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MEDICAL APPROACH TO THE DIAGNOSIS OF KIDNEY STONES*

BOY FRAME, M.D.**

A gratifying experience in the professional life of a physician is that of detecting an underlying metabolic disorder in a patient with kidney stones. It is especially satisfying if the defect can be corrected and the recurrence of kidney stones thereby prevented.

When a patient with a kidney stone is first seen, it is often difficult to decide on the extent of the initial medical evaluation. Many patients pass but one stone in a lifetime and in this instance detailed investigation hardly seems warranted. However, when the patient is first seen it is not known whether or not that particular patient will be the one to have recurrent stone formation. Perhaps at the time of the next patient contact urinary tract infection, renal insufficiency, or both, will be present which will complicate metabolic study and treatment.

What are the diagnostic steps from the metabolic point of view in a patient with recurrent nephrolithiasis? Certainly the kidney stone should be obtained if at all possible for crystal analysis. Unfortunately patients will often have an extensive metabolic investigation and the physician later learns that the patient has his kidney stone at home in a dresser drawer as a souvenir.

Numerous techniques are available for stone analysis. Only the quality of crystal present in the stone is important, not the quantity. It must be remembered that kidney stones may be laminated and that the stone nucleus alone may harbor the clue to the basic metabolic error. With experience many stones can be identified by the naked eye with the aid of a hand lens. The spiked calcium oxalate stone which may be particularly painful is quite characteristic. Distinct and multiple laminations may give a clue to the presence of calcium phosphate stones. The cystine stone is smooth in outline, waxy in appearance and homogeneous in texture. Chemical spot tests of either the urine or urinary sediment are available for crystal identification. Microscopic examination of the urinary sediment may be of help but may also be misleading. The hexagonal crystals of cystine are most distinctive and unforgettable once observed under the microscope. It must be remembered that the crystal identified

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**Fifth Medical Division.
in the urinary sediment may not be the same crystal present in the kidney stone, itself. This is especially true after various forms of treatment have been initiated that predispose to crystal formation different from that originally present.

In some institutions polarized microscopy and x-ray diffraction are used for crystal identification but require special equipment and considerable experience. At the Henry Ford Hospital we are fortunate in having Mr. Parsons of the Physics Department who has analyzed approximately 1200 kidney stones for crystal structure by x-ray diffraction. With his techniques and experience he is able to give a prompt report of the crystal structure in kidney stones as well as in other body concretions. Many times he has given a correct lead to the underlying metabolic defect, particularly with regards to cystine stones. For practical purposes stone analysis should inform the physician whether he is basically dealing with a defect in calcium, cystine or uric acid metabolism. The situation may be complicated in those patients who pass stones containing several different types of crystals. For instance, a patient initially may have passed a uric acid or cystine stone, but after treatment with urinary alkalinization superimposed calcium stones may form.

**CALCIUM-CONTAINING STONES**

In temperate United States calcium-containing kidney stones present the greatest difficulty in diagnosis and treatment. The important first step in evaluating the patient with a calcium stone is to determine whether or not hypercalcemia is present. The serum calcium determination and its clinical interpretation is fraught with many errors and difficulties. Hospital laboratories in general do less than an ideal job in the accurate determination of the total serum calcium. Solutions of known calcium composition have been sent as unknowns to laboratories in community hospitals and the values obtained have ranged up to 100% in error. The normal range and standard deviation should be defined for each hospital laboratory. This is not easy because it is not always certain that supposed normal controls do, indeed, have normal serum calcium levels. Nevertheless, each laboratory should periodically insert a series of so-called normal serum samples for calcium determination in an attempt to keep a check on standards and normal values. There is no data as to whether a diurnal variation in the level of serum calcium exists; if it occurs, it must be of a minor degree. In normal subjects, a reduction in oral calcium intake for several days will result in a slight but significant depression of the mean serum calcium. Also, after the ingestion of several glasses of milk, the serum calcium may be slightly increased. It is, therefore, important to obtain the serum calcium when patients have been on an average calcium intake and also when in the fasting state. A difference of .2 to .3 mgm% can be important when one is evaluating patients with borderline hyper- or hypocalcemia. With most methods, a value over 10.5 mgm% should be suspect for hypercalcemia.

