

died, and at the Royal Hospital for Sick Children no case of perinatal splenic rupture has been admitted in the last 14 years.

Diagnosis is not easy in the infant who has had exchange transfusion as some abdominal distension is common after such a procedure. The diagnosis of haemoperitoneum in this patient was confirmed by paracentesis. Abdominal tap is of value when positive, but if negative is not always reliable. In this case the unusual feature was the low serum bilirubin even considering the effect of phototherapy. Presumably the large amount of the infant's blood which was in the peritoneal cavity was unavailable for breakdown and conversion to bilirubin.

The thrombocytopenia, fragmentation of RBC, consumption of factors, particularly I, V, VIII, and later probably VII, together with the release of fibrin degradation products (FDPs) was most likely due to the local stimulation of coagulation from thromboplastins released from the ruptured spleen. Though causing a further fall in platelets, the exchange transfusion with ACD blood seemed to halt the consumption of factors and to lower the level of FDPs. There may also have been an element of disseminated intravascular coagulation (Chessells and Wigglesworth, 1971) but hypotension and local coagulation were probably the major factors in this case. The coagulopathy with consumption of platelets and coagulation factors was probably responsible for the continuation of the haemorrhage, initiating a vicious circle (Digilio, Bacchetta, and Ferreri, 1960).

In the present case it was difficult to define when the suprarenal haemorrhages occurred. The hypotension and coagulopathy were probably important contributory factors.

Summary

A male infant of 36 weeks' gestation, weighing 3080 g, with erythroblastosis, ruptured spleen, and bilateral suprarenal haemorrhages is described. The infant survived after exchange transfusions and splenectomy.

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Hypertension, oedema, and suppressed renin aldosterone system due to unsupervised salt administration

Hypertension is recognized as a complication of incorrectly prepared hyperosmolar infant feeds, especially in the presence of acute diarrhoeal disease (Taitz and Byers, 1972). We report an infant who was given a glucose-salt-water mixture on medical advice and developed hypertension and oedema after being given an excess of salt.

Case report

A previously healthy, bottle-fed Asian girl, aged 3 months, developed diarrhoea. After 3 days the parents sought medical help and were advised to give her clear fluid feeds prepared by adding 5 teaspoons of glucose and 1 saltspoon of salt to 600 ml (20 fl oz) water. Having no saltspoon in the house, they prepared each feed by adding 2 teaspoons of glucose and 5 pinches of salt to 240 ml (8 fl oz) water. The baby drank eagerly one litre of the glucose saline solution every 24 hours. Urine output appeared normal and she had 5 loose stools a day. During the next 3 days she developed swelling of the face, abdomen, and legs, and was admitted to The Hospital for Sick Children.

She had gained 450 g in weight, had facial and leg oedema, and her blood pressure was 140/80 mmHg. Investigations showed plasma sodium 144 mEq/l, potassium 3.6 mEq/l, urea 5 mg/100 ml, urine osmolality 66 mOsm/l, and 24-hour urine sodium 20 mEq. Plasma creatinine and albumin were normal. Plasma renin activity (PRA) was subnormal on admission (55 pgAI/ml per h) and was associated with a low plasma aldosterone concentration (Paldo) of 11.9 ng/100 ml. Mean values for children of this age are 1392 (range 472-3130) pgAI/ml per h and 20.2 (range 8.3-75) ng/100 ml, respectively (Dillon and Ryness, 1974).

The feed which was made up at home was analysed and contained sodium 117 mEq/l, glucose 4 g/100 ml, and osmolality 450 mOsm/l. Thus, the sodium concentration was far higher than the 30 mEq/l intended.

The baby was treated by a restricted fluid intake of 100 ml/kg body weight per day given as a solution of 5% glucose in water orally with added potassium chloride. After 36 hours the oedema subsided and blood pressure was 90/60 mmHg. Plasma electrolytes remained normal, 24-hour urine sodium excretion fell to 5 mEq, PRA and PAldo rose to 1225 pgAI/ml per h and 22.8 ng/100 ml, respectively. She remained neurologically normal, was regraded onto milk, and has remained well.

Discussion

We report this case to emphasize how the unsupervised addition of salt to infant feeds of any type may give rise to a dangerously high intake of sodium. This baby's 24-hour sodium intake before admission was 117 mEq (about 25 mEq/kg) per day. She was excreting about 4.5 mEq/kg per day and thus was retaining about 15 mEq/kg per day. She had minimal diarrhoea at this time and stool losses of sodium would not be great. If gastrointestinal water losses had been greater, she might well have presented with hypertonic dehydration. Indeed, the latter condition has been reported in association not only with high solute milk feeding but with both commercial and 'home-made' oral electrolyte solutions prescribed for gastroenteritis when either the instructions have not been followed or excess salt added (Colle, Ayoub, and Raile, 1958; Franz and Segar, 1959).

Severe hypertension may well have been avoided by the development of oedema in our patient. The suppression of the renin aldosterone system in this infant, presumably by the exogenous salt and water load, is interesting. We are unaware of a similar observation in a child of this age. We suggest that this degree of suppression may be a factor in the development of hypernatraemia when abnormal water losses occur in infants fed on hyperosmolar feeds. The importance of the renin aldosterone system in control of sodium homeostasis is well established. If this system is fully suppressed by a high solute intake, there is no reserve capacity to excrete excess sodium.

In conclusion we wish to emphasize that in infants with mild diarrhoea there should be no need to give more than 5 mEq sodium/kg per day. Giving fifth normal saline with glucose at the rate of 150 ml/kg per day would provide the necessary sodium. Unless salt can be given accurately in small amounts, it may be safer to advise glucose and water only.

Summary

This paper describes an infant with gastroenteritis, who developed hypertension and oedema after administration of inaccurately prepared oral glucose salt solution. The renin aldosterone system was suppressed in this child and it was suggested that this may be a factor in the development of hypernatraemia when abnormal water losses occur in infants fed on hyperosmolar feeds. Unless salt can be given accurately in small amounts it may be safer to advise feeds of glucose only in infants with mild diarrhoea.

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Thymic dysplasia, persistence of measles virus, and unexpected infant death

After extensive post-mortem investigations of cases of 'cot death' or sudden infant death syndrome, a wide variety of pathological changes has been found, many of doubtful significance. Currently there has been an interest in the histological changes in the conducting tissue of the heart (Ferris, 1973). Unfortunately it is uncertain whether these changes are the cause or the effect of some cases of cot death. Virological studies have shown a greater incidence of isolation of viruses, particularly respiratory, from cot deaths when compared with controls (Ray *et al.*, 1970; Ferris *et al.*, 1973).