RECOVERY FROM PERITONITIS FOUR YEARS AFTER BILATERAL TRANSPLANTATION OF URETERS

BY

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Not until the past five years have the biochemical abnormalities that follow uretero-colic anastomosis been extensively studied. Ferris and Odel (1950) found that of 141 patients who had undergone bilateral uretero-sigmoidostomy, 80% showed a reduced alkali reserve and 79% a hyperchloraemia. The condition of hyperchloraemic acidosis was usually associated with a high blood urea. These findings were confirmed in the series of 201 cases collected by the British Association of Urological Surgeons (Jacobs and Stirling, 1952), in which 78% had a lowered alkali reserve, and 31% the full syndrome of hyperchloraemic acidosis with raised blood urea. In only 6% were the levels of the blood urea, chloride, and alkali reserve all normal. Hypokalaemia was present in about a third of the patients.

Symptoms attributable to electrolyte disturbances in patients with bilateral transplantation of the ureters include general weakness and fatigue, thirst, nausea, vomiting, anorexia, a salty taste in the mouth, hyperventilation, and loss of weight. There may be rectal urgency, and polyuria with diarrhoea. Osteomalacia due to chronic acidemia with consequent renal loss of calcium may occur with bone pains and multiple fractures (Pines and Mudge, 1951; Badenoch, 1952; Moore, 1952).

Excessive reabsorption of chloride and urea from the colon forms the basis of the electrolyte disturbance, but it is now considered that an impaired renal function is necessary for hyperchloraemic acidosis to develop (Lapides, 1951; Parsons *et al.*, 1952; Pyrah, 1954). Such an impairment results from an ascending infection from the colon, or back-pressure effects, leading to a diminished capacity of the distal tubules to acidify the urine. Hypokalaemia is probably mainly due to loss of

base in patients with polyuria and diarrhoea, and is therefore a secondary manifestation (Jacobs and Stirling, 1952).

Any intercurrent illness associated with diarrhoea or vomiting, but especially an attack of acute pyelonephritis, may precipitate acute symptoms in patients with bilateral transplantation of the ureters. The electrolyte balance of these patients can be more easily upset than in the normal. Westlake (1954) has described the recovery of a man in acidaemic coma with hypokalaemia, precipitated by acute pyelonephritis, four years after operation. In this paper the recovery is reported of a man with transplanted ureters who had intestinal obstruction and peritonitis due to a band four years after total cystectomy for carcinoma. The post-operative care was undoubtedly complicated by the transplantation. As patients with bilateral ureteric transplants into the colon become more numerous such problems will be seen more often in non-urological surgical units, as in the present case.

Case Report

In 1945 the patient, aged 55, underwent a partial cystectomy for papillary transitional-cell carcinoma. He remained well until November, 1950, when he again developed haematuria. In December, 1950, Mr. V. C. Pennell performed a total cystectomy with transplantation of the ureters into the sigmoid colon, by Coffey's method, in two stages. This was complicated by the development of a faecal fistula between the rectum and the anterior abdominal wall which was excised in March, 1952. After this third operation he remained well except for loss of appetite and a slight loss of weight. An x-ray film of the dorso-lumbar spine in November, 1952, showed moderate osteoporosis, and another in May, 1954, following a slight fall from a ladder, a fracture of the lower end of the right ulna with extreme osteoporosis. In January, 1955, his blood urea was 78 mg./100 ml.

On March 24 he was readmitted to Addenbrooke's Hospital with a history of six days' upper abdominal colic and vomiting which over the last 24 hours had become faeculent. When examined he was an ill, dehydrated, but fully conscious man with no clinical evidence of acidosis or uraemia. His abdomen was resonant but greatly distended, with tenderness in the upper half and a generalized positive release sign; a few bowel sounds were present. A straight x-ray film of the abdomen showed distended loops of the jejunum and proximal ileum, with fluid levels.

Laparotomy revealed peritonitis with a fibrous band between the lower ileum and the anterior abdominal wall.

Blood Electrolyte State and Fluid Balance During Post-operative Period

Post-operative Day:	0	1	2	3	4	5	6	7	8	9	10	11	12	13	14	27
Serum Na (mEq./l.) (normal 135-140)		142		151		162			173		158		141			136
" K " (" " 4-5)		3.0		3.3		2.7			3.7		3.7		3.1			6.0
" Cl " (" " 100-105)		109		108		117			113		114		109			107
Blood urea (mg./100 ml.) (normal 20-40)		160		201		228			230		115		58			31
Blood CO ₂ -combining power (mEq. l.) (normal 25-30)				30		30.4			35.4		29.1		23.6			22.7
Daily oral intake (g.):																
Potassium citrate					10	10	20	20	20	12	12	12	12	20	20	Stop
" chloride										7.5	7.5	7.5				
Daily intravenous fluid (pints*):																
Blood	1															
Normal saline	2	1														
N/5 saline with 4% dextrose	2	2	3	3	1											
Darrow's solution		1	1	1	1											
Daily oral intake (oz.*)	8	14	5	6	55	67	66	116	119	166	159	126	144	102	102	96
" gastric aspirations (oz.)	15	36	37	4	6	6										
" output of urine (oz.)	0	59	31	67	38+	30	25	11+	38+	29+	56	58	52	32+	38+	76
Antibiotic	Oxytetracycline 6-hourly		250 mg.	i.v.		Chloramphenicol	250 mg.	orally	6-hourly				Sulphadimidine hourly		1 g.	8-

* To convert pints and fluid ounces to millilitres, multiply by 568 and 28 respectively.

A loop of small intestine was twisted round the band, and there were three small perforations in the distended proximal intestinal loop which were closed after division of the band. There was no evidence of secondary deposits in the pelvis or liver.

Post-operatively he developed a paralytic ileus which persisted for five days, but at no time did he manifest definite clinical evidence of electrolyte disturbance, except for drowsiness and dulling of his mental faculties whilst the blood urea was at its height. The accompanying Table shows the fluid balance and blood electrolyte states over the post-operative period. When last seen (October, 1955) he was still well.

The ileus was treated by gastric aspirations and intravenous fluids, with oxytetracycline, 250 mg. intravenously six-hourly. A rectal tube collected the urine. On the first post-operative day the serum chloride, serum sodium, and blood urea levels were raised, and the serum potassium was reduced. On the third day, as the gastric aspirations decreased, the chloride, sodium, and urea levels had risen, and on the fifth day after operation the serum chloride was 117 mEq/litre, the serum sodium 162 mEq/l., and the blood urea 228 mg./100 ml. On this day the serum potassium had fallen to 2.7 mEq/l. Oral potassium in the form of potassium citrate was given as soon as the oral intake became 2 oz. (57 ml.) hourly. At first 2 g. was given four-hourly, but two days later this was raised to 4 g. This high dosage of potassium citrate was maintained up to his leaving hospital, but on the twelfth day the serum potassium was still only 3.1 mEq/l. By the 27th day, however, it had become normal.

The blood urea reached a peak of 230 mg./100 ml. on the eighth day, but had fallen to normal by the time of leaving hospital. The serum chloride and sodium levels remained raised, despite the gastric aspirations, until the tenth day, after which they persisted in the high range of normal. The blood CO₂-combining power was normal on the third and fifth post-operative days, but had risen to 35.4 mEq/l. by the eighth day, presumably as a result of the potassium citrate therapy. It fell to a level of 23.6 mEq/l. on the twelfth day and was still at about this level, on the acid side of normal, at the time the patient left hospital, despite the large doses of oral potassium citrate.

Discussion

Our patient must be presumed to have had an electrolyte abnormality before his admission with peritonitis. There was a loss of appetite and loss of weight in the year previously, osteoporosis was seen on radiography in 1952 and 1954, and the blood urea was raised to a level of 78 mg./100 ml. three months before admission. From these facts one might deduce that he had been in a state of hyperchloraemic acidosis. As is often the case with surgical emergencies, no electrolyte estimates were carried out pre-operatively, and there was thus no baseline to work from in the first few post-operative days. We felt more in the dark than usual until we discovered how effectively a rectal tube drained off the urine. Furthermore, this prevented the reabsorption of chloride and urea.

The serum chloride and serum sodium levels were raised on the first post-operative day, and the CO₂-combining power was normal, despite the previous vomiting and gastric aspiration. The chloride and sodium levels rose as the ileus subsided. It would appear, in fact, that the loss of gastric fluid compensated the probable hyperchloraemic acidosis, and may indeed have prevented a fatal outcome. Except immediately after the operation, when normal saline was given, only 4% dextrose-N/5 saline and the alkaline Darrow's solution were given intravenously. The urinary output remained satisfactory from the first day and made the course easier to control. The use of antibiotics, at first oxytetracycline and later chloramphenicol, was important, not only in the treatment of the peritonitis but also in the prevention of ascending renal infection.

The low serum potassium persisted despite the large doses of oral potassium. This probably indicated a large total body deficit due to long-standing increased potassium losses in the urine. The sequence of events may be that chronically damaged kidneys cannot excrete a concentrated urine and the volume is determined by the total amount of solute to be excreted. This leads to a polyuria, and higher amounts of potassium than usual are "washed out" in the urine despite a urinary potassium concentration within the normal range. Potassium chloride was not given to our patient intravenously because of the raised serum chloride with the fear of acidosis, though it might have been used in conjunction with a suitable alkali. The availability of 14.5% potassium citrate for intravenous use would have been an advantage—potassium citrate is more stable than potassium bicarbonate and easier to make than potassium lactate as recommended by Pyrah (1954)—and this is now being prepared in this hospital for the addition to intravenous fluids as an alternative to 10% potassium chloride.

Summary

It is common for patients with bilateral transplantation of the ureters into the colon to develop an electrolyte abnormality, of which a hyperchloraemic acidosis with a raised blood urea is the most serious. The abnormality is due to an excessive reabsorption of chloride and urea from the colon in association with an impaired renal function. Hypokalaemia is a secondary complication, and occurs in a third of these patients.

Any intercurrent illness associated with diarrhoea or vomiting, but especially an attack of pyelonephritis, may precipitate acute symptoms. The recovery of a patient with peritonitis and intestinal obstruction from a band four years after bilateral uretero-sigmoid anastomosis is described.

Following operation recovery occurred after gastric aspirations, rehydration with intravenous fluids, the administration of potassium, and a course of antibiotics. A rectal catheter drained off the urine satisfactorily. It was felt that the gastric aspirations necessitated by the development of a paralytic ileus compensated the tendency to a hyperchloraemic acidosis and diminished the alkali requirement, which is normally such an important feature of the treatment. Comments are made on the electrolyte difficulties met with post-operatively, particularly in the treatment of the hypokalaemia.

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For every seven children killed on the roads of Northern Ireland, ten die in accidents in the home, the Northern Ireland Ministry of Health and Local Government stated recently. As the result of home accidents over 2,000 people needed hospital in-treatment and 25,000 had to attend out-patients annually. The cost to public funds was estimated at £150,000. The number of beds occupied in one hospital by such accident cases in 1955 represented the equivalent of 3,000 "patient" days.