There is evidence to suggest that climate may affect levels of serum calcium. In a recent study serum calcium and also serum phosphorus and antirachitic levels were found to average higher in Puerto Rican than in Michigan women, perhaps a reflection of greater exposure to sunshine in the former. It is also possible
that serum calcium levels are normally higher during the summer months than in the winter. This has previously been shown to be true in patients with sarcoid who are thought to be unduly sensitive to the hypercalcemic action of vitamin D.

Prolonged venous stasis and vigorous forearm exercise during venipuncture have also been shown to increase the level of serum calcium to a significant degree. This occurs since protein free plasma is lost from the occluded vein resulting in a relative increase in plasma proteins to which calcium is bound. It is, therefore, important that technicians be instructed to keep the arm tourniquet on as briefly as possible and to have the patient refrain from using vigorous forearm contraction during the venipuncture. It has also recently been determined that the serum calcium value may be increased when the serum is allowed to come in contact with cork stoppers. Therefore, rubber stoppers are recommended, especially when samples are mailed and the serum inadvertently is in contact with the stopper for long periods of time.

Physicians seldom reflect on the complexity of the calcium fractions when ordering a serum calcium. The non-filterable calcium accounting for approximately 45% of the total serum calcium is bound primarily to albumin and, to a lesser extent, to various globulins. The ultrafilterable fraction, including complexed calcium, accounts for approximately 55%. A reduction in serum albumin for any reason is one of the most common causes of a depressed total serum calcium. Under these circumstances, tetany is seldom present since the concentration of ionic calcium is usually maintained. An elevation of the total serum calcium does not normally occur as a result of increased protein binding that might occur in various hypergammaglobulinemic states such as sarcoidosis and multiple myeloma. The hypercalcemia here is due to other mechanisms. Complexing of serum calcium to bicarbonate, citrate, phosphate and other ions accounts for a relatively small portion of the total serum calcium and ordinarily is not of clinical importance. However, in renal insufficiency, the complexed fraction may be increased, primarily because of binding with sulphates and phosphates.

There have been numerous attempts to improve the significance of the serum calcium value by fractionation into its various components. Determination of the ionic fraction is too difficult for most general hospital laboratories and has not as yet been proved more helpful than the determination of the total serum calcium alone in the evaluation of hypercalcemic states. A recent study of 166 serum ultrafilterable calcium determinations in 150 individuals showed no constant deviations of diagnostic significance in patients with malignancies, hyperparathyroidism, hypoparathyroidism, multiple myeloma, sarcoidosis, connective tissue disorders, muscle dystrophy, vitamin D resistant rickets and osteoporosis. The total serum calcium, therefore, if done accurately, is sufficient in evaluation of hypercalcemic states.

Once definite hypercalcemia has been established in a patient with nephrolithiasis, many underlying diseases need be considered. Table I lists the more common conditions giving rise to hypercalcemia which may be accompanied by nephrolithiasis.
Causes of Hypercalcemia and Nephrolithiasis

1. Hyperparathyroidism
2. Sarcoidosis
3. Malignancy, myeloma
4. Hypervitaminosis D
5. Immobilization
   a) Osteoporosis
   b) Paget's disease
6. Milk alkali syndrome

The diagnosis of hyperparathyroidism is largely a diagnosis of exclusion since there is no specific laboratory proof. If other causes of hypercalcemia can be excluded in a patient with nephrolithiasis, hyperparathyroidism is the most likely diagnosis. The presence of hypercalcemia in a patient with sarcoidosis or thyrotoxicosis does not automatically exclude hyperparathyroidism. There are numerous case reports in the literature where hyperparathyroidism has been present in conjunction with other hypercalcemic conditions. While hypercalcemia is common in malignancy, myeloma and thyrotoxicosis, nephrolithiasis appears rarely in these conditions. Perhaps this is a reflection of the short life expectancy in most patients with malignancy and the early diagnosis usually made in thyrotoxicosis.

The serum inorganic phosphorous is of relatively little value in the differential diagnosis of hypercalcemia and nephrolithiasis. As with the determination of serum calcium, its accuracy and proper evaluation is fraught with pitfalls. Dietary restriction of phosphorus will result in a depression of the fasting serum inorganic phosphorus. The administration of nonabsorbable alkalis will also lower the serum phosphorus and is one of the most common causes of hypophosphatemia in patients with dyspepsia and peptic ulcer. While classically the serum inorganic phosphorus is depressed in hyperparathyroidism, a normal or even increased value is fully compatible with the condition, especially in the presence of renal insufficiency. Hypophosphatemia may occur in almost all other hypercalcemic states and is not particularly helpful in differentiating hyperparathyroidism from other causes of hypercalcemia. It is to be remembered that the serum inorganic phosphorus is ordinarily higher in infants and also in postmenopausal women.

