subsequently converted to uric acid through the enzymatic action of xanthine oxidase. Inhibitors of xanthine oxidase impede this conversion, and since xanthine is more soluble than uric acid, it can precipitate and contribute to nephropathy at elevated levels. While most mammals, aside from humans and higher primates, metabolize uric acid to allantoin, a highly soluble compound, this does not result in the clinical complications seen in humans [9]. Endogenous synthesis of uric acid in humans remains relatively stable at approximately 300-400 mg per day, with dietary sources contributing less than 50% to total daily uric acid production. On a typical Western diet, the total daily excretion of uric acid is estimated to be around 10 mg/kg of body weight [10]. However, dietary factors, particularly the consumption of high-purine foods, can significantly increase urinary uric acid excretion by 50% or more [11]. The primary risk factor for the formation of uric acid stones is acidic urine, which is often linked to low urinary ammonia levels. Other important risk factors include hyperuricosuria and low urine volume. Hyperuricosuria not only leads to the formation of uric acid stones but also contributes to the development of calcium oxalate urolithiasis, the most common type of kidney stone, accounting for 70% to 80% of cases. It is hypothesized that monosodium urate may act as a nidus for the precipitation of calcium salts, while also eliminating mucopolysaccharides that typically inhibit calcium oxalate crystallization [7][13][7].

Classification of Etiology

The etiology of uric acid stones can be classified into three categories: idiopathic, acquired or secondary, and congenital [14][15]. Idiopathic causes, which are most frequently linked to metabolic disorders, include conditions such as aciduria (low urinary pH), diabetes mellitus, metabolic syndrome, and obesity. Acquired or secondary causes, which are less common, are associated with factors such as low urinary pH, low urinary volume, and hyperuricosuria. These include gout, which is characterized by elevated serum uric acid levels and hyperuricosuria, persistent diarrhea linked to irritable bowel syndrome or post-surgical changes, cancer (especially during chemotherapy), and high-purine dietary intake (e.g., organ meats, poultry, fish, and red meat). Additionally, medications such as probenecid, sulfinpyrazone, indomethacin, losartan, and salicylic acid can exacerbate uric acid levels [16][17]. Congenital causes, though rare, include conditions like Lesch-Nyhan syndrome, Von-Gierke disease, and familial hypouricemic hyperuricosuria, among others [16][17]. The incidence of uric acid stones has notably increased in patients with metabolic syndrome, and most patients with uric acid stones also exhibit some degree of metabolic syndrome, impaired glucose tolerance, or overt diabetes [4][15][19]. Aciduria remains the most critical factor in the formation of uric acid stones, with both diet-dependent and diet-independent causes, such as metabolic syndrome, contributing to a net acid load. Additionally, reduced hepatic ammonia synthesis plays a significant role in this process, leading to increased net acid excretion, as ammonia is a key urinary buffer [20][21]. Uric acid exhibits much lower solubility in urine with a pH of 5.5 or lower, while its solubility significantly increases at higher pH levels, particularly at pH 6.5 or more [20]. Diurnal variations also influence uric acid crystallization, as the lowest urine production and lowest urinary pH occur in the early morning, making this period the most favorable for uric acid crystal formation [10].

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Uric Acid and Gout

Gout and hyperuricemia are associated with uric acid uropathy in 15% to 25% of patients [22][23][24][25]. Under normal physiological conditions, uric acid is secreted 70% by the kidneys and 30% by the intestine, but this proportion can change with renal insufficiency [7]. Of note, in addition to uric acid nephrolithiasis, uric acid itself is nephrotoxic. It can cause acute kidney injury by stimulating renal vasoconstriction, increasing inflammatory mediators, injuring renal microvasculature, and impairing renal autoregulation [26].

