[Hydronephrosis]

GRAND ROUNDS

February 11, 1959

Patient [redacted] - A colored male was born at [redacted] in [redacted] after a normal pregnancy. At the age of 3 years, he was taken to a doctor in [redacted] because of dribbling incontinence and enuresis that was said to have existed all his life. The doctor thought the "neck of the bladder" was malformed and inserted an indwelling catheter.

In [redacted] 1949, the patient was found to have spasmodic flank pain on the right side. Fever was also present.

Blood: Hgb. 9; RBC 3 million; BUN 67; CO_2 14 mEq.

Urine: Sg 1001; numerous WBC and a positive culture for pseudomonas. Albumin +.

A suprapubic cystotomy was made and a catheter left in place, while urethral dilatations were done which restored the urethral flow of urine. The suprapubic cystotomy was closed. Infection persisted. It was found that bilateral hydronephrosis was present with hydroureters due to bladder neck obstruction. In spite of all available antibiotics fever continued for week after week, and pseudomonas remained. Periods of acidosis were recorded and more than one episode of convulsions (never occurred again until 1958). Finally the clinical condition improved, and the patient was discharged.

In 1952, he was readmitted from the Clinic. His bladder was frequently distended. Hgb. 87; BUN 120 mg.; P.S.P. 5% excretion in 30 minutes; low urine S.G. with numerous W.B.C. Retrograde pyelography showed a distended bladder, trabeculated, with numerous diverticulae but no reflux into the ureters. The patient was treated by blood transfusion. Urological opinion was that renal damage was more pertinent than the urethral obstruction. With hospital care the general condition improved.

In [redacted], 1956, bilateral nephrostomy was done to relieve obstruction. In [redacted] 1956, these nephrostomy openings were closed and a suprapubic cystotomy was done for the second time. At that time the height of the child fell below the 3rd percentile level, the blood urea fluctuated between 50 and 100. Anemia was constant.

The patient was readmitted in [redacted], 1957, because of vomiting, anemia and oliguria.

Blood: BUN 86; Ca 9.1; P 5; Na 142; K 4.9; Hgb. 7.0; CO_2 17.7; Cl 106.

Urine: Pseudomonas, B. subtilis, aerobacta, and strep faecalis all present. Maximal Sg 1010. Serum albumin 4.5; globulin 3.6.

Patient was readmitted in [redacted], 1958, for consideration of further surgery to reopen urethral pathway or to reopen nephrostomy drainage. No surgery was done.

The patient was given a low protein diet and forced fluids plus 3 grams/day of $NaHCO_3$.

Na 141	Hgb. 8.6
K 5.0	Ca 8.3
CL 112	P 5.6
CO_2 22	BUN 100

In [redacted] 1958, the patient had a convulsion and went to [redacted] complaining of pain in the legs, weakness, lassitude, unable to walk, unable to speak clearly, with heavy breathing, and a low grade fever. There was no vomiting or diarrhea. The patient was suspected of having a brain tumor. An L.P. was done - pressure 330 mm. Pandy +. Sent to my service at T.C.H. for 2 days where the patient was found to have severe dehydration (yet voiding well) severe acidosis, azotemia, and renal failure (osteoporosis of the bones was strongly suspected).

CO_2	5 mEq.	BUN 166
Cl	113	Creatinine 6.2
Na	135	Alkaline phosphatase 50.7
P	4.9	Hb. 5.4

Ca 3.9 to 4.2 (done 5x)

BP 120/84

I.V. therapy commenced. Another convulsion occurred and the patient sustained multiple fractures. Transferred to [redacted] for additional orthopedic care.

Given 3 liters of fluid per day (more than double the requirement for a normal child of same weight), two liters of ringers lactate, and 1 liter of maintenance fluid. Given I.V. Ca gluconate. Voided $2\frac{1}{2}$ liter/day. Given Shohl's solution (A Citrate/citric acid) 30 cc t.i.d., Vitamin D, (a lactate 2 grams, b.i.d.). Blood transfusion. After 14 days, energy returned, muscle power returned, etc., and general condition was very good and the patient mentally alert. (Bun 60). Osteoporosis was generalized and over a 6 week period Ca rose to 8.5 mg. Phosphorus was never high (indeed it has fallen recently to 2.9). Blood chemistry returned to normal state of acid base balance and blood urea 30-40 mgm. To prevent a trend towards metabolic alkalosis K was added to I.V. fluid (20 mEq/1).

The patient has remained feeling well until three weeks ago when headache developed along with sudden hypertension (150/110) controlled with antihypertensive drugs. (Apresoline + Serpasil). Fractures have not healed nor has any significant recalcification of bone occurred. The child needs about 1500 cc of extra fluid/day, 10 grams of NaCl and extra K (Orange juice, etc.).

Problems for Discussion:

1. Recognition of early obstruction and treatment of obstruction in early life.

   a. All or nothing phenomenon?

   b. Ileal bladder or ureter verus lead pipe ureters.

   c. Removal of infections. (Never get rid of infection while tube in situ).

2.a. Renal damage. Progressive destruction of the kidney with hypertrophy of remaining nephrons.

   b. Specific tubular damage - loss of base-acidosis.

   c. With acidosis osteoporosis - fractures. Is parathyroid involved? Treatment.

   d. Hypertension. Why?