Many other laboratory tests and procedures have been utilized in the evaluation of patients with calcium-containing stones. The serum protein electrophoresis is helpful in excluding myeloma and sarcoidosis. The serum alkaline phosphatase may be elevated in most conditions giving rise to hypercalcemia and, therefore, is of little help except in association with subpereosteal resorption when it supports the diagnosis of hyperparathyroidism. In the differential diagnosis of hypercalcemic states, the cortisone tolerance test may be helpful. When cortisone is administered in the dose
of 150 mgm. a day for ten days, the serum calcium will frequently return to normal in sarcoid, hypervitaminosis D and in many malignant states. The hypercalcemia of hyperparathyroidism, on the other hand, is generally unaffected by such a course of corticosteroids, but exceptions have been noted.

During the past few years many attempts have been made to utilize the phosphaturic effects of parathyroid hormone in the diagnosis of hyperparathyroidism. The percent tubular reabsorption of phosphorus (%TRP), phosphate clearance (Cp) and the phosphate excretion index (PEI) popularized by various authors have been attempts at improving this diagnostic approach. Interested physicians in most clinics have lost enthusiasm for these tests since they are not specific and both false positive and false negative results have been reported. They have never been especially helpful to us in the diagnosis of any patient with hyperparathyroidism, and we now use them only in borderline cases and only in conjunction with the total clinical and laboratory information.

Various calcium infusion tests have also been developed in an attempt to improve diagnosis in hyperparathyroid states. These tests are based on the theory that intravenous calcium inhibits synthesis and release of parathyroid hormone, thereby reducing urinary phosphorus excretion. Supposedly in the patient with an autonomous parathyroid adenoma, calcium infusion is unable to effect parathyroid hormone release. Under these circumstances, urinary phosphorus either remains the same or may be increased. Goldsmith has developed a four hour test utilizing calcium infusion while measuring the effect on the phosphate-creatinine excretion ratio in the urine. In his experience this ratio is normally reduced during the second hour after calcium infusion but is increased in patients with hyperparathyroidism. With the use of his infusion test, Goldsmith has diagnosed hyperparathyroidism in patients with nephrolithiasis, even in the absence of hypercalcemia. We have observed both false positive and false negative results with calcium infusion tests which reduces their value in diagnosis. There is recent evidence to suggest that calcium infusion may influence the renal excretion of phosphorus, even in the absence of the parathyroid glands which, if true, would weaken the physiologic basis for the test. It is also to be remembered that the parathyroid glands should theoretically be suppressed in other hypercalcemic states. Under these circumstances calcium infusion may not affect the already suppressed parathyroid glands and results similar to those expected in hyperparathyroidism may be observed. In other words, the calcium infusion test does not differentiate hyperparathyroidism from other hypercalcemic states.

There have been exciting new advances in methods to assay blood levels of parathyroid hormone. Radioimmunoassay techniques at the present time are being improved and soon may be sensitive enough to be of greater help in the diagnosis of hyperparathyroidism. Insulin and growth hormone appear to be better antigenic stimuli than parathyroid hormone and presently assays for these hormones are approximately 5 times as sensitive as that for parathormone. New advances in this area of diagnosis are expected soon.
HYPERCALCIURIA AND NEPHROLITHIASIS

When hypercalcemia has been ruled out in a patient with a calcium-containing kidney stone, an attempt should be made to evaluate the presence of hypercalciuria. Renal excretion of calcium is complex and some of the important factors are only beginning to be understood.