Epidemiology

Uric acid stones account for approximately 10% of all urinary stones in the United States and between 5% and 40% of cases globally [27][28]. In the United States, the overall lifetime risk for urinary stones is 10.6% in men and 7.6% in women. Most untreated patients suffer from recurrent abdominal pain, urinary tract infections, and progressive kidney dysfunction, eventually culminating in renal failure. Despite advancements in treatment and patient care, the annual economic burden of nephrolithiasis increased from $1.3 billion in 1994 to $2 billion in 2000 [9]. Uric acid calculi represent 10% to 15% of all urinary tract stones, predominantly affecting men, especially those between 60 and 65 years of age [29]. In Western countries, uric acid calculi are often associated with obesity, hyperglycemia, metabolic syndrome, and hypertension [30]. The prevalence of uric acid uropathy varies according to age, gender, and environmental factors. For instance, individuals over 65 years old are twice as likely to develop uric acid stones compared to younger populations. Traditionally, men have been affected up to three times more than women, though this pattern is changing as more women are diagnosed with the condition [31][32]. Patients with uric acid stones typically exhibit a higher risk of recurrence and related surgical interventions compared to those with calcium urolithiasis [33]. The incidence of uric acid nephrolithiasis is notably high among specific ethnic groups. For instance, approximately 50% of Hmong individuals (from Laos and Thailand) are affected by uric acid stones, whereas only 10% of non-Hmong individuals from the same regions develop them [34]. In some Middle Eastern regions, uric acid comprises a third of all urinary calculi [35]. In most Asian countries, the prevalence of uric acid stones is exceedingly low, with reported incidences under 1% [36]. However, Okinawa is a notable exception, where uric acid stones account for 15% of all urinary calculi [37]. These disparities are attributed to a combination of genetic, dietary, and environmental factors. Environmental elements also significantly impact the frequency of uric acid disorders and stone formation, with the condition being more common in hot, arid climates. A study revealed that the prevalence of nephrolithiasis was 9% among factory workers in high-temperature environments, in contrast to just 0.9% among those working in standard room temperatures [38][34].

Pathophysiology

The formation of uric acid stones is influenced by several factors, including consistently low urinary pH, hypovolemia, and hyperuricosuria (defined as a 24-hour urinary uric acid excretion exceeding 750 mg/day in females and 800 mg/day in males) [37][39]. These values are based on statistical analyses of numerous 24-hour urine tests from individuals without nephrolithiasis and not derived from supersaturation ratios, crystallization rates, or other sources. Clinically, a serum urinary uric acid level of 800 mg/day is generally considered acceptable in patients who do not exhibit aciduria or hyperuricemia, while an optimal level for those undergoing treatment for elevated uric acid is below 600 mg/day [40].

Decreased Urinary pH

Aciduria is observed in nearly all individuals with uric acid nephrolithiasis [41][42]. A decrease in urine pH promotes the formation of uric acid stones by altering the dissolution of uric acid [43][44]. Uric acid solubility is heavily dependent on pH; while aciduria with a pH lower than 5.5 is insufficient to induce uric acid stones, formation is unlikely when urine pH exceeds 6.5 [7]. Aciduria typically results from reduced ammonia production, as ammonia is the principal urinary buffer [45]. In the proximal convoluted tubule, insulin stimulates glutamine metabolism, generating ammonia (NH3), which subsequently forms ammonium ions (NH4+) in the presence of free hydrogen. Metabolic syndrome, impaired glucose metabolism, and diabetes contribute to proximal renal tubular steatosis, lipotoxicity, and insulin resistance,

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which in turn causes aciduria. One mechanism by which this occurs is through steatosis in the proximal tubule cells, which inhibits the Na/H exchanger 3 (NHE3), a key regulator of ammonium excretion [21][46][47][48]. Insulin resistance at the NHE3 receptor may further contribute to defective NH4+ secretion [12]. A study comparing patients with known uric acid stones to a control group of individuals without uric acid stones revealed that, at all body mass index (BMI) levels, those with uric acid stones exhibited more acidic urine and reduced ammonia/net acid excretion, even when both groups followed the same diet [49].

