AUCTORES
Globalize your Research

International Journal of BioMed Research

Maciej Pawlikowski *

Open Access

Research Article

Biomineralogy of Kidney Stones Crystallization

Maciej Pawlikowski[1*]

[1] AGH – University of Science and Technology, Cath. Mineralogy, Petrography and Geochemistry, al. Mickiewicza 30, 30-049 Kraków, Poland.

***Corresponding Author:** Maciej Pawlikowski, AGH – University of Science and Technology, Cath. Mineralogy, Petrography and Geochemistry, al. Mickiewicza 30, 30-049 Kraków, Poland.

Received date: January 08, 2022; **Accepted date:** March 01, 2022; **Published date:** April 23, 2022

Citation: Pawlikowski M. (2022) Biomineralogy of Kidney Stones Crystallization. *International J. of Biomed Research. 2(4): DOI:* 10.31579/IJBR-2021/064

Summary

One of the unsolved mysteries is why kidney stones are formed, for example, in one renal pelvis, while other pelvises are not subject to the process of biomineralization, i.e. crystallization on biological media. Stones also crystallize in the ureters and bladder. The study presents details of the crystallization of kidney stones in a place that mineralogy calls the crystallization center. This publication discusses the method of crystallization of stones and their structure.

Keywords: biomineralogy; kidney stones; biomineralization

Introduction

The basis for understanding the issues of the formation of uric stones and the treatment of kidney stones is answering the questions related to the initiation of crystallization in the renal pelvises (1-24). This applies to both the formation of the so-called crystallization centers, in which stone formation is initiated, and the causes of excessive urine mineralization. Both issues are decisive in the development of urolithiasis.
This article presents the results of many years of mineralogical and chemical studies of these phenomena.

Study results

Biomineralogical studies regarding the formation of kidney and bladder stones, recurrent stones and mineralogy of the stones themselves have been conducted at AGH – University of Science and Technology in Krakow – since the 1970s. About 800 stones were examined during this time. The studies prove that urinary stones start to crystallize in crystallization centres. A crystallization center is a place where the surface of the wall of the renal pelvis is damaged (Fig. 1). This damage may be caused by crystals crystallizing from excessively mineralized urine. The edges of these crystals, as they pass through the kidney pelvis with urine, can mechanically damage the pelvic wall, "cutting" the tissues and causing bleeding that appears in the urine. The factor that damages the pelvic wall may also be microorganisms that infect the urinary system (Fig. 1, B.1.). The toxins produced by them in their life processes are aggressive chemicals that can damage the pelvic wall, among other things. There may also be genetic defects in the biological, atomic structure of the pelvic wall, passed down through generations.

