important to ascertain whether the inhibition extends to pepsin secretion, and this aspect is currently under review.

We wish to thank Professor S. K. Kyalwazi for his support and encouragement, and Mr. E. Zirimabagabo for technical help. The prostaglandins used in this study were either synthesized by Dr. W. P. Schneider in our laboratory or supplied by the Upjohn Company of Canada. We gratefully acknowledge the financial support of the Upjohn Company, Kalamazoo, Michigan, U.S.A.

During the performance of this work D. C. C. was on secondment from the Department of Clinical Surgery, Edinburgh.

References

Bundy, G., Lincoln, F., Nelson, N., Pike, J., and Schneider, W. (1971). Annals of New York Academy of Sciences, 180, 76.
Classen, M., Koch, H., Deyhle, P., Weidenhiller, S., and Demling, L. (1970). Klinische Wochenschrift, 48, 867.
Horton, E. W., Main, I. H. M., Thompson, C. J., and Wright, P. M. (1968). Gut, 9, 655.

- BRITISH MEDICAL JOURNAL 20 JANUARY 1973
 Jacobson, E. D. (1970). Proceedings of the Society for Experimental Biology and Medicine, 133, 516.
 Karim, S. M. M. (1971). Journal of Obstetrics and Gynaecology of the British Commonwealth, 78, 289.
 Karim, S. M. M., and Sharma, S. D. (1972). Journal of Obstetrics and Gynaecology of the British Commonwealth, 79, 737.
 Karim, S. M. M., Sharma, S. D., and Filshie, G. M. (1972 a). Proceedings of Brook Lodge Symposium on Prostaglandins.
 Karim, S. M. M., Somers, K., and Adaikan, Ganesan P. (1972 b). In pre-paration.
 Kirton, K. T., and Forbes, A. D. (1972). Prostaglandin, 1, 319.
 Main, I. H. M. (1969). British Journal of Pharmacology, 36, 214P.
 Ramwell, P., and Shaw, J. E. (1968). Journal of Physiology, 195, 34P.
 Robert, A., Nezamis, J. E., and Phillips, J. P. (1968 b). Gastroenterology, 54, 4263.
 Robert, A., Nezamis, J. E., and Phillips, J. P. (1968 b). Gastroenterology, 55, 481.
 Robert, A., Stowe, D. F., and Nezamis, J. E. (1971). Scandinavian Journal of Gastroenterology, 6, 303.
 Wada, T., and Ishizawa, M. (1970). Japanese Journal of Clinical Medicine, 28, 2465.
 Way, L., and Durbin, R. P. (1969). Nature, 221, 874.
 Wilson, D. E., Phillips, C., and Levine, R. A. (1971). Gastroenterology, 61, 201.
 Yankee, E. W., and Bundy, G. L. (1972). Journal of the American Chemical Society, 94, 3651.

Multiple Renal Silica Calculi

A. M. JOEKES, G. ALAN ROSE, JUNE SUTOR

British Medical Journal, 1973, 1, 146-147

Summary

Investigation of a patient with a history of renal colic, who had taken the equivalent of 2 g magnesium trisilicate after every meal for many years, showed that he was forming silica calculi. The nature of the stone was diagnosed only after quantitative analysis.

Introduction

Silica stone was first described by Herman and Goldberg (1960) in the U.S.A. and was attributed to the ingestion of magnesium trisilicate for treatment of oesophagitis. Five more cases, also in the U.S.A., were reported by Lagergren (1962). Few cases have been described since then, and none in this country. Herring (1962) did not report any in his survey of 10,000 urinary calculi analysed by crystallographic and x-ray diffraction techniques. It was therefore thought appropriate to report a case of recurrent silica stones in this country.

