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REVIEW ARTICLE

Pathophysiology associated with forming urinary stones

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Abstract Urolithiasis has become a chronic disease that has had a major impact on both the quality of life and working situation of the patient. It has a significant impact on the health system due to its high recurrence. Different authors have identified several factors inherent to human biology and sociodemographic variables that may lead to the development of kidney stones. Thus, in this review, the main factors that influence the formation of kidney stones are presented, and that may help in a timely intervention on some of them.

PALABRAS CLAVE
Urolitiasis;
Factores sociodemográficos;
Factores de riesgo

Fisiopatología asociada a la formación de cálculos en la vía urinaria

Resumen La urolitiasis se ha convertido en una enfermedad crónica que ha tenido un gran impacto en la calidad de vida y en la situación laboral de quien la padece; su tasa de prevalencia y recurrencia es cada vez mayor, lo que genera un gran impacto socioeconómico en cualquier país al afectar el sistema de salud. Se han identificado numerosos factores inherentes a la biología humana y algunas variables sociodemográficas, que favorecen el desarrollo de cálculos renales; por lo cual, en esta revisión se describen los principales factores que influyen en la formación de urolitiasis, permitiendo intervenir oportunamente sobre algunos de ellos.

Introduction

At present, calculi in the urinary tract can be considered as a chronic disease with a major impact on the quality of life.[1] In the United States, prevalence of urolithiasis is 8.8%

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for general population, being higher in men than in women (10.6 and 7.1%, respectively).[2]

Approximately 13 of every 1000 hospital admissions are due to renal and ureteral lithiasis; it is a frequent cause of admission in the emergency department and it can trigger various complications.[3]

Its prevalence is increasing and it depends on diverse factors, resulting in an economic burden on the health system. Costs increase due to frequent consultations, diagnostic tests, prolonged hospital stays, surgical procedures, broad-spectrum antibiotics or extended regimens, and secondary hospitalizations due to complications triggered by this disease. Currently in the United States, where there are available studies on the costs, urolithiasis costs more than five billion dollars annually, including diagnosis, treatment and disability caused by the disease.[4,5]

This demonstrates the implication of this disease for the health system, given that approximately 77% of the individuals affected by urolithiasis are included in the productive groups of the population in any country.[6] However, these numbers may vary due to different factors such as gender, age, history of urinary tract infections, disorders of metabolic origin, dietary excess and/or deficiency, among others.[3] This paper aims to describe some of the theoretical concepts associated with stone formation in the urinary tract.

Methodology

A literature search was conducted through Medline via Ovid, SciELO, and Embase from 1986 to 2014; with the following Keywords: ''urolithiasis and climate,'' ''lifestyles and urolithiasis,'' ''socio-demographic factors and urolithiasis,'' ''gender and urolithiasis,'' ''age and urolithiasis,'' ''race and urolithiasis,'' ''genetics and urolithiasis,'' ''anatomical abnormalities and urolithiasis,'' ''diet and urolithiasis,'' ''stress and urolithiasis,'' ''hormones and urolithiasis;'' the different factors were also combined with ''kidney stones'', and the search was conducted both in Spanish and English. It is a narrative review, instead of being a systematic review/meta-analysis.

Epidemiology

It is the third most common urological disease after urinary tract infection and prostate disease. Its prevalence varies between 2 and 20% worldwide and it appears to relate to the geographical and socioeconomic characteristics of different populations.[7] Its incidence has considerably increased in recent decades in all industrialized countries, due to profound modifications in dietary habits and lifestyle, characterized by a high calorie intake, combined with reduced physical activity, among other risk factors.[6]

Urolithiasis has a high recurrence rate after the first episode (50%)[8]; 14% have recurrence within a year, 35% at 5 years, and 52% in 10 years.[3] This risk appears to be greater in the western hemisphere, with reported prevalences of 9.5% in Europe, 12% in Canada, 13–15% in the USA, and 5.1% in the eastern hemisphere. However, the highest risk has occurred in some countries like Saudi Arabia (20.1%).[9]

The most prevalent stones are calcium (60–65%), including calcium oxalate, calcium phosphate, and others, followed by struvite (5–15%) and uric acid (5–10%); cystine stones are uncommon and they account for 1–3% of all calculi types.[10,11]

Pathophysiology

The sequence of events in the formation of any urinary calculus includes: urinary saturation, supersaturation, nucleation, crystal growth, aggregation of crystals, crystal retention, and, finally, calculus formation. Normally, these crystals pass through the urinary tract without problem; however, occasionally, if they become very large, they may cause the obstruction of the kidney drainage system resulting in severe pain, bleeding, infection or kidney failure, sending the patient to an emergency room.[12]

There are different theories about the process of stone formation. One proposes that the stone is formed when a normally soluble salt (e.g. calcium oxalate) supersaturates the urine, and crystals begin to form; if they are sufficiently large, they can get fixed to the urothelium (usually in the terminal portion of the collecting ducts), to then grow slowly. Another theory assumes stone formation begins in the medullary interstitium, then Randall plates form in the papilla, on which oxalate or calcium phosphate crystals start to deposit.[7,13]

During the passing of urine by the kidney, such large particles can be formed that they can be retained; these serve as a nucleus for the formation of future stones.[7]

A solution favoring the development of urolithiasis is considered saturated regarding a substance when it contains in dissolution the highest possible concentration, that is, if an additional amount of this substance is added to this solution, it precipitates and forms crystals; the concentration at which this saturation is reached and crystallization begins is called thermodynamic solubility product (Ksp).[9,12]

In clinical practice, supersaturation can be the result of any increase in urinary excretion of solvents (for example, calcium, oxalates, and cystine) or reduction in urine volume due to a decrease in fluid intake or extrarenal fluid loss.[10]

Other mechanisms by which crystals remain in the kidney:

Calcium oxalate monohydrate crystals (COM) connect rapidly with the surface of renal epithelial cells because the surface of these crystals behaves as if positively charged, while the lumenal surface of epithelial cells of the tubules behaves as if negatively charged; thus this adherence is due to reactions of electrical charge, which make the crystal behave as if positively charge, and link to electrically negative molecules that emerge from the apical surface of the tubular cell; in a study, Lieske et al. concluded that immediately after adhesion, anchored crystals can serve as a preferential site for biding additional crystals; later, the crystal is endocytosed by the tubular cell, where after internalization the plasma membrane domain covering the crystal appears to exhibit increased adhesiveness for crystals because the attachment of added crystal was higher for at least 24 h after the first crystal bound; therefore, the presence of any attached or internalized COM crystal results in an increased number and/or affinity of crystal adhesion sites on the cell surface.[12,14,15]