The level of ultrafilterable serum calcium and the glomerular filtration rate determine the concentration of calcium in the glomerular filtrate. As renal damage progresses in hypercalcemic states, the amount of filtered calcium and hence the amount of calcium appearing in the urine is frequently reduced. There is evidence that complexed calcium is less readily reabsorbed in the renal tubules than is ionic calcium since infusion of such complexing substances as citrate, sulfate and EDTA greatly increases the urinary excretion of calcium. Current data suggests that parathyroid hormone increases tubular reabsorption of calcium. Hence, after the administration of parathyroid hormone the urinary excretion of calcium is initially decreased and only later when hypercalcemia accounts for an increased filtered load does the urinary calcium excretion increase. Following parathyroidectomy on the other hand, urinary calcium is initially increased and only after the serum calcium falls does the urinary excretion of calcium decrease. It has been stated that in mildly hypercalcemic states a normal or reduced urinary calcium concentration supports the diagnosis of hyperparathyroidism over other causes of hypercalcemia. The reliability of this thesis has not been fully tested. Vitamin D influences renal excretion of calcium, especially when administered in large doses. In some instances, such as in hypoparathyroidism, administration of vitamin D is followed by hypercalciuria before elevation of the serum calcium occurs. Hence, the effectiveness of therapy with vitamin D in hypoparathyroidism cannot be evaluated by the response in urinary calcium alone. Solute diuresis, especially after intravenous sodium salts, enhances urinary excretion of calcium and in many respects renal tubular handling of calcium and sodium are parallel. Current evidence based on micropuncture techniques indicates that most of the filtered calcium is reabsorbed in the proximal convoluted tubules, but some reabsorption occurs in the distal tubules as well.

The determination of the 24 hour urine calcium is of value only if considered in light of the total clinical and laboratory picture. There has been considerable disagreement as to the normal 24 hour urine calcium excretion value. This is due to the fact that different authors have used varying degrees of dietary calcium restriction during the test. In general, most normal subjects excrete less than 300 mgm. of calcium per 24 hours on a diet of average calcium intake. When milk and other dietary products are removed from the diet, urinary calcium excretion will generally be less than 200 mgm. per 24 hours. If a markedly restricted calcium intake of less than 150 mgm. in 24 hours is taken, the urinary calcium excretion should then be less than 150 mgm. per 24 hours. The patient as well as the nurse should be instructed in the correct urine collection technique and also adequate urinary collection should be confirmed by urinary creatinine determination. It is important to remember that calcium salts may precipitate on the walls of the specimen bottle and thereby falsely reduce the urinary calcium value. A small amount of concentrated acid added
to the urine collection bottle will keep the urinary calcium in solution. However this makes it more difficult for the laboratory in that the specimen then has to be neutralized before the calcium determination is performed.

Determination of the urinary pH may be of help in the evaluation of renal lithiasis, especially if the urine is free of infection. A persistently alkaline urine should always raise the question of an underlying renal tubular defect such as renal tubular acidosis. In this case hyperchloremic acidosis may be apparent from the results of the serum electrolytes. In borderline cases, the ammonium chloride load test may be used to determine whether there is a defect in the renal mechanisms for handling an acid load. Ammonium chloride solution, 1 gram per kilo, administered in a palatable solution with observation of the urinary pH for 6-7 hours will help define such cases. A decrease in the urinary pH below 5.5 indicates adequate renal tubular mechanisms for urine acidification and virtually rules out renal tubular acidosis. Measuring the hydrogen ion clearance index as proposed by Elkinton is a more refined way of testing for renal tubular acidosis.

Determination of the 24 hour urine oxalate will be of help in the diagnosis of the rare inborn error of metabolism oxalosis, which gives rise not only to repeated calcium oxalate stones, but also to a general tissue deposition of calcium oxalate. In this instance, there is a persistent and excessive excretion of urinary oxalate greater than 100 mgm. per 24 hours. It is to be remembered that calcium oxalate nephrolithiasis is very common and most patients with such stones do not have a metabolic error in oxalic acid metabolism. In these latter patients, the daily urinary oxalate excretion is less than 50 mgm.

Table II is a list of the more common causes of hypercalciuria that are to be considered in the differential diagnosis of the patient with kidney stones and increased calcium in the urine.

### Table II

<table>
<thead>
<tr>
<th>Causes of Hypercalciuria</th>
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<tbody>
<tr>
<td>1. Hyperparathyroidism</td>
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<tr>
<td>2. Hyperthyroidism</td>
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<tr>
<td>3. Immobilization</td>
</tr>
<tr>
<td>4. Osteoporosis (certain types)</td>
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<tr>
<td>5. Cushing’s syndrome</td>
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<tr>
<td>6. Metastatic cancer, myeloma</td>
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<tr>
<td>7. Renal tubular acidosis</td>
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<td>8. Fanconi syndrome</td>
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<tr>
<td>9. Vitamin D intoxication</td>
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<tr>
<td>10. Sarcoidosis</td>
</tr>
<tr>
<td>11. “Idiopathic” hypercalciuria</td>
</tr>
<tr>
<td>12. Paget’s disease</td>
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</tbody>
</table>
It is important to remember that the urinary calcium concentration is only one of many factors which determine whether or not a patient will have kidney stones. Patients may have marked hypercalciuria for long periods without developing kidney stones. On the other hand, there are many patients with normal urinary calcium excretion who have repeated calcium nephrolithiasis.