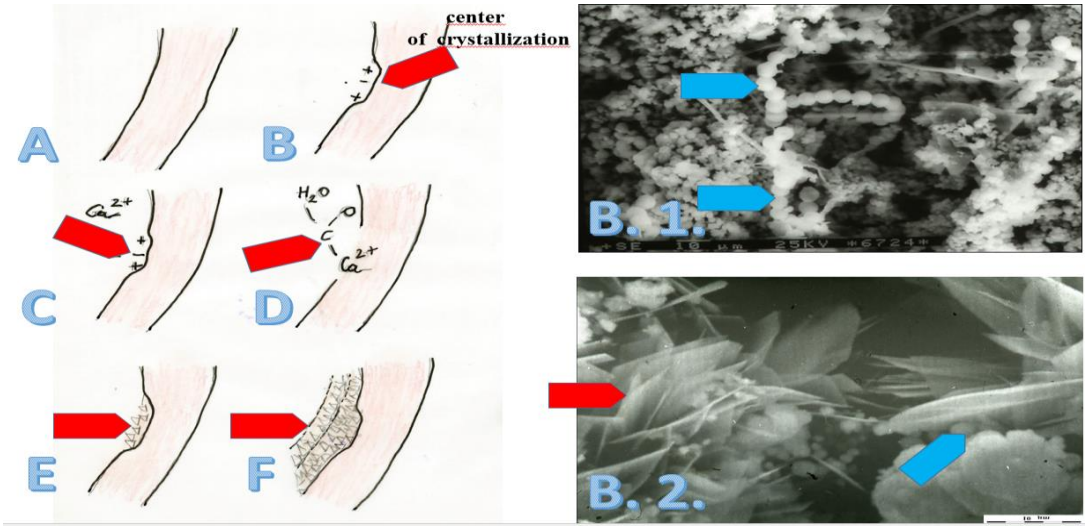

Figure. 1. *Diagram of the development of a staghorn type kidney stone in a renal pelvis. A – image of the cross-section of smooth, undamaged wall of the renal pelvis. B – cross-section of the wall of a renal pelvis with a damaged area (center of crystallization of the staghorn stone). Due to destruction of interatomic bonds, this area has free charged bonds (+, - Fig. 1, C), where e.g. calcium cations from the urine can attach and start the crystallization of stones. B.1. – a colony of bacteria (arrows) from the renal pelvis, producing toxins in their life processes that destroy the wall of the renal pelvis. Scanning microscope. B.2. – initial stage of oxalate crystallization (red arrow) on the wall of the renal pelvis with bacteria presence (blue arrow). Scanning microscope. D – next phase of oxalate crystallization on the wall of the renal pelvis. E – the appearance of the first oxalate crystals (whewellite) on the wall of the renal pelvis. F – subsequent stages of oxalate stone growth (arrow).*

All these damages, regardless of their genesis, lead to the occurrence of "broken" interatomic bonds in the crystallization center, in biological structure of the pelvic wall. This leads to the formation of free, electrically charged bonds. These charged bonds "catch" cations and anions from the urine. Upon attachment to the biological structure, the attached ions become initiators of stone crystallization (Figure 1, B.2.). Further attachment of calcium, CO_2 groups, etc. causes further growth of a staghorn stone that "copies" the shape of the renal pelvis (Figure 1, B.–F.).

Development of such mineralization in the crystallization center may be caused by increased urine mineralization, e.g. as a result of a malfunctioning kidney causing excessive re-sorption of water from the urine. The causes of excessive urinary mineralization may also be due to processes outside the kidneys. These include "overproduction" of PTH, i.e. disorders of the parathyroid and thyroid glands. It results in the removal of Ca, but also P from the bone. The excess of Ca^{2+} in the blood plasma may also be the result of diseases, including inflammation, but also neoplastic changes. The effect of excessive pancreatic calcium bicarbonate synthesis is unknown. Their excessive amount that enters the duodenum along with pancreatic juice may, through intestinal absorption, increase the level of ionized calcium in the blood.

The stones growing cyclically in the renal pelvis (Figure 2, A, B) have the structure of wet sand. They can break down quite easily under the influence of natural or artificially induced shocks (lithotripsy). This applies especially to "young" stones, because "old" stones harden when recrystallized, which makes breaking them much more difficult.