Case Report

A 68-year-old man was first seen in November 1971 with a history of two episodes of presumed renal colic in 1940 with no precise diagnosis made at the time. After a fall in 1966 he developed a severe left-sided renal colic and radiography showed a large stone in the lower end of the left ureter with a much smaller stone immediately below it. Shortly afterwards a small stone weighing 100 mg was passed. At the beginning of 1969 he started having a series of renal colics affecting both the left and the right side. In June a stone was apparently removed from a diverticulum in the bladder. Some months later a small stone about 4 mm long was passed. This was slightly yellowish and very hard. He was free of any further colic until the beginning of 1971,

A. M. JOEKES, B.M., F.R.C.P., Nephrologist G. ALAN ROSE, M.R.C.P., M.R.C.PATH., Consultant Chemical Pathologist JUNE SUTOR, M.SC., PH.D., Research Fellow

when he passed two small stones. He again passed a small stone after a renal colic in September 1971. On no occasion was the passage of stones accompanied by macroscopic haematuria.

On examination he was an active, well-preserved man. Blood pressure was 190/90 mm Hg lying and standing. There was a healthy, rather wide, left paramedian scar. No other significant abnormal physical signs were found.

Investigations were: haemoglobin 12.7 g/100 ml, white cell count 7,000/mm³, packed cell volume 41%, E.S.R. 25 mm in 1 hr, plasma urea 46 mg/100 ml, creatinine 1.1 mg/100 ml, sodium 140 mEq/l., potassium 4.0 mEq/l., bicarbonate 26 mEq/l., total protein 7.6 g/100 ml, calcium 9.6 mg/100 ml, phosphate 2.4 mg/100 ml, uric acid 8.1 mg/100 ml, 24-hour creatinine clearance 75 ml/min, 24-hour calcium excretion 85 and 84 mg, 24-hour urinary uric acid excretion 370 and 360 mg.

An intravenous pyelogram showed that the kidneys concentrated well and that the upper urinary tract was normal. There was a 1-cm low density calculus in the pelvic portion of the left lower ureter. The bladder was slightly trabeculated with a moderate residue after micturition.

A radioactive renogram with 20 μ Ci of ¹³¹I Hippuran and a urine flow rate of 4 ml/min showed a normal curve for the right kidney with no excretory abnormality. The left kidney showed considerably less good function with evidence of an obstructive lesion with a very slow excretory phase.

The day after the intravenous pyeolography the patient had a severe suprapubic pain eventually radiating to the left flank. This continued for two days and he then passed the calculus that we had seen in the pelvic portion of the left ureter.

STONE ANALYSIS

Quantitative stone analysis was carried out by using the system of Westbury and Omenogor (1970), but as extended by Westbury (1972) to include oxalate determination. In addition, the usual qualitative tests were performed (see table). It was also noted that the powdered material was insoluble in dilute HCl but was soluble in dilute NaOH, and could be precipitated from this solution on acidification with HCl, and that the fresh precipitate was soluble in further excess HCl. These reactions are those of silica, the presence of which was therefore suspected. The confirmation of this suspicion was sought by x-ray crystallography. Silica occurs in calculi in the opaline state which is non-crystalline, but on heating it can be transformed into various crystalline forms depending on the temperature of ignition. The x-ray powder diffraction method was therefore used to determine if any crystalline material was present in the stone. Small samples from

St. Peter's Hospitals and Institute of Urology, University College, London WC2H 9AE

Chemical Analysis of Renal Calculi

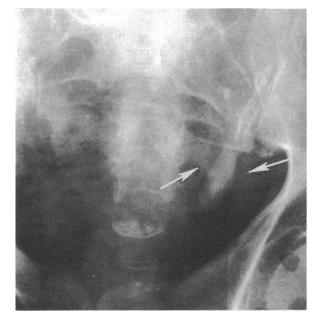
Date of stone removal or passage						••	1966	1971
Weight	••	••	••	••	••	••	3.6 g	0·30 g
Description	n	••	••	••	••	••	White semi- lunar	White ovoid
Calcium		•••			••		1.4%	2.5%
Oxalate	••	••	֥	••	••	••	6.7%	4.5%
Phosphoru Magnesiun	8	••	••	••	••	••	0.8%	1·5 % 0·5 %
Uric acid	u 				••		Nil	Nil
Ammonia							Nil	Nil
Cystine	••	••	••	••	••	••	Nil	Nil
Carbonate Ash	••	••	••	••	••	••	Nil	Nil 82.8%
Asn	••	••	••	••	••	••		62.0%

different areas of the calculus were powdered and photographed with CuKa radiation. The resulting diffraction patterns did not contain the discrete lines given by crystalline matter. A piece of the stone was then heated for 24 hours in a furnace at 1,000°C. The x-ray powder method showed that this material was crystalline and that it consisted of tridymite, one of the high temperature forms of SiO₂ stable between 870 and 1,470°C and metastable at room temperature. The calculus therefore consisted predominantly of opaline silica.

