NEPHROLITHIASIS IN CHILDREN

BY

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The following two cases are examples of nephrolithiasis in children recently observed in the Hospital for Sick Children, Toronto.

Case 1. J. C., a boy aged thirteen years, was previously in hospital in December, 1939, with two days' history of frequency and haematuria. The urine showed a cloud of albumin and was loaded with red blood cells. The non-protein nitrogen was 67 mgm. per cent., the creatinine 1.8 mgm. per cent. A diagnosis of acute haemorrhagic nephritis was made. His condition cleared up on the usual treatment and he was discharged in six weeks. He was followed in the out-patient department, and the urine remained clear for two months; he then had dysuria and passed two stones. He was readmitted April 1, 1940, for investigation. Analysis of the stones showed calcium oxalate and magnesium and calcium phosphate. X-ray showed many small bilateral calculi in the calices and bilateral hydronephrosis, especially the left side. The non-protein nitrogen, creatinine and cholesterol were normal, but the urine concentrated to a specific gravity of only 1010 (volume 70 c.c.). Urine culture gave no growth from bladder or ureters. Blood calcium on admission was 13.2 mgm. per cent., phosphorus 4.1 mgm. per cent. Subsequent estimations were normal and below normal. Phosphatase was 26 units. X-ray examination of the long bones was negative. Calcium balance was negative with two-thirds excreted in the urine.

Following an unsuccessful attempt to dissolve the stones by medical treatment, a left pyelolithotomy was done on May 30, 1940, to be followed by a similar operation on the right side at a later date.

Case 2. The second case was treated surgically. B. M., a girl, was aged two-and-a-half years when first admitted in October, 1937, with bilateral renal calculi. Kidney function tests were normal. The urine cultured bacillus coli. Right nephrolithotomy was performed and three calcium phosphate stones removed. She returned in November, 1937, with a right ureteral calculus as well as those in the left kidney pelvis. The urine concentrated to a specific gravity of 1015. B. coli were found in the urine on culture. In January, 1938, a left nephrolithotomy was done and three calcium carbonate stones removed. During that year she passed stones on three occasions and returned in December, 1938, with a stricture of the left ureter, which was dilated, and left pyelonephritis; new stones were present on the right side. She remained moderately well for three months and then returned in March, 1940, with severe pain in the left side and a soft fluctuant mass—an infected hydronephrosis. X-ray showed bilateral calculi. Non-protein nitrogen was 109 mgm. per cent. and creatinine 3.45 mgm. per cent. The haemoglobin was 43 per cent. Despite transfusion, fluids and irrigations by indwelling ureteral catheters, she died suddenly April 12, 1940, at five years of age. No autopsy was obtained.

At this hospital, since 1924, there have been four other recurrences of nephrolithiasis. Three of these followed one to two years after a nephrectomy for unilateral stone on the opposite side, and were removed by lithotomy without further trouble. The other was a boy (L. D.) aged twenty-three months on admission in June, 1924, with bilateral renal calculi and a left ureteral stone. These were removed from the left kidney and ureter and on analysis showed calcium carbonate and triple phosphates. Pyuria persisted and the child had an acute flare-up with anuria in December, 1925. X-ray showed a large calculus in the right kidney pelvis and multiple stones in the left, and a diseased and functionless left kidney. Following right nephrolithotomy and left nephrectomy, symptoms and pyuria entirely cleared.

There have been fifty-two cases of urinary calculi at the Hospital for Sick Children, Toronto, since 1924: three urethral, one vesical, sixteen ureteral, and thirty-two renal. In the latter group the average age at onset was seven years, with range from two months to thirteen years. Sex incidence was seventeen females to fifteen males. Seventeen were right-sided, three left-sided and seven bilateral. The commonest symptoms were pain, vomiting, haematuria, frequency and passage of stones. The urine showed white cells, red cells and albumin. Of fourteen recorded estimations of non-protein nitrogen, six were greater than 35 mgm. per cent. Urinary infection was present in thirteen cases; absent in ten; not reported in nine. The coli group of organisms was the most frequent type, being present in eleven; staphylococcus aureus in one; micrococcus ureae in one; streptococcus haemolyticus was present in two; and staphylococcus albus in one in association with the colon bacillus.

TREATMENT. In nine cases the stone was passed, with cure in six. Operations were performed as follows: nephrolithotomy in nine cases, nephrectomy in five, nephropexy in one. There was no operative mortality. One death was attributable to the lithiasis (B. M.); four deaths occurred from other infection, with stones found at post mortem, but with no symptoms (the other bilateral cases).