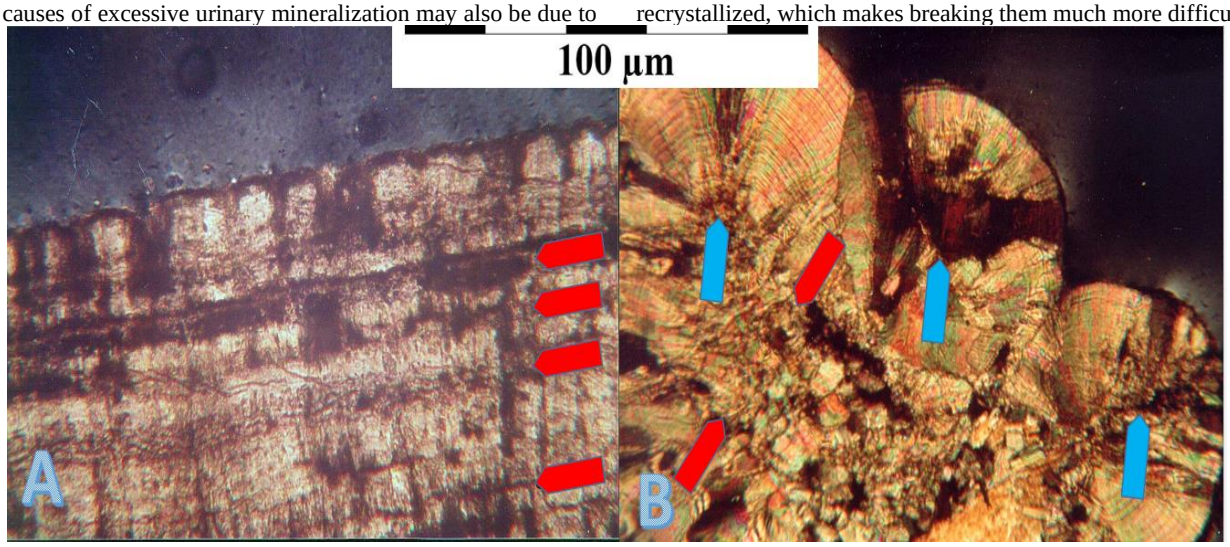

Figure 2. *The growth of staghorn type stones. A – visible cyclicity of oxalate crystallization. Arrows show the crystallization cycles. B – oxalate stone (weddelite). Concretions, aggregate oxalate forms (blue arrows) crystallizing on the crystallization centers of the oxalate layer (red arrows). Polarizing microscope, X polaroids.*

The "kidney sand" left after disintegration or breakdown of stones is flushed out by the urinary system. However, if a grain of "sand" remains in the ureter or bladder (Fig. 3 A., A.1., A.2., A.3., B, C, D), it may form another center of crystallization, where minerals crystallizing from urine (oxalates, phosphates, urates, etc.) keep growing.