Decreased Urine Output

A reduction in urine output leads to the concentration of urinary solutes and supersaturation, which can precipitate uric acid crystals [50][51]. The most common cause of hypovolemia is dehydration, but gastrointestinal conditions such as Crohn's disease, irritable bowel syndrome, and ileostomies can also induce hypovolemia. These conditions may be accompanied by the loss of bicarbonate through diarrhea and hypocitraturia, which further contribute to metabolic acidosis and aciduria [45].

Hyperuricosuria

The renal glomerulus filters uric acid almost entirely, and it is mostly reabsorbed in the proximal convoluted tubule. Approximately 10% of the filtered uric acid is excreted in the urine. Elevated uric acid levels in the urine typically result from excessive purine intake, especially from red meats, organ meats, certain fish, and alcohol [7]. More recently, diets high in salt and fructose have been associated with increased aldose reductase activity, leading to heightened intracellular uric acid production [52]. Rare genetic disorders can also result in elevated urinary uric acid levels. Lesch-Nyan syndrome, a rare X-linked condition, is characterized by choreoathetosis, developmental motor delays, dystonia, and self-mutilation. It arises from mutations in the hypoxanthine-guanine phosphoribosyltransferase (HGPRT) pathway, which leads to excessive purine degradation and increased uric acid levels in both serum and urine [53]. Congenital renal hypouricemic hyperuricosuria is another genetic condition caused by mutations in the URAT1 channel [54][55][56].

Crystallization Inhibitors

Urine contains several components that suppress urate crystallization and thereby prevent stone formation. The most notable of these is citrate, which elevates urinary pH; hypocitraturia is commonly observed in nephrolithiasis [57][58]. Certain urinary macromolecules are particularly effective in inhibiting crystal growth, including uromodulin (Tamm-Horsfall protein), urinary prothrombin fragment 1, and chondroitin sulfate [58].

Histopathology

A study examined papillary biopsies from 23 patients with uric acid stones, comparing them to control patients and those with calcium oxalate stones. The biopsies from the uric acid cohort exhibited intraluminal crystals, with the majority also presenting varying degrees of calcium oxalate. In comparison to patients with calcium oxalate stones, individuals with uric acid stones exhibited reduced interstitial inflammation and less basement membrane plaque [59].

History and Physical

History

Patients diagnosed with uric acid nephrolithiasis frequently display associations with obesity, diabetes, impaired glucose tolerance, or metabolic syndrome, and, in certain cases, underlying gastrointestinal conditions such as irritable bowel syndrome or Crohn's disease. The typical clinical presentation often includes severe, colicky abdominal, back, or groin pain, with radiating discomfort. Additionally, patients commonly present with gross hematuria, dark or brownish urine, and dysuria.

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Physical Examination

Physical examination findings in individuals with uric acid nephrolithiasis may include costovertebral tenderness or symptoms indicative of gout, such as tophi, podagra, or painful joint swelling accompanied by redness. Assessing flank tenderness and evaluating other indicators of acute kidney injury can assist in establishing a diagnosis.

Evaluation

Laboratory

Urine dipstick testing typically reveals acidic urine and hematuria. Urine microscopy may identify uric acid crystals, which are typically rhomboid or rosette-shaped and are generally yellow or reddish-brown. A 24-hour urine collection should be conducted to assess the uric acid level, urine pH, citrate excretion, creatinine, and volume. A urine pH below 5.5 is commonly observed in uric acid nephrolithiasis. Additionally, serum uric acid levels and a basic metabolic panel should be evaluated [60].

Plain Abdominal X-ray

A kidney, ureter, and bladder (KUB) X-ray is commonly utilized as an initial diagnostic tool for abdominal pain, particularly to exclude peritonitis. Uric acid stones are radiolucent, in contrast to calcium-based stones, which are radioopaque.

Ultrasonography

Ultrasonography is an easily accessible imaging modality that does not involve harmful ionizing radiation and can be performed at the bedside. It is particularly useful for diagnosing kidney stones once they reach a certain size (greater than 0.4 cm) and for identifying related findings such as hydronephrosis, changes in renal echogenicity, and other abnormalities in renal structures. However, ultrasonography cannot differentiate hydronephrosis from ureteropelvic junction obstruction or the benign extrarenal pelvis, nor can it identify ureteral stones or distinguish between uric acid and calcium stones. This imaging technique can also be employed to measure the resistive index, which is elevated in cases of obstructive uropathy on the affected side [61]. The overall sensitivity and specificity of ultrasonography in detecting kidney stones are reported to be 45% and 88%, respectively [62]. When a CT scan is unavailable, the combination of KUB and renal ultrasound has been referred to as a "poor man's CT scan" due to its diagnostic utility in renal stone disease.

Noncontrast Computed Tomography

Noncontrast-enhanced computed tomography (CT) of the kidney is the preferred imaging modality for diagnosing nephrolithiasis. It is more precise and effective than KUB or ultrasound, providing detailed information about the size, density, and location of kidney or ureteral stones. Additionally, it can reveal associated deformities even in the absence of uric acid stones [63][64]. Pure uric acid stones typically present with CT measurements of approximately 500 Hounsfield units, whereas calcium stones measure around 900 Hounsfield units [65]. In patients with kidney stones and a urinary pH of 5.5 or lower, stones measuring 500 Hounsfield units or less can reliably be diagnosed as uric acid stones [65]. Noncontrast CT is preferred because contrast agents may obscure stone visualization.

Treatment / Management

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Management of uric acid kidney stones includes lifestyle modifications, medical treatments aimed at reducing uric acid production and excretion, and urinary alkalinization [22]. Among these, urinary alkalinization is considered the most effective therapy, with a target urine pH of 6 to 6.5. Renal ultrasonography can be used for monitoring uric acid kidney stones, as these stones are not visible on KUB X-rays.

Dietary Interventions

Adequate hydration, with a daily intake of at least 2 liters of water to increase urine volume, is essential for the management of nephrolithiasis [40]. A diet low in purine-rich foods and animal proteins is also recommended to reduce uric acid production in patients with uric acid nephrolithiasis. Although orange juice and lemonade may be beneficial, the fructose content of these beverages can contribute to the formation of uric acid stones, necessitating careful consideration. In addition, recommendations to reduce obesity and manage hypertension and hyperglycemia are essential in minimizing the burden of uric acid kidney stones (A1).

Medical Management [14]

Urinary alkalinization remains the cornerstone of treatment for uric acid stones, with the goal of maintaining a urinary pH consistently above 6.5. Both ultrasound and CT scans can be employed to monitor treatment response. Potassium citrate is typically the preferred agent, although sodium citrate and sodium bicarbonate may also be used. However, sodium-based alkalinizing agents can increase urinary calcium excretion, which may promote the formation of calcium-based nephrolithiasis [67]. Common doses for potassium citrate include 15 to 30 mEq, administered 2 or 3 times daily. Sodium bicarbonate is typically prescribed at 500 to 1000 mg, 3 times daily, while acetazolamide (500 mg daily) may be used to increase urinary pH by inhibiting bicarbonate absorption, although it can reduce citrate excretion and cause volume depletion (A1). Xanthine oxidase inhibitors are recommended for patients with hyperuricemia or hyperuricosuria. These agents should be adjusted to achieve optimal serum uric acid levels (6 mg/dL or less) and urinary levels (600 mg/d or less). Allopurinol is generally prescribed in doses ranging from 100 to 300 mg daily, with the typical dose being 300 mg. Febuxostat, administered at 40 to 80 mg daily, is typically used when patients do not tolerate allopurinol. However, its use was restricted in 2019 by the FDA due to concerns about an increased risk of cardiovascular death compared to allopurinol [68][69]. For further details, refer to related StatPearls articles on hyperuricemia, allopurinol, and febuxostat [70][71][72].