Discussion

The nature of the stone in this case was not suspected for five years despite several missed opportunities for inspired guesses. Firstly, the low radiodensity of the stone (see fig. 1) should have aroused some suspicions. Such low density might be due to cystine or magnesium ammonium phosphate. The former can be readily excluded, however, by the simple nitroprusside screening test, while the latter is most unlikely in the absence of infection of the urinary tract. Secondly, the stone analysis should have disclosed the unusual nature of the chemical composition. Stone analysis, however, is performed rather badly in most hospital laboratories, and, furthermore, such analysis is usually qualitative and not quantitative so that trace constituents can be erroneously reported as main ingredients. This presumably explains why chemical analysis of one of this patient's stones at another hospital showed "calcium, phosphate, and oxalate." Quantitative analysis in our laboratory immediately showed zero or low amounts of the usual constituents. Finally, on handling the stone it was immediately apparent that it had an unusually low density, feeling very light for its size.

On direct questioning the patient admitted that he had had indigestion for many years for which he had taken magnesium trisilicate mixtures after every meal. He is not certain whether this habit had already started in 1940 or began some time after



Low density silica calculus in lower end of ureter (arrowed).

this. The equivalent of 2 g magnesium trisilicate had been taken daily for many years. Such a rate of ingestion would not seem to be unduly excessive, and it seems surprising that more cases of silica stone have not been reported. This may be a reflection on the lack of enthusiasm shown by many hospitals for stone analysis. It is therefore strongly urged that stone analysis should be quantitative, especially since it has been shown by Westbury and Omenogor (1970) that this procedure is actually simpler than qualitative analysis.

References

- Herman, J. R., and Goldberg, A. S. (1960). Journal of the American Medical

Herman, J. K., and Goudderg, A. S. (1900). Journal of the American Medical Association, 174, 128.
Lagergren, C. (1962). Journal of Urology, 87, 994.
Herring, L. C. (1962). Journal of Urology, 88, 545.
Westbury, E. J. (1972). In preparation.
Westbury, E. J., and Omenogor, P. (1970). Journal of Medical and Laboratory Technology, 27, 462.

Tumoral Calcinosis in England

G. SLAVIN, L. KLENERMAN, A. DARBY, S. BANSAL

British Medical Journal, 1973, 1, 147-150

Summarv

Two cases of tumoral calcinosis are presented in patients living in England. The clinical and pathological features are described and attention is drawn to the need to consider exotic diseases in patients who have originated from or lived in the tropics.

The cause of tumoral calcinosis is not known. It may be a metabolic disease of obscure aetiology but local trauma often appears to be a factor in its development.

Introduction

Tumoral calcinosis is an uncommon condition of obscure actiology which is characterized clinically by lobulated, calcified, cystic masses usually situated near large joints such as the hips, shoulders, and knees and characterized pathologically by fibrous-walled cystic spaces containing structureless calcific debris and associated with a variable inflammatory reaction. It was named by Inclan (1943) who drew attention to the predominant characteristics of soft tissue swellings with massive

Northwick Park Hospital and Clinical Research Centre, Harrow, Middlesex HA1 3UJ

G. SLAVIN, M.R.C.PATH., M.R.C.P., Consultant Histopathologist L. KLENERMAN, F.R.C.S., Consultant Orthopaedic Surgeon A. DARBY, M.R.C.PATH., Senior Registrar, Histopathology S. BANSAL, F.R.C.S., Registrar, Orthopaedic Surgery