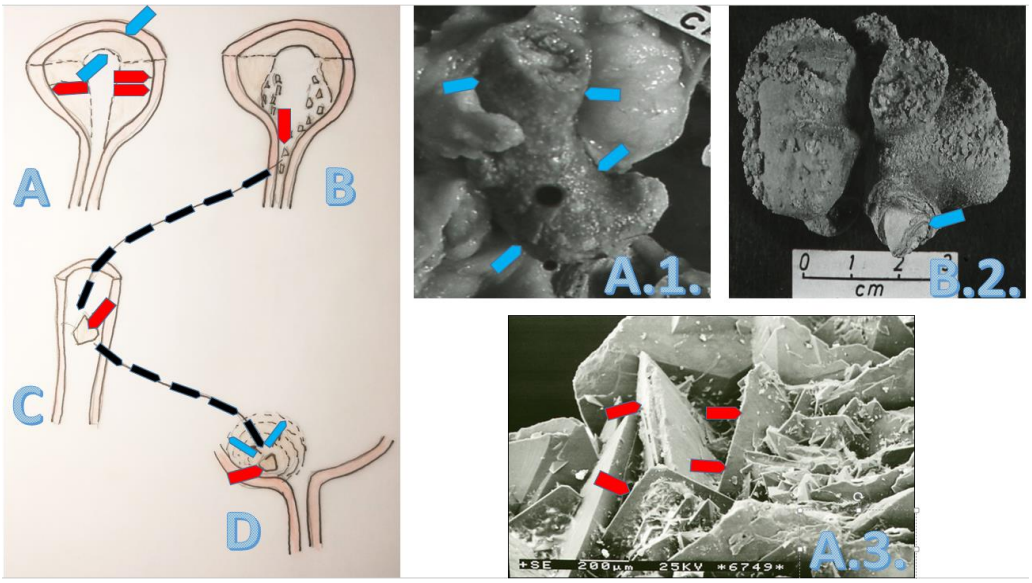

Figure 3. *A – Crystallization of a staghorn type kidney stone in a renal pelvis (cross-section). The points of stone crystallization initiation on the inner walls are shown by the red arrows. Crystallizing and growing stone in subsequent growth stages systematically reduces the "light" of the urine flow, in extreme cases leading to a complete blockage of its flow. A.1. – Stone in the renal pelvis (arrows) completely blocking the outflow of urine. A.2. – Staghorn type whewellite-weddelite stones surgically removed from the kidney. The arrow indicates the apparent cyclicity of oxalate crystallization. A.3. – Structure of whewellite crystals on the surface of a staghorn kidney stone at the site of urine flow. Edges of whevellite crystals can be sharper than a razor blade (arrows). Scanning microscope. B – natural shocks or lithotripsy cause the stone to break down into fine fragments (even single crystals). Some of them can remain as deposits in the renal pelvis, the ureter (Figure 3.B.) or the bladder (Figure 3.D.). If the urine is still over-mineralized, secondary kidney stones can crystallize on these grains*

The stone that forms in the ureter on a grain of kidney sand hinders the flow of urine from the kidney to the bladder (Fig. 4). The stone may, after reaching the appropriate size, close the urine outflow (Figure 5).

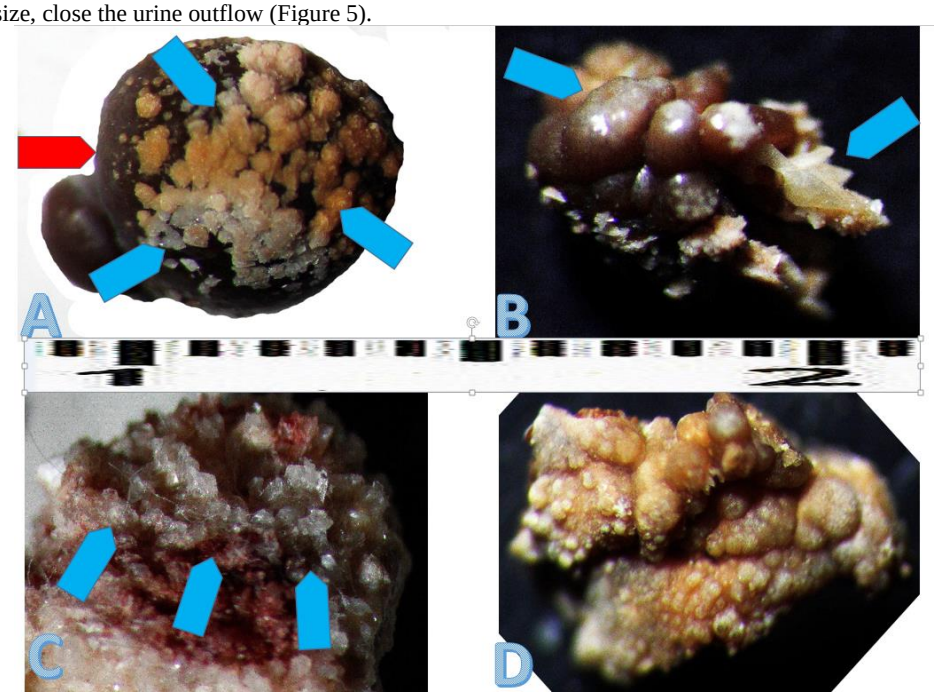

Figure 4. *Stones crystallizing on fragments of a staghorn type stone that formed after its disintegration. A – stone from the renal pelvis. Weddelite crystals (blue arrows) on a brown crumb of a staghorn stone (red arrow). B, C – stones from the urinary tract. Beautiful crystals growing on a fragment of a disintegrated stone of the staghorn type (arrows). D – fragment of a staghorn type stone with bulky, growing oxalate aggregates.*

Disintegration or breakdown of a kidney stone in the renal pelvis is not a guarantee that the problem of recurrent crystallization will be fully solved. Removal of the stones eliminates pain, blockage of urine flow, etc., but to deal with recurrent urolithiasis, liquidation of the crystallization center in the renal pelvis and reduction of excessive urine mineralization must be achieved. The decision how to do that is up to the doctors.