As previously mentioned, humans and primates are among the few mammals without endogenous uricase, which converts uric acid to allantoin, an inert molecule. In cases of hyperuricemia-induced stones, recombinant uricase formulations (including pegloticase and rasburicase) are employed when urate-lowering therapies do not achieve the desired reduction in uric acid levels. Although highly effective, recombinant uricase is typically considered second-line therapy due to cost, the need for intravenous infusion, concerns about infusion reactions, and potential cardiovascular side effects. The 2020 American College of Rheumatology guidelines recommend against using recombinant uricase as first-line therapy [73]. Pegloticase, derived from porcine uricase, was FDA-approved in 2010 for the treatment of refractory gout. It requires infusions lasting at least 2 hours every 2 weeks, with an average of 4 treatments and a median therapy duration of 3 months. Clinical trials have reported infusion reactions in 6.7% of patients, with 0.4% experiencing anaphylactoid reactions. Patients who do not achieve lower uric acid levels after pegloticase treatment often develop high titers of anti-pegloticase antibodies. Rasburicase, also a recombinant uricase, is FDA-approved to prevent hyperuricemia associated with tumor lysis syndrome. It is prescribed as daily infusions for 5 days, although it carries an FDA warning for anaphylaxis, methemoglobinemia, and hemolysis. The risk of anaphylaxis increases significantly after the initial dose. Rasburicase is contraindicated during pregnancy and in patients with glucose-6-phosphate dehydrogenase deficiency (G6PD), and testing for G6PD deficiency is advised for those at high risk [74].

Medical Expulsive Therapy

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Medical expulsive therapy is often utilized for smaller stones, with selective alpha-blockers such as tamsulosin demonstrating efficacy in facilitating spontaneous stone passage. This approach has been shown to increase the likelihood of spontaneous stone passage by approximately 30% for stones located in the distal ureter [75][76] (A1). A novel experimental treatment using theobromine, an alkaloid compound found in cocoa beans and dark chocolate, has been described. Theobromine inhibits uric acid crystallization and has also been employed clinically for the treatment of hypertension [77][78].

Surgical Management

While medical management is preferred for uric acid kidney stones due to the inherent solubility of these stones in alkaline environments, surgical intervention is required in cases of treatment failure or in the presence of severe urinary tract infections. A urinary tract infection complicated by a blocked kidney requires urgent surgical intervention, which may involve emergent drainage through a double J stent or percutaneous nephrostomy [79][80]. Extracorporeal shock wave lithotripsy is effective for smaller calcific and radio-opaque kidney stones (less than 2 cm) but may require contrast (either intravenous or retrograde ureter catheter injection) for the visibility of uric acid stones. Ureteroscopic stone fragmentation and retrieval is an option for stones less than 2 cm in size. Percutaneous nephrolithotomy is typically reserved for larger stones greater than 2 to 2.5 cm in size [81][82].

Diagnosis, Prognosis, Complications, and Patient Education

Uric acid nephrolithiasis often presents symptoms similar to those of calcium-based nephrolithiasis, making it important to consider a differential diagnosis. Other conditions that can mimic the clinical presentation of uric acid nephrolithiasis include acute appendicitis, acute cholecystitis, pyelonephritis, biliary colic, constipation, ectopic pregnancy, hydronephrosis, intestinal obstruction, and pelvic inflammatory disease. These conditions must be ruled out to confirm the diagnosis and determine appropriate treatment. There are several ongoing clinical trials in the United States that aim to deepen the understanding of uric acid nephrolithiasis. These studies include investigations into the renal uptake of fatty acids in patients with idiopathic uric acid nephrolithiasis, as well as exploring the pathophysiology and pathogenesis of the condition. Some trials are also evaluating the role of pioglitazone and weight loss in the development of uric acid stones. The outcomes of these studies could provide valuable insights into better treatment strategies and preventive measures for this condition.

