Scanning Electron Microscopy

Volume 1986 | Number 2 Article 44

5-29-1986

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SCANNING ELECTRON MICROSCOPY/1986/II (Pages 757-760)
SEM Inc., AMF O'Hare (Chicago), IL 60666-0507 USA

0586-5581/86$1.00+05

UROLITHIASIS IN A PATIENT INGESTING PURE SILICA: A SCANNING ELECTRON MICROSCOPY STUDY

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(Received for publication February 20, 1986: revised paper received May 29, 1986)

Abstract

A patient who repeatedly produced urinary calculi, had consumed about 3 g of cristobalite (SiO_2) per day for many years. Investigations using scanning electron microscopy revealed minute particles containing silicon in the core of the stone as well as in urine sediment. A mechanism similar to that proposed for the effect of silicon-containing drugs against gastric ulcer, may play a role in this formation of silicon-containing urinary stones.

KEY WORDS: Urolithiasis, Low-cristobalite, Scanning electron microscopy, Heterogeneous nucleation, Whewellite, Apatite, Drug abuse, Silica stones, X-ray diffractometry Element dispersive x-ray analysis

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Introduction

Urinary calculi consisting of pure SiO_2 are very rare in man, in contrast to the stones of some animal species with high dietary intake of silicates (Farrer and Rajfer, 1984). A mere dozen of cases have been reported in the literature, nearly all referring to an abuse of magnesium trisilicate-containing drugs for prevention of peptic ulcer in male patients. No case of a female patient was known. Alpaugh and Johnson (1984) had reported SiO_2 stones in patients without antacid medication, admitting that artifacts could not be wholly excluded.

Kim et al (1983), however, had found discrete silicon-bearing deposits in 3 out of 180 stones using energy-dispersive x-ray analysis (EDXA) in scanning electron microscopy (SEM). They concluded that siliceous precipitation might be much more common in urinary calculi than currently assumed, even without a history of excess medication.

This paper reports about a patient forming urinary calculi with a minute proportion of siliceous deposits after a long history of regular oral ingestion of pure SiO_2.

Case Report

A 48-year-old female patient had passed two urinary calculi within the last 15 months. No stone episode had been observed in the past 15 years. The physical phase analysis of the first stone had revealed 80% whewellite (Calcium oxalate monohydrate) and 20% apatite (Calcium phosphate). Careful examination for possible metabolic factors leading to stone formation brought no positive result. Dietary and drug anamnesis revealed continuous ingestion of about 3 g of pure silica ("Kieselerde") per day during the last 12 years which was supposed to prevent brittle fingernails and hair.

SEM Investigation

The second stone was prepared for SEM as described by Blaschke and Schmandt (1978) (Fig.1) and investigated with a Cambridge Stereoscan 180 equipped with an EDXA unit PGT, type EPS 1000. Morphologically, the stone appeared as a papillary stone; it consisted of whewellite crystals in radial array around a central core of apatite (Fig.2). The proportions of both components corresponded to the phase composition of the first stone. Within the apatite core, which is often found in oxalate-rich urinary calculi (Leusmann, 1983), we found some discrete particles containing only silicon and magnesium, according to EDXA signals (Figs. 3 and 4).

These disseminated particles were embedded in a matrix of cryptocrystalline apatite, showing neither preferential texture nor focal enrichment. Their diameters ranged from less than 1 to a few microns, their shape was irregular, and no crystal faces were seen.None of these grains were detected in the outer whewellite portion of the stone.

In parallel to these investigations we had examined the "Kieselerde" by x-ray diffractometry, and identified the substance as pure low-cristobalite, a modification of SiO_2 (Fig.5). Further attempts to identify the above described Si/Mg-containing particles by both x-ray diffractometry and electron diffractometry in a Transmission Electron Microscope (TEM) failed. It appears most likely, however, that the corpuscles found in the stone differ chemically from the ingested SiO_2, especially by their high Mg content. In EDXA, the proportion of Si to Mg signals was constant within the particles and also between individual grains. Although the patient had stopped SiO_2 ingestion some 3 months ago, the urine sediment was studied to get additional information. Even though small particles with a high silicon content were detected, no magnesium was present in them (Figs.6 and 7), and no further identification was possible.

