Severe Hyponatraemia in Hyperlipaemic Diabetic Ketosis

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The occurrence of spuriously low measured plasma electrolyte values in the presence of hyperlipaemia has been recognized for some time (Albrink et al., 1955). This is due to displacement of the plasma water by lipid so that the water content of a given volume of plasma is depressed below its normal value of 94-5%. By conventional flame photometry techniques the electrolyte content of a measured volume of plasma is determined irrespective of its water content. Therefore in the presence of high concentrations of a displacing substance there will be important and variable discrepancies between the measurements obtained and the true concentrations of electrolytes in the aqueous phase, which is what determines their biological effects. Measurement of osmolality by depression of freezing point is not influenced by the presence of displacing substances and reflects the concentration of solutes in the plasma water. It is recognized that uncontrolled diabetes, especially when of gradual onset, may be accompanied by severe hypertriglyceridaemia (Bagdade et al., 1967). We report here an extreme example of such a case with consequent complications in the management of fluid and electrolyte balance.

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Case Report

In 1969 the patient, a 35-year-old married housewife, presented with tiredness and urinary frequency. She was found to have a urinary infection and glycosuria, with a typical diabetic glucose tolerance curve. At that time she weighed 80 kg and she was started on a 130-g carbohydrate diet; however, she failed to gain and was referred for follow-up.

In 1971 she was admitted to hospital with a three-day history of malaise and an eight-hour history of vomiting, confusion, and breathlessness. She was stuporous and clinically dehydrated. The heart rate was 140/min with sinus rhythm, the blood pressure 140/100 mm Hg, and the extremities cold, with vasomotor disturbances. The respiratory system was normal apart from a respiratory rate of 40/min. The abdomen was generally tender and the liver edge was palpable. There were no localizing neurological signs, but her optic fundi showed intense lipaemia retinialis. There were no eruptive xan thomata.

Laboratory investigations on admission gave the following results: blood glucose 600 mg/100 ml; plasma sodium 86 mEq/l, potassium 2.2 mEq/l, bicarbonate 5 mEq/l, urea 40 mg/100 ml,
calcium 8·1 mg/100 ml, phosphate 1·0 mg/100 ml, creatinine 1·3 mg/100 ml, triglycerides 16,000 mg/100 ml (normal 60-160 mg/100 ml), cholesterol 1,200 mg/100 ml, acetone 47 mmol/l. (normal <0·5 mmol/l); serum aspartate aminotransferase 130 IU/l. (normal 25-65 IU/l); blood lactate 2·2 mmol/l. (normal 1·1-3 mmol/l); W.B.C. 20,600/mm³, platelet count normal; PaO₂ 102 mm Hg, PaCO₂ 16 mm Hg, blood pH 7·33. A haemoglobin estimation could not be performed because of the hyperlipaemia. Chest radiograph and electrocardiogram were both normal and microscopical examination of the urine showed scanty cellular casts. Two hours after admission plasma osmolality was 340 mosmol/kg H₂O (normal 285-295 mosmol/kg H₂O). At the same time evaporation to dryness of a known weight of plasma gave a value for plasma water of 71% by weight (normal 94-5%).

The patient was treated with intravenous fluids, potassium supplements, and repeated intravenous and intramuscular injections of insulin. In the first 24 hours a total of 16·5 l. of fluids was infused, of which 7·5 l. was normal saline, 6·0 l. was 5·0% dextrose, and 3·0 l. was 2·5% dextrose. A total of 450 mEq of potassium chloride was infused in the first 24 hours and the urine output was 6·0 l. Osmolality of a random specimen of urine after 20 hours was 355 mosmol/kg H₂O.

Although serial estimations of plasma triglycerides were not made in the acute phase, it would appear that as the triglyceride level fell so the values for plasma sodium and osmolality tended to return to normal. At a time (two hours after admission) when the plasma osmolality was 340 mosmol/kg H₂O the plasma sodium was only 96 mEq/l. 13 hours later the plasma osmolality had fallen to 316 mosmol/kg H₂O while the plasma sodium had risen to 114 mEq/l.

The patient recovered quickly from this acute ketotic episode. Within 24 hours she was fully conscious and the peripheral circula-

Comment

The patient is of interest from several viewpoints. She was severely acidic, with a base deficit of 16 mEq bicarbonate/l. and moderately ketonaemic, with a plasma acetone level of 47 mmol/l. It has previously been reported that severe hyperlipaemia with hyperglycaemia is infrequently accompanied by ketoacidosis (Bagdade et al., 1967). It is suggested that there is usually sufficient circulating insulin to inhibit uncontrolled lipolysis, thus preventing ketoacidosis.

The initial rise in plasma sodium in our patient occurred concurrently with the period of saline infusion. At an early stage calculations based on the plasma water content indicated that the patient was in danger of becoming hypernatraemic. When the measured plasma sodium was 96 mEq/l and the plasma water 71% calculation gave a value for sodium of 136 mEq/l plasma water. The saline infusion was therefore discontinued when the measured plasma sodium had reached 104 mEq/l. After the saline infusion was stopped and dextrose infusion substituted, the plasma sodium continued to rise and the plasma osmolality fell. Over 22 hours the plasma osmolality fell from 340 mosmol/kg H₂O to 300 mosmol/kg H₂O, while during the same period the blood glucose had fallen only from 400 mg/100 ml to 350 mg/100 ml. Thus the fall in osmolality cannot be explained in terms of a falling blood glucose level.

The repeated estimation of plasma water content is time-consuming, and our experience with this patient indicates the value of plasma osmolality in assessing acute electrolyte disturbances in the presence of severe hyperlipaemia.

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References