The use of alkalinizing agents, such as sodium bicarbonate, can effectively manage uric acid nephrolithiasis but comes with potential risks. Sodium bicarbonate can increase sodium levels in the body, leading to fluid overload, especially in patients with conditions like high blood pressure, congestive cardiac failure, or liver cirrhosis. Furthermore, high sodium levels can exacerbate the formation of calcium oxalate calculi by promoting the excretion of calcium and sodium. To mitigate these risks, acetazolamide can be used alongside sodium bicarbonate to help improve urinary alkalinization. Additionally, potassium citrate is commonly used for urinary alkalinization, although it must be used with caution in patients prone to hyperkalemia, such as those with chronic kidney disease. For patients without insurance, alternatives like LithoLyte, a citrate supplement and urinary alkalinizer, may be considered. The prognosis for uric acid nephrolithiasis is generally favorable when appropriate treatment is followed. Medical treatment for the dissolution of existing uric acid stones has been proven to be effective, and recurrence can usually be prevented through proper dietary habits, avoiding dehydration, and the correct use of alkalinizing agents. Patients should also manage elevated serum and urinary uric acid levels to reduce the risk of stone formation. Regular monitoring and adherence to treatment are essential to prevent complications. However, uric acid nephrolithiasis can lead to complications such as urinary tract obstruction, which can result in renal failure and sepsis if not properly managed. Treatment methods like extracorporeal shock wave lithotripsy and ureteroscopy may also present risks, including the need for retreatment, urinary tract infections, hematoma formation, and sepsis. In some cases, percutaneous nephrolithotomy may be necessary, but it carries the risks of sepsis, hematuria, retroperitoneal hematoma, blood loss, and the need for arterial embolization to control excessive bleeding.

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Management of uric acid nephrolithiasis requires multidisciplinary collaboration. Nephrologists play a critical role in determining appropriate treatment and prevention strategies, while urologists may be consulted for cases involving large stones or hydronephrosis. Persistent elevation of serum uric acid levels may also warrant a referral to a rheumatologist for further evaluation. This coordinated approach ensures that the patient receives comprehensive care, optimizing outcomes and minimizing complications. Patient education is a vital component of managing uric acid nephrolithiasis. Patients should be educated on the importance of maintaining adequate fluid intake and adhering to dietary recommendations to control uric acid levels. They should also be instructed on the proper use of medications, including alkalinizing agents, to manage and prevent stone formation. By following these recommendations, patients can reduce their risk of recurrent nephrolithiasis and improve their overall health. In conclusion, uric acid nephrolithiasis is a condition that, when diagnosed and treated appropriately, generally has a favorable prognosis. Early detection, proper medical treatment, and lifestyle modifications can prevent recurrence and reduce the risk of complications. The involvement of a multidisciplinary healthcare team is essential to providing comprehensive care, ensuring the best possible outcomes for patients.

Conclusion

Uric acid nephrolithiasis remains a prevalent and significant condition with a growing incidence globally, particularly in developed nations where metabolic syndrome, obesity, and dietary habits are closely linked to the formation of these stones. The pathophysiology of uric acid stones is complex, involving factors such as low urinary pH, dehydration, hyperuricosuria, and genetic predispositions. The clinical management of uric acid nephrolithiasis primarily focuses on increasing urinary pH, improving hydration, and addressing hyperuricosuria, which can lead to the dissolution of the stones in many cases. Dietary modifications, particularly reducing the intake of high-purine foods, are crucial in managing hyperuricosuria and minimizing the formation of uric acid stones. In addition, the role of medications, such as xanthine oxidase inhibitors, can help to reduce uric acid production, offering therapeutic benefits for patients at risk or already suffering from these stones. Importantly, advances in understanding the mechanisms of crystal formation, as well as the identification of crystallization inhibitors in urine, provide new avenues for future treatment strategies and prevention of recurrence. The epidemiological data indicates a diverse distribution of uric acid stones across different populations, with certain ethnic groups and environmental factors, such as hot climates, playing a significant role in disease prevalence. Furthermore, the role of uric acid in comorbid conditions like gout, renal impairment, and hypertension underscores the importance of early detection and preventive measures. In conclusion, uric acid nephrolithiasis represents a multifactorial condition that requires a multifaceted approach to management, including dietary control, pharmacological intervention, and attention to underlying health conditions. Continued research into the genetic and molecular pathways contributing to this condition will provide valuable insights into more effective prevention and treatment options. As the prevalence of metabolic disorders increases globally, the impact of uric acid nephrolithiasis will likely continue to rise, emphasizing the need for better preventive strategies and tailored therapeutic approaches.