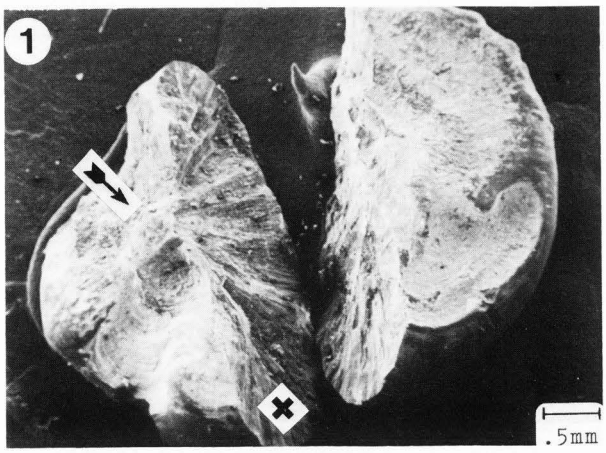
Discussion

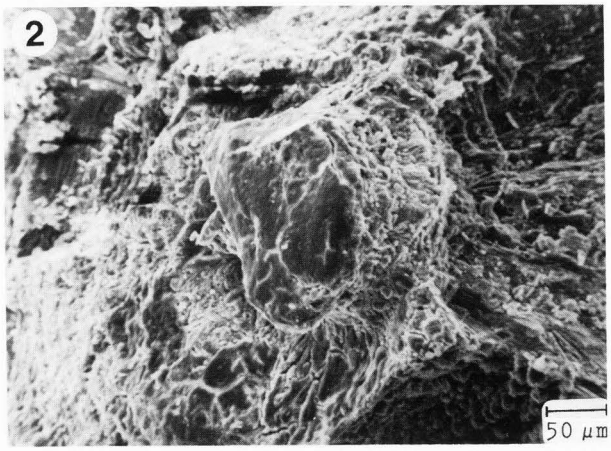
Several questions may be raised from the above findings: (1) Is there any connection between the formation of urinary calculi and the ingestion of SiO_2 in this case, or, in particular, are the Si/Mg-containing particles somehow responsible for stone formation? (2) What is the mode of silicon enrichment in the kidney? and (3) Why do the particles inside the calculi contain additional Mg, in contrast to those in the urine sediment consisting exclusively of silica?
Some answers are proposed in the following as inferences from a single case, however, they cannot be more than hypothetical.

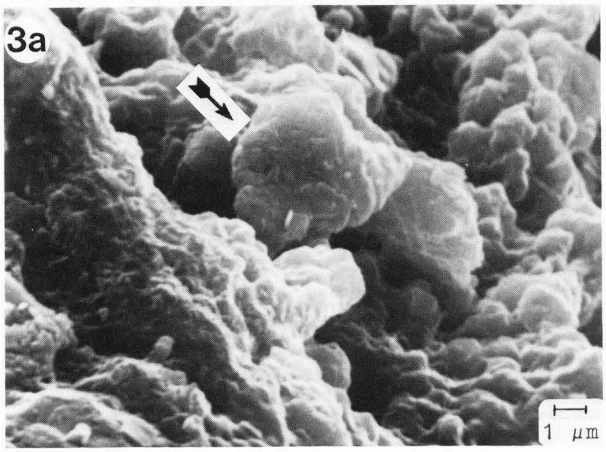
Ad (1): Since there was no metabolic disorder leading to stone formation (such as primary hyperparathyroidism, hypercalciuria or any other supersaturation of the urine with lithogenic ions, renal tubular acidosis, or decreased excretions of stone inhibitors), and since the Si/Mg particles were found only in the central apatite core of the stone, a connection between SiO_2 ingestion and the formation of urinary calculi appears likely and plausible. In particular, the Si/Mg-containing corpuscles could have acted as heterogeneous "nuclei" initiating stone development.
Ad (2): Two models appear possible: a) chemical solution of ingested SiO_2 in the gastrointestinal tract, and precipitation of a siliceous compound in an oversaturated urine, and b) penetration of SiO_2 particles through the intestinal wall into the blood stream, and subsequent excretion with the urine (as called "persorption" by Brosig and Rost, 1977). None of the two models claim definitive priority. The particles found in the stone are chemically different from the ingested low-cristobalite, but this was not ascertained for the particles observed in the urine sediment. Solution experiments with low-cristobalite at 37°C in diluted hydrochloric acid and sodium hydroxide, simulating the pH-conditions in the stomach or small intestine, respectively, revealed no recognizable solution. By way of the regular ingestion of SiO_2 over a long period, a relatively high silicon level in the blood could have developed despite a low solution product. Page et al. (1941) had reported that after oral consumption of Mg-trisilicate, some 5% of the ingested silicon was excreted with the urine. Whether this is valid for low-cristobalite, too, has not been ascertained so far. Ad (3): Attempts to identify the particles by electron diffraction have failed, too, possibly due to the crystallographic amorphism of the corpuscles. This has also been stated in other investigations of pure silica stones (Farrer and Rajfer, 1984). Since amorphous compounds usually are chemically impure, it appears possible that some Mg enrichment of the particles within the stone has taken place during its growth in the kidney. in contrast, SiO_2 particles that are not incorporated in a stone, may be washed out rapidly with the urine before any enrichment with Mg, and so appear as pure silica in the sediment.