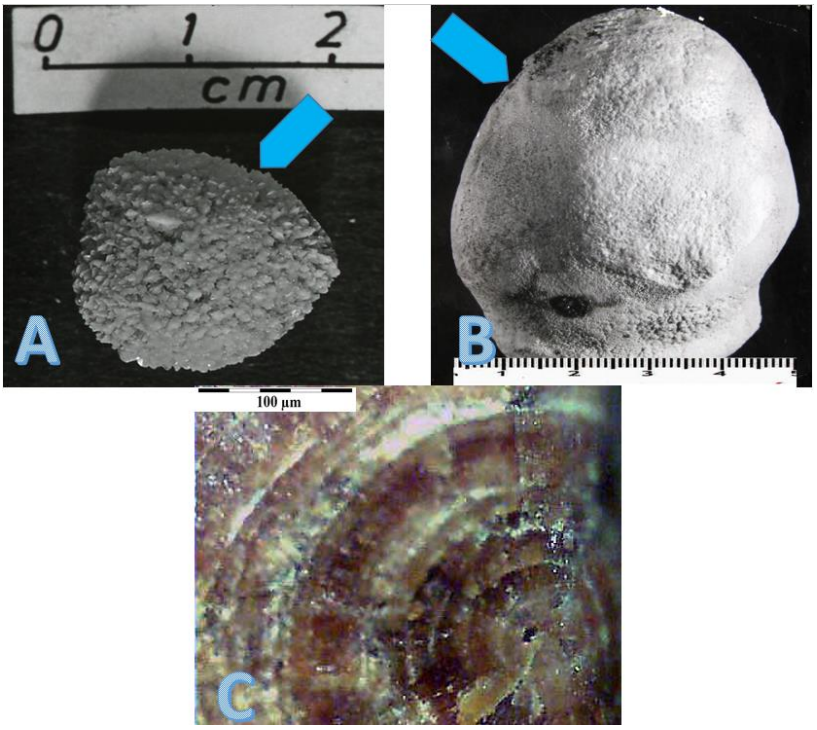

Figure 5. *Stones crystallizing in the bladder. A – stone covered with a crystalline brush of whewellite crystals. B – a large stone that crystallized on a grain that got into the bladder after disintegration of a staghorn stone originally present in the renal pelvis. C – microscopic image of the internal structure of the cyclically crystallizing oxalate stone shown in Figure 5.B.*

Conclusion

Mineralogical studies of kidney stones provide a large amount of information important in their diagnosis and treatment. The obtained results indicate that crystallization of stones in the renal pelvis begins at a site where its wall has been damaged. It may be the result of mechanical damage caused by crystals crystallizing from urine. The damage can also be caused by aggressive compounds formed during infection, produced by various microorganisms that infect the urinary system.

One or more crystallization centers may form and be subsequently mineralized. The place where damage to the pelvic wall forms, i.e. the formation of the crystallization center, is accidental.

Research indicates that there is a need for standard analysis of kidney stones and sharing the results with a urologist. Further research on urolithiasis should focus on experimental dissolution of stones in vitro and on selecting substances that block crystallization centers of stones, which will prevent recurrence of urolithiasis.

A separate scope of research should be diagnosis of non-renal causes of excessive blood and, consequently, urine mineralization, from which kidney stones crystallize.