In general, the determination of the urinary calcium excretion is not of much help in the diagnosis of hyperparathyroidism since almost all conditions causing hypercalcemia also have associated hypercalciuria. An exception is the milk alkali syndrome where, because of renal insufficiency, urinary calcium excretion is normal or decreased despite hypercalcemia. Reduced calciuria also occurs in other hypercalcemic conditions after the development of significant renal failure. The hypercalciuria of hyperparathyroidism may be extreme, but seldom do such patients have nephrolithiasis. The cause for this is not known. The calciuria in involutional osteoporosis is variable, probably because there are many causes of osteoporosis and only certain types are accompanied by increased urinary calcium excretion. The incidence of kidney stones in patients with Cushing's disease has been a significant complication in some series. In multiple myeloma and metastatic cancer the renal excretion of calcium may be greatly increased, but rarely do these patients present with kidney stones. Hypercalciuria in renal tubular acidosis is variable and does not seem to be the determinate factor in the occurrence of kidney stones. The reduction of urinary citrate that occurs in the syndrome may be an important consideration in this regard. The hypercalciuria in the Fanconi syndrome again may be quite marked, but only occasionally are kidney stones a problem. Perhaps the associated aminoaciduria present in the syndrome helps maintain calcium in solution by enhancing chelation with amino acids.

There are several subclassifications of the entity known as idiopathic hypercalciuria which is frequently accompanied by recurrent nephrolithiasis and which may be difficult to distinguish from hyperparathyroidism. In some instances, the hypercalciuria is accompanied by hypophosphatemia, but the serum calcium is normal. Two separate theories have been postulated to explain idiopathic hypercalciuria. One theory is a primary renal tubular defect for calcium resulting in hypercalciuria and a tendency towards early hypocalemia, which is then corrected by a secondary hyperparathyroidism. The latter would account for the depression of serum phosphorus often present in the condition. The other incriminates a hyperabsorption of calcium from the intestinal tract and hence secondary hypercalciuria. Theoretically, one would expect in the latter instance a depression of the parathyroid glands and an increased serum inorganic phosphorus, but this does not occur. Nevertheless, evidence obtained in balance studies after the administration of sodium phytate and cellulose phosphate supports the contention that hyperabsorption of calcium may be an important factor. Under these circumstances, fecal calcium is increased and urine calcium decreased without the occurrence of a negative calcium balance. It has also been demonstrated that certain of the newer diuretic agents such as bendrofluorazide decreases urinary calcium excretion in patients with idiopathic hypercalciuria. The concurrent administration of both cellulose phosphate and bendrofluorazide is probably...
the best present method of reducing urinary calcium excretion in patients with idiopathic hypercalciuria and recurrent nephrolithiasis.

**Uric Acid Stones**

The radiolucent renal stone is almost always composed of uric acid except for the rare cases of glycinuria and xanthinuria. Metabolic evaluation in patients with uric acid lithiasis brings into consideration many etiologic factors. The patient may have classic gout or only a family history of gout. In the latter instance, hyperuricemia and also an increased urinary excretion of uric acid is usually present. The determination of both the serum and urinary uric acid may be misleading, especially if the patient has been on salicylates or diuretic agents. Some patients with uric acid lithiasis have hyperuricemia and hyperuricosuria without a past, personal or family history of gout. Other family members may also have increased concentration of uric acid in the serum and urine without evidence of gout. These patients are said to have essential or idiopathic hyperuricemia. Uric acid lithiasis may be a serious problem in these patients and to protect the kidney against gouty nephropathy, they should be treated with uricosuric agents as vigorously as if they had true gout.

There is also an increased incidence of uric acid stones in patients with chronic enteritis and colitis. In this instance the diversion of water excretion to the bowel results in a decreased urinary flow and a persistently acid pH of the urine which predispose to uric acid lithiasis.

Not infrequently patients with uric acid lithiasis will have normal concentrations of uric acid in the serum and urine. In this instance, a persistently acid pH of the urine is frequently observed. It has been postulated that these patients have a renal tubular defect for effective urinary alkalinization which predisposes to uric acid crystalization. It has been suggested that a defect in ammonia production by the kidney in such patients may be an important limiting factor.