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حصى حمض اليوريك: مراجعة محدثة

الملخص:

الخلفية: حصى حمض اليوريك هي حالة شائعة تتميز بتكون حصى الكلى نتيجة ترسب بلورات حمض اليوريك. غالبًا ما يرتبط هذا الاضطراب بفرط حمض اليوريك في الدم والبول الحمضي، مع وجود عدة عوامل خطر تشمل السمنة، داء السكري، ارتفاع ضغط الدم، ومتلازمة الأيض. تمثل حصى حمض اليوريك حوالي 5-40% من حصوات المسالك البولية على مستوى العالم، وقد تسببت تاريخيًا في آلام شديدة وتتطلب علاجات متقدمة.

الهدف: تهدف هذه المراجعة إلى تحديث الفهم حول حصى حمض اليوريك، مع التركيز على مسبباتها، وبائيتها، وعلم وظائف الأعضاء، واستراتيجيات العلاج الحالية.

الطرق: تقوم المراجعة بتلخيص البيانات من العديد من الدراسات حول التكوين الكيميائي الحيوي، وعوامل الخطر، وإدارة حصى حمض اليوريك. وتسلط الضوء على دور درجة حموضة البول، وفرط اليوريسوريا، وانخفاض حجم البول، والاستعدادات الوراثية في تكوّن الحصوات. كما يتم مناقشة تأثير النظام الغذائي، والاضطرابات الوراثية، والعوامل البيئية .

النتائج: تتكون حصى حمض اليوريك بشكل رئيسي في ظل ظروف حموضة البول المنخفضة، مما يعيق ذوبان حمض اليوريك. تزيد الحميات الغذائية الغنية بالبيورينات، والجفاف، والاضطرابات الأيضية بشكل كبير من خطر تكوّن الحصوات. تشمل عوامل الخطر الرئيسية فرط اليوريسوريا، السمنة، والبول الحمضي. تسمح التطورات في العلاج الآن بحل العديد من حصوات حمض اليوريك عن طريق زيادة درجة حموضة البول وحجمه مع إدارة فرط اليوريسوريا. ومع ذلك، لا يزال تكرار الإصابة أمرًا شائعًا، وترتبط الحالة بتكاليف صحية مرتفعة.

الخاتمة: لا يزال حصى حمض اليوريك يشكل تحديًا سريريًا كبيرًا مع مسببات وعوامل خطر متنوعة. يعد التشخيص المبكر والتدخل، بما في ذلك التعديلات الغذائية، والترطيب الكافي، والإدارة الطبية لزيادة درجة حموضة البول وتقليل مستويات حمض اليوريك، أمرًا بالغ الأهمية في الوقاية من التكرار. إن البحث المستمر في الآليات الجزيئية لتكوين الحصوات والعلاجات الجديدة أمر أساسي لتحسين نتائج المرضى.

الكلمات المفتاحية: حصى حمض اليوريك، حصوات الكلى، فرط حمض اليوريك في الدم، درجة حموضة البول، متلازمة الأيض، فرط اليوريسوريا، الوقاية من الحصوات.

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