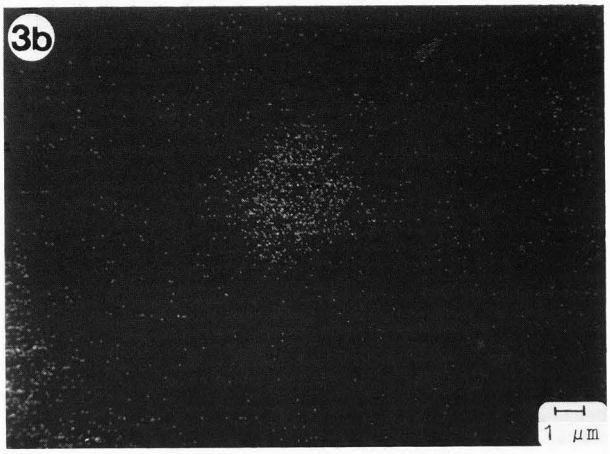
Acknowledgements

This study has been performed at the "Institut für Medizinische Physik" (Director Prof. Dr. G. Pfefferkorn); the authors would like to thank all persons who helped to make this investigation possible.









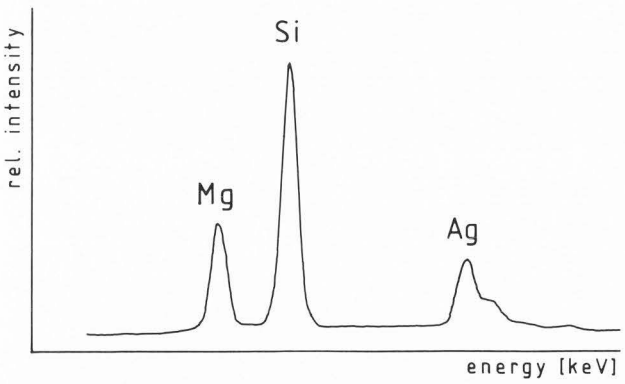


Fig.4

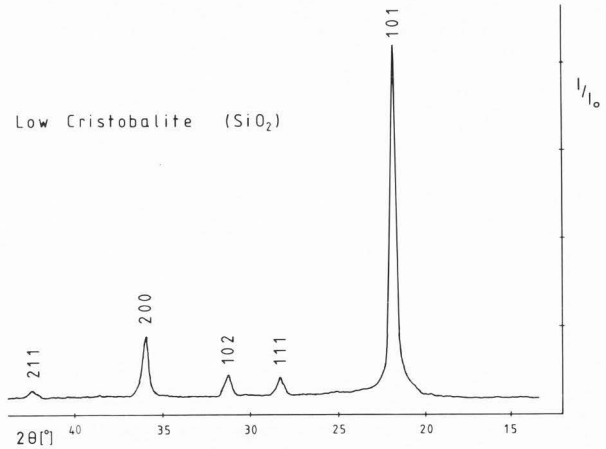


Fig.5

Fig.1: Bisected calculus prepared for SEM investigation. Central apatite core (arrow) and whewellite crystals (cross) in radial array.
Fig.2: Central apatite core of the calculus (higher magnification of Fig.1).
Fig.3: Section of the central core in still higher magnification with a particle containing Si and Mg (arrow)(a) and element distribution of Si and Mg (EDXA) (b).
Fig.4: EDXA analysis of the siliceous corpuscle in Fig.3.
Fig.5: X-ray diffractogram of the drug ingested by the patient ("Kieselerde"), revealing pure SiO_2 as low-cristobalite (ASTM-No. 11-695).

