Secondary Chloride-losing Diarrhoea

Observations on Stool Electrolytes in Infants after Bowel Surgery

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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.

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but in spite of their withdrawal the diarrhoea continued almost unremittingly 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
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![Graph showing concentration of Na⁺, K⁺, and Cl⁻ in random stool samples from 3 infants with excessive chloride loss.]

**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 0125 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 Hirschspring'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.

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References

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