DIAGNOSIS. Seven cases had one to two previous admissions with symptoms referable to stone and diagnosis of: abdominal pain, two cases; acute nephritis, two cases; chronic appendicitis, two cases; intestinal obstruction, one case.

Etiology. Experimentally stones have been produced by artificial hyperexcretion of oxamide, calcium oxalate, calcium carbonate; by parathormone or large amounts of vitamin D, producing excessive calcium excretion in the urine; by impregnation of urinary epithelium with crystalline matter and infection, lime salts being precipitated; and by vitamin A starvation causing keratinization of urinary epithelium and lime salt precipitation in alkaline urine.

Clinically, phosphaturia, oxaluria, uraturia are associated with an increased incidence of calculi. Cystin, xanthin and fibrin stones are also found with hyperexcretion. Hyperparathyroidism produces excess calcium excretion in the urine and bilateral lithiasis. Decreased solubility and precipitation of

crystalloids may also be produced by abnormal colloid changes, even though the concentration is not increased.

Infection by urea-splitting bacteria plays a part by precipitating lime salts in the epithelium and by preventing acidification of the urine by the production of ammonia. In a series in Cleveland (Higgins and Mendenhall, 1939) this was present in 77 per cent. of cases. Also focal infection was found in 57 per cent. of cases, suggesting an association. Other observers give figures varying from 20 to 60 per cent.

Urostatics as a primary cause has no clinical or experimental proof but is important in relation to stagnation and infection. Urinary reaction is an important factor in maintaining solution and within certain limits determines the composition of the stone. Amorphous phosphate, carbonate and triple phosphate are precipitated in intensely alkaline urine. Urates, oxalates and crystalline phosphate and carbonate are precipitated in neutral or slightly acid urine—oxalates pH 5.9; urates 5.6; phosphates 6.2; uric acid 6.5. Experimentally alkaline stones will not form in acid urine, whereas alkalization intensifies the process.

Malnutrition and vitamin A deficiency are suggestive factors, but proof is not clinically conclusive. Other factors are bone diseases, foreign bodies, bacterial clumps and sutures.

Medical treatment. Many methods have been tried in an attempt to dissolve urinary calculi. Small stones have been dissolved by continuous irrigation with phosphoric or other weak acids, by means of indwelling catheters, but denser stones, especially of oxalate and urate, have not responded to this treatment. Crowell used alkaline lavage in dealing with cystine stones.

Acid ash diet with vitamin A was popularized by Higgins (1939) and others. Soft calcium phosphate and carbonate stones not infrequently dissolve, but denser ones, with compaction and secondary internal crystallization are unaffected. The pH shift is often difficult to decide, as stones are usually mixed and certain types are found in both alkaline and acid urine. Keyser (1939) reports six alkaline calculi dissolved by acidification and two urate ones by alkalization, but states that this method usually fails.

Phosphaturia can be corrected with sodium benzoate and glycocoll without change in urinary reaction. This is due to the production of hippuric acid, which increases the solubility of calcium phosphate. Rapidly recurring stones have been successfully prevented by this method.

Dissolution is therefore usually unsuccessful and should be attempted only with softer stones and for a short period (a few weeks), or where surgery is prohibited.

Surgical treatment. Nephrolithotomy is the method of choice, and the following precepts for the prevention of recurrence of calculi should be observed:

Be sure to remove all the stones; have them analysed. X-ray immediately and at regular intervals for several years. Correct any metabolic errors producing hyperexcretion, and investigate when hyperparathyroidism is suspected. Give a diet low in the stone-forming crystalloid and with acid or alkaline ash as indicated. To maintain alkalization or acidification, drugs may be necessary

(e.g. urate stones, low purine diet and alkaline ash: oxalate stones, low oxalate diet, acid ash and vitamins B and D, associated with an endogenous source of oxalate; phosphate and carbonate stones, acid urine). The pH of the urine should be estimated with nitrazene paper by the patient. Give vitamin A. Eliminate infection by drugs such as sulphanilamide or mandelic acid. Remove foci of infection. Correct urostatics. Give at least two quarts of fluid daily.

Due to these measures, the incidence of recurrences of calculi has sharply decreased in the past few years, from about 30 to 50 per cent. in 1915 down to about 5.3 per cent. at the present.

REFERENCES

- Higgins, C. C., and Mendenhall, E. E. (1939). *J. Urol.* **42**, 436.
Keyser, L. D. (1939). *Loc. cit.*, 420.