Patients with uric acid lithiasis should always be evaluated for underlying blood dyscrasias such as leukemia, lymphoma and myeloproliferative disorders. Uric acid stones are especially likely to occur following treatment of leukemia with various antileukemic agents where there is an increased breakdown of nucleoprotein.

The traditional therapy of uric acid lithiasis has been fluid administration and alkalinization of the urine. Administration of uricosuric agents such as benemid may temporarily aggravate uric acid stone formation by an initial increase in uric acid excretion. A recent new approach offers promising therapy for the future. Allopurinol, a xanthine oxidase inhibitor, has been found to reduce synthesis of uric acid and thereby reduce its urinary excretion. This therapy may be especially helpful in those patients with gouty nephritis and ureteral obstruction from uric acid crystallization. Allopurinol may be used in conjunction with other modes of therapy including urinary alkalinization and uricosuric agents. One theoretic objection, that has not been a problem to date, is that the increased urinary excretion of xanthine occurring after the administration of Allopurinol may conceivably lead to increased formation of xanthine stones.
Cystinuria

A rare and interesting metabolic defect leading to nephrolithiasis is that of cystinuria. A family history of nephrolithiasis and onset early in life should lead one to suspect this diagnosis. The typical hexagonal crystals of cystine are readily identifiable in the urinary sediment. Urinary chromatography may also be of help in identifying the increased urinary excretion of not only cystine but also other dibasic amino acids such as lysine, glycine and ornithine. Recent evidence has thrown some doubts upon the long accepted theory of a renal transport defect for cystine in this condition. The renal tubular transport defect may actually involve cysteine rather than cystine.  

The time honored therapy for cystinuria has been similar to that for uric acid lithiasis, mainly hydration and urinary alkalinization. Night time hydration has been stressed as an important factor in prevention of cystine crystallization. Recently a new approach to the treatment of cystinuria has been introduced. It has been demonstrated that D-penicillamine can combine as a disulphide linkage with cysteine and thence be excreted in the urine. Since D-penicillamine cysteine is more soluble than cystine itself, cystine lithiasis is less likely to occur. At the present time, D-penicillamine is an expensive mode of therapy, but perhaps production costs can be reduced. Also other similar, but less expensive agents undoubtedly will be introduced. In some instances, skin rash and signs of renal irritation have necessitated the discontinuance of D-penicillamine.

Unusual Stones

Silicate stones are common in cattle where there is an increased intake of sand and dirt present in hay and other feed. The use of certain antacids such as magnesium trisilicate have also been rarely reported to be associated with the formation of silicate stones in man. The finding of silicate stones in the urine should also raise the possibility of malingering. Such was the experience at this hospital several years ago when a female patient brought in several small pebble-like stones that she claimed she had passed in the urine. These stones proved to be calcium silicate by x-ray diffraction and, when faced with the evidence, the patient admitted that she had selected the stones from her driveway. She used the story of nephrolithiasis and renal colic as a method of obtaining narcotics.

Rare instances of xanthine stones have been reported in the literature. As mentioned previously, the use of Allopurinol in the treatment of uric acid lithiasis may theoretically increase the tendency towards xanthine stone formation.

Rarely, renal tubular defects for other amino acids such as glycine have led to formation of pure amino acid stones. These unusual conditions are best diagnosed by the use of urinary paper or column chromatography. Rarely in the metabolic defect alkaptonuria precipitation of the dark stones containing homogentisic acid occurs in the urine.

In this discussion of nephrolithiasis the important factors of urinary obstruction and infection have been omitted. These certainly complicate diagnosis and therapy but are major subjects in themselves.
DIAGNOSIS OF KIDNEY STONES

SUMMARY

The correct medical diagnosis of kidney stones requires the concerted and cooperative effort of the internist, urologist and clinical pathologist. Stone analysis early will often give a clue to the metabolic error and prevent many unnecessary and expensive laboratory procedures. Once the stone is defined as being calcium, uric acid or cystine stone, the metabolic studies can be planned in a logical sequence. Early definition of the metabolic defect before the occurrence of infection or renal insufficiency not only simplifies investigation, but also allows for early definitive therapy in most instances. It is the physician’s duty to encourage the patient with kidney stones to submit to an early and adequate metabolic evaluation. In the long run this will undoubtedly save the patient time, convenience and expense, in addition to decreasing the chance of renal insufficiency. Treatment of pain, infection and obstruction in a patient with urinary tract stones without adequate metabolic evaluation is to be condemned.

REFERENCES