References

1. Aggarwal K. P., Narula S., Kakkar M., Tandon C. (2013) Nephrolithiasis: molecular mechanism of renal stone formation and the critical role played by modulators. *BioMed Research International.* 21.
2. Badowski J, Kozak J, Socha-Witulska E, Żabiński M, Hokeja J, Łesiów R. (1993) Renografia z testem diuretycznym i badania ultrasonograficzne w uropatii obstrukcyjnej. *Rocz. Med*. 1(2): 499-504
3. Borkowski A., Borówka A., (1994) Nowe metody leczenia kamicy górnych dróg moczowych, *Warszawa*: 41–196.
4. Cakıroğlu B., Eyyupoğlu E., Hazar A. I., Uyanik B. S., Nuhoğlu B. (2016) Metabolic assessment of recurrent and first renal calcium oxalate stone formers. *Archivio Italiano di Urologia e Andrologia.* 88:101–105.
5. Cunningham P., Noble H., Al-Modhefer A.-K., Walsh I. (2016) Kidney stones: pathophysiology, diagnosis and management. *British Journal of Nursing* 25(20):1112–1116.
6. Dawson C. H., Thomson C. R. V., (2012) Kidney stone disease: pathophysiology, investigation and medical treatment. *Journal of Clinical Medicine*. 12(5):467–471
7. Giannossi L., Summa V., (2012) A review of pathological biomineral analysis techniques and classification schemes. In: Aydinalp C., editor. An Introduction to the Study of Mineralogy. In: Tech, IMAA-CNR, Italy.
8. El-Ghar ME, Shokeir AA, El-Diasty TA, Refaie HF, Gad HM, El-Dein AB. (2004) Contrast enhanced spiral computerized tomography in patients with chronic obstructive uropathy and normal serum creatinine: a single session for anatomical and functional assessment. *J Urol.* 172(3): 985-988.
9. Krakós M, Kobos J, Andrzejewska E. (2004) Badanie unerwienia ściany moczowodu w zwężeniu połączenia miedniczkowo-moczowodowego u dzieci z wodonerczem wrodzonym. *Urol. Pol*. 57 (4): 71-76.
10. Kwak C., Kim H. K., Kim E. C., Choi M. S., Kim H. H., (2003) Urinary oxalate levels and the enteric bacterium Oxalobacter

formigenes in patients with calcium oxalate urolithiasis. *European Urology*. 44(4):475–481
11. Kok D. J., Papapoulos S. E., Bijvoet O. L. (1990) Crystal agglomeration is a major element in calcium oxalate urinary stone formation. *Kidney International*. 37(1):51–56
12. Leńko J., (1976) Kamica moczowa, *Warszawa*: 129–141.
13. Pawlikowski M., (1987) Mineralizacja organizmu człowieka żyjącego. (Mineralization of human living organism). *Prace Mineral*. 79: 121.
14. Pawlikowski M. (1993) Kryształy w organizmie człowieka. (Crystals of human organism). Secesja. (Atlas):132.
15. Pawlikowski M., Kwinta A., (2006) Kamica moczowa jako ciągły proces krystalizacji i rozpadu kryształów w świetle badań mineralogicznych, *Urologia Polska* 4.
16. Pawlikowski M, Kwinta A, Zwolinska B, Kwinta K., (2013) Green fiber lasers in urology. *Folia Med*. *Cracov*, 53(2): 23-34.
17. Pawlikowski M., (2016) Biomineralogia kamieni nerkowych i żółciowych (Biomineralogy of kidney stones and gallblader stones) Wyd. Nauk. Akapit, 56 p.
18. Ratkalkar V. N., Kleinman J. G. (2011) Mechanisms of stone formation. *Clinical Reviews in Bone and Mineral Metabolism.* 9(3-4):187–197.
19. Różański W., i in., (2003) Składniki morfotyczne kamieni moczowych – kryształy, „*Urologia Polska*" 1 : 11–16.
20. Sayer J. A., (2008) The Genetics of nephrolithiasis. *Nephron Experimental Nephrology*. 110(2):37–43
21. Schepers M. S., Vander B. G., Romijn J. C., Schröoderand F. H., Verkoelen C. F. (2002) Urinary crystallization inhibitors do not prevent crystal binding. *Journal of Urology* 167(4):1844–1847.
22. Tsujihata M., (2008) Mechanism of calcium oxalate renal stone formation and renal tubular cell injury. *International Journal of Urology* 15(2):115–120.
23. Zieliński J., Leńko J., (1992) Urologia, Warszawa.

Supplemntary Figures: