Secondary Chloride-losing Diarrhoea

Observations on Stool Electrolytes in Infants after Bowel Surgery

IAN AARONSON

From The Hospital for Sick Children, Great Ormond Street, London

Aaronson, I. (1971). *Archives of Disease in Childhood*, 46, 479. **Secondary chloride-losing diarrhoea. Observations on stool electrolytes in infants after bowel surgery.** Among 40 infants with postoperative diarrhoea, 3 were found to have a stool chloride concentration in excess of the sum of sodium and potassium ion concentrations. All 3 had had a recent episode of intestinal obstruction and had developed a sugar intolerance.

Congenital chloride-losing diarrhoea is a rare disorder, first described 25 years ago by Gamble *et al.* (1945) and Darrow (1945). In these patients, copious water stools are found to contain an unusually large amount of chloride ion, which characteristically exceeds the sum of the stool sodium and potassium ion concentrations, and is associated with hypokalaemia and a metabolic alkalosis.

After bowel operations in infants, severe diarrhoea not infrequently occurs, often for which no cause can be seen. The possibility that some abnormality might occur in the handling of chloride ion in the bowel to produce diarrhoea has led to an investigation of the stool chloride, sodium, and potassium ion concentrations in these patients. In three cases, the concentration of stool chloride was found to exceed the sum of the sodium and potassium ion concentrations, in one case by a very large amount.

Material and Method

Random stool samples, uncontaminated by urine, were collected from 40 patients under 18 months of age who had developed diarrhoea after bowel surgery. In 32 of these, operation had been undertaken for intestinal obstruction. Six of these patients had been given an ileostomy and 13 a transverse or descending colostomy. Patients with a known possible cause for the diarrhoea, e.g. a gut pathogen, sugar intolerance, or extensive bowel resection, were included in the survey. 59 separate stool samples were collected. In addition, a sample was collected for comparison from each of a further 18 patients in this age group, who were considered to be passing normal stools, 6 of which were from ileostomies and 6 from colostomies.

After homogenization and digestion by nitric acid, sodium and potassium ion concentrations were measured by flame photometry, and the chloride by titration against silver nitrate. The results were expressed in terms of stool water by calculation after drying an aliquot by water bath and a concentrated sulphuric acid chamber.

A note was made from the patient's records of the serum electrolyte values during the period of observation, and a record made of the intake of the patients during the 24 hours preceding the stool collection. Patients receiving supplementary oral chloride were excluded.

Results

The range of values obtained from all patients with diarrhoea while on a variety of feeds is shown in Fig. 1, where the stool chloride concentration in mEq/l. stool water is plotted against the sum of the sodium and potassium ion concentrations. Fig. 2 shows the range of values, similarly expressed, obtained from the control group. In all cases, serum electrolyte values were unremarkable.

The 3 patients in whom a large concentration of chloride in the stools was detected were the subject of repeated analyses and the values obtained for stool chloride, sodium, and potassium ion concentrations are recorded in Fig. 3.

Case Reports

Case 1. A male infant who developed signs of intestinal obstruction 48 hours after a normal delivery. At laparotomy, Hirschsprung's disease affecting the whole of the colon was confirmed, and an ileostomy was fashioned. Over the next week, the ileostomy actions became progressively more watery and profuse. An intolerance to disaccharides in the diet was shown,

FIG. 1.—Relation of concentration of Cl^- to $\text{Na}^+ + \text{K}^+$ in random stool samples from 40 infants with diarrhoea after bowel surgery.

but in spite of their withdrawal the diarrhoea continued almost unrelentingly for the next 4 months.

Six samples of ileostomy fluid were analysed over this period (Fig. 3). The second sample at 4 weeks showed an abnormally high Cl concentration of 124 mEq/l., with Na 115 mEq/l. and K 5 mEq/l. This

feature was again seen in the fifth sample at 13 weeks, when Cl was found to be 130 mEq/l., Na 116 mEq/l., and K 10 mEq/l. The serum electrolyte concentrations were estimated at frequent intervals, but did not show any remarkable features, and were readily maintained within normal limits by the occasional intravenous

FIG. 2.—Relation of concentration of Cl^- to $\text{Na}^+ + \text{K}^+$ in random stool samples from patients with normal stools after bowel surgery.

FIG. 3.—Concentration of Na^+ , K^+ , and Cl^- in random stool samples from 3 infants with excessive chloride loss.

administration of sodium chloride solution. Oral intake at the time of the analyses consisted only of minced chicken. For a period of several weeks during the second month of life, *Esch. coli* type O125 and *Ps. pyocyanea* were isolated from the stools, but the presence of these organisms did not appear to be reflected in the volume of the diarrhoea.

By 5 months of age the diarrhoea had spontaneously subsided, and he remains well on a normal diet.

Case 2. A female infant who, within 48 hours of a breech delivery, developed a functional intestinal obstruction which necessitated a transverse colostomy. Colostomy actions were immediately copious and watery. An intolerance to oral lactose was found, and managed by a lactose-free regimen (Galactamin, Trufood) but the diarrhoea continued unabated. Repeated stool cultures failed to reveal any pathogen.

Three stool samples collected on successive days during the seventh week showed a progressive rise in the chloride ion concentration, which came to exceed the sum of the sodium and potassium ion concentrations (Fig. 3). The second sample showed a Cl^- concentration of 130 mEq/l. with Na^+ 112 mEq/l. and K^+ 14 mEq/l. In the third, Cl^- had risen to 137 mEq/l., with Na^+ 113 mEq/l. and K^+ 13 mEq/l. During the period of these analyses, the patient's clinical condition and serum electrolytes levels were adequately maintained on the oral Galactamin feeds, though she had previously, and subsequently, required intravenous fluid and electrolyte replacement.

The diarrhoea persisted until, during the fourth month, there was a further episode of acute intestinal obstruction. At laparotomy, dense peritoneal adhesions with chronically dilated loops of large and small bowel were found. She subsequently developed a jejunal fistula and died one week after operation. At necropsy no other abnormality was detected in the alimentary tract.

Case 3. A male infant who developed several episodes of abdominal distension and constipation during the first week of life. He was found to have Hirschsprung's disease, and at 2 weeks a sigmoid colostomy was fashioned. The colostomy actions rapidly became very watery and by the third post-operative day intravenous rehydration was necessary. An intolerance was found to have developed to both disaccharides and monosaccharides in the diet. No pathogen could be isolated from the stools. In spite of a complete withdrawal of all oral feeds and the maintenance of nutrition and hydration entirely by intravenous Aminosol Vitrum, Intralipid Vitrum, and electrolyte solutions, the diarrhoea continued unabated.

At this time, at 5 weeks of age, the first stool sample to be analysed showed the extraordinary Cl^- concentration of 243 mEq/l., with Na^+ 120 mEq/l. and K^+ 6 mEq/l. While on the same parenteral regimen, over the next 5 days there was an improvement in the diarrhoea when the stool Cl^- was found to have fallen to 108 mEq/l., with Na^+ 104 mEq/l. and K^+ 21 mEq/l. The third sample, 2 days later, showed this trend to be continued. Serum electrolytes were maintained throughout this period within the normal limit. The diarrhoea subsided over the next few weeks.

Over the next month, oral feeds were reintroduced, but sugars were not fully tolerated until 7 months of age. At this time, two further analyses were carried out which showed a normal electrolyte pattern. The patient has subsequently remained well and has undergone a resection of the aganglionic segment of bowel and closure of the colostomy uneventfully.

Discussion

Stool electrolytes are infrequently studied in diarrhoea. Reported observations show that chloride loss is usually low and is less than the sum of the sodium and potassium loss (Darrow and

Pratt, 1950); the results obtained in this survey confirm these observations. The values obtained from the control (non-diarrhoeal) group generally conform with earlier reports of stool electrolytes (Wilkinson, Stevens, and Hughes, 1962).

In cases of congenital chloride-losing diarrhoea with alkalosis previously reported, the stool chloride concentration exceeded the sum of the sodium and potassium ion concentrations irrespective of the oral intake (Evanson and Stanbury, 1965; Owen, 1964). The findings reported here in random stool samples are therefore considered to be indicative of an abnormal handling of chloride ion by the bowel. As balance studies had not been undertaken no conclusions could be drawn from the serum electrolyte or acid base studies in these patients.

The mechanism whereby excessive faecal loss of chloride occurs in congenital chloride-losing diarrhoea is not clearly understood. Potassium depletion appears to play a part, and chloride loss may be reduced as the alkalosis is corrected by replacing potassium (Evanson and Stanbury, 1965). The case of chloride-losing diarrhoea with alkalosis reported in an adult as a result of intestinal obstruction (Ariel, 1954) indicates that the condition may arise entirely as a secondary phenomenon, and be totally corrected by administering potassium. A similar occurrence has been observed in ulcerative colitis, and produced experimentally in rats by severe potassium depletion (Gardner *et al.*, 1949). The diminution in the amount of potassium lost in the stools as the chloride content rises (Fig. 3) suggests that here some relation also exists between these two ions. Though it would have been quite simple to see whether potassium replacement could abolish the abnormal chloride loss, the relatively little potassium appearing in ileostomy fluid, and the extraordinary amount of chloride found in the third patient, cast some doubt that this is the entire explanation.

It has been observed at The Hospital for Sick Children that after intestinal obstruction in infants the handling of sugars by the bowel frequently becomes abnormal (Howat and Aaronson, 1971). This observation is reflected in the 3 patients reported here. In the cases of chloride-losing diarrhoea reported by Owen (1964) and by Tucker *et al.* (1964), sugar intolerance was also noted and a corresponding enzyme

deficiency detected. It seems reasonable to speculate that the function of a mucosal enzyme system concerned in some way with chloride transport may become similarly impaired after an episode of intestinal obstruction and lead to a secondary chloride-losing diarrhoea. The evidence (Wilson, 1962) that an active transport system is concerned with chloride absorption lends some support to this possibility.

By whatever mechanism, it seems that the appearance of an excess of chloride in the stool may not be as uncommon as had hitherto been supposed, so that examination of stools for chloride, sodium, and potassium ion content may be relevant in investigating persistent diarrhoea in infancy. Pending further understanding of this group of patients, it appears wise to ensure adequate potassium replacement whenever excessive chloride loss is encountered.

I am grateful to Professor Barbara Clayton, for advice; to Mr. John Mitchell for assistance with the analysis; to the physicians and surgeons of The Hospital for Sick Children for co-operation; and to Professor O. H. Wolff for permission to report his patients. This work was carried out with the assistance of the Medical Research Council.

REFERENCES

- Ariel, I. M. (1954). Chloridorrhea: syndrome associated with diarrhea and potassium deficiency. *Archives of Surgery*, **68**, 105.
- Darrow, D. C. (1945). Congenital alkalosis with diarrhea. *Journal of Pediatrics*, **28**, 519.
- Darrow, D. C., and Pratt, E. L. (1950). Fluid therapy: relation to tissue composition and the expenditure of water and electrolyte. *Journal of the American Medical Association*, **143**, 365 and 432.
- Evanson, J. M., and Stanbury, S. W. (1965). Congenital chloridorrhea or so-called congenital alkalosis with diarrhoea. *Gut*, **6**, 29.
- Gamble, J. L., Fahey, K. R., Appleston, J., and MacLachlan, E. (1945). Congenital alkalosis with diarrhea. *Journal of Pediatrics*, **28**, 509.
- Gardner, L. I., MacLachlan, E. A., Terry, M. L., McArthur, J. W., and Butler, A. M. (1949). Chloride diarrhea and systemic alkalosis in potassium deficiency. *Federation Proceedings*, **8**, 201.
- Howat, J. M., and Aaronson, I. (1971). Sugar intolerance in neonatal surgery. *Journal of Paediatric Surgery (in the press)*.
- Owen, G. M. (1964). Metabolic alkalosis with diarrhea and chloride-free urine. *Journal of Pediatrics*, **65**, 849.
- Tucker, V. L., Wilmore, D., Kaiser, C. J., and Lauer, R. M. (1964). Chronic diarrhea and alkalosis. *Pediatrics*, **34**, 601.
- Wilkinson, A. W., Stevens, L. H., and Hughes, E. A. (1962). Metabolic changes in the newborn. *Lancet*, **1**, 983.
- Wilson, T. H. (1962). *Intestinal Absorption*. Saunders, Philadelphia.

Correspondence to Dr. Ian Aaronson, Department of Urology, Middlesex Hospital, London W.1.