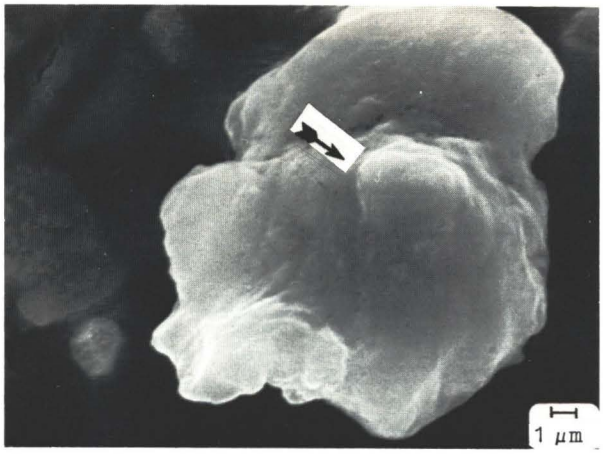


Fig.6a: Particle of urine sediment with local enrichment of Si (arrow)

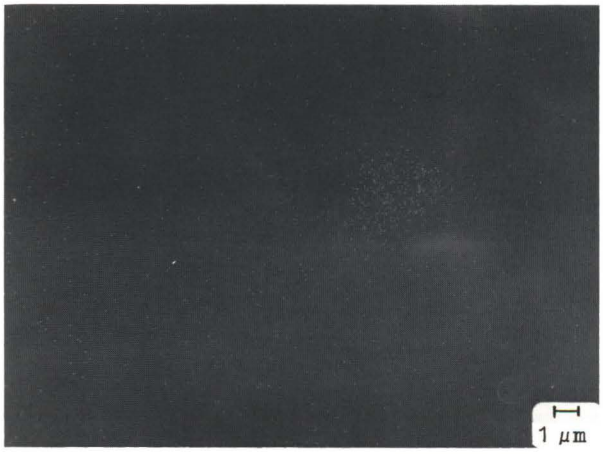


Fig.6b: Element distribution of Si in the above Fig.6a

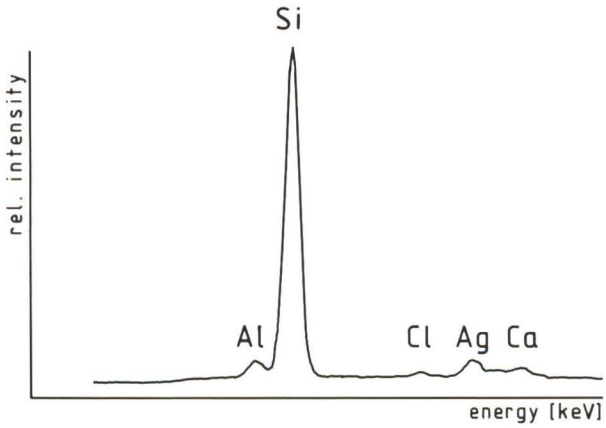


Fig.7: EDXA analysis of the Si-rich area in Fig.6

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Discussion with Reviewers

J.L. Meyer: In view of the very low solubility of SiO_2, is it certain that the source of this silicon in the urine and the stone is the ingested "Kieselerde"?
Authors: No, but very probable.

J.L. Meyer: Have you looked for silicon particles in the stones of patients not ingesting SiO_2?
Authors: In most cases, drug anamnesis of the patients is not known. In a series of nearly 1,500 SEM-investigated urinary calculi, Si is detected by EDXA at a frequency of about 1%.

S.R. Khan: In your discussion, you mention that "since the Si/Mg particles were found only in the central core of stones, a connection between SiO_2 ingestion and the formation of urinary calculi appears likely and plausible". Does not the presence of silica deposits only in the core and its absence from other parts of the stone, even when the patient was still passing silica containing particles and was on Kieselerde, suggest just the opposite? If there was a connection between the two then should not the silica deposits be present throughout the stone?
Authors: Silica particles are only found within the apatite core of the stone. There might be a special affinity of silica to apatite because of structural relationships contrary to Ca-oxalate which might be an explanation to these findings.