

primary hypertrophy of the muscular layers of the pulmonary arterioles with extension of the muscle layer into the normally non-muscular peripheral arteries has been reported.⁴

The three siblings described presented with cyanosis and respiratory distress soon after birth. Both clinical examination and heart catheterisation showed evidence of pulmonary arterial hypertension with right to left shunting. There was no history of intrauterine exposure to aspirin, indomethacin, or other prostaglandin synthetase inhibitors, nor was there evidence of intrauterine hypoxia, perinatal asphyxia, parenchymal lung disease, myocardial dysfunction, or metabolic abnormalities.

Histological examination of the lung of the three infants showed an appreciable increase in pulmonary arterial smooth muscle and decreased lumen size. The nature and severity of the structural remodelling of the pulmonary arterial bed in these infants suggests that the underlying pathological process was initiated in utero, and that the observed alterations could not have been solely the result of postnatal development. We believe that the structural pulmonary vascular abnormalities described could explain the intractable pulmonary hypertension and the lack of response to medical treatment in the three infants.

None of the clinical conditions commonly associated with persistent pulmonary hypertension of newborn were present in any of the infants and the underlying cause of the development of new muscle

in the walls of the small, normally non-muscular, intra-acinar arteries remains unknown. The parents of these three neonates are first degree cousins and have two other healthy children. This suggests that persistent pulmonary hypertension of newborn due to abnormal muscularisation of the small pulmonary arteries may be a recessively inherited genetic trait.

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Hyponatraemia in diabetes without ketoacidosis

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SUMMARY At diagnosis six of 18 diabetic children had hyponatraemia with hyperglycaemia but no signs of dehydration or lipaemia. With insulin treatment alone plasma sodium concentrations in two children returned to normal. These children do not require specific treatment to correct the hyponatraemia.

Hyponatraemia is a common finding in both inpatient and outpatient hospital populations.¹ Causes include long term diuretic treatment, renal failure, use of hypotonic intravenous infusion, inappropriate antidiuretic hormone secretion, and

hyperglycaemia.² The latter is a constant feature in the child with newly diagnosed or poorly controlled diabetes. Hyponatraemia may be present when the child is ketoacidotic and severely dehydrated owing to salt and water depletion.³ In the absence of these signs, however, the frequency of hyponatraemia in the diabetic child at diagnosis is unknown.

This study reports a retrospective analysis of the frequency of hyponatraemia in a group of diabetic children at diagnosis, in relation to the presence or absence of ketoacidosis and dehydration.

Patients and methods

The medical records of 18 diabetic children admitted

at the time of diagnosis to this paediatric unit over a two year period were studied. The children's ages at diagnosis ranged from 6 months to 14 years. The presence or absence of dehydration assessed by standard clinical criteria was noted.

Plasma urea, electrolytes, and glucose concentrations were determined by standard methods using a Technicon SMA 6-60 and a single channel Technicon analyser. Each plasma sample was visually examined for gross lipaemia. Ketoacidosis was based on ketonuria, decreased arterial blood pH, and plasma bicarbonate.

Results

Plasma glucose concentrations were greater than 11 mmol/l in all 18 children (range 17.0 to 57.2 mmol/l). Ketoacidosis was present in eight children, six of whom were hyponatraemic and had a plasma sodium concentration of less than 133 mmol/l. Two children had normal plasma sodium concentrations and were well hydrated; a further two children who were well did not have plasma sodium measurements performed.

The remaining six children presented without evidence of ketoacidosis or dehydration but had plasma sodium concentrations of less than 133 mmol/l in the absence of lipaemia. Plasma potassium concentrations were normal. The details are summarised in the Table. Plasma glucose concentrations were noticeably raised. None received intravenous saline and insulin was administered every six hours by subcutaneous injection. All six children were clinically well at diagnosis and repeat measurements of plasma sodium concentrations were only available in two children. In both cases, plasma sodium concentrations had returned to normal within 16 hours of insulin treatment alone.

Table Plasma sodium, glucose, and urea concentrations in six diabetic children who showed no evidence of dehydration or ketoacidosis

Case No	Sodium (mmol/l)	Glucose (mmol/l)	Urea (mmol/l)
1	127 (140)	32.5 (10.0)	6.6 (5.4)
2	130 (140)	21.8 (11.1)	4.0
3	130	28.4	6.6
4	128	20.8	5.7
5	130	33.6	5.9
6	129	17.0	5.7
Normal range	133-144	—	2.5-7.5

The values in parentheses represent repeat estimations after 16 hours of insulin treatment.

Discussion

The association between hyperglycaemia and hyponatraemia is well recognised.⁴ Plasma sodium concentrations are higher in diabetic patients who are adequately controlled than in those who are poorly controlled.⁵ In a recent survey of a large number of biochemical results obtained in a routine laboratory, hyperglycaemia was more common in hyponatraemic patients than in patients with a normal plasma sodium concentration.²

Twelve of 16 diabetic children in this study in whom plasma sodium was measured at diagnosis had hyponatraemia. Appreciable hyperlipidaemia was a contributory factor in causing a spurious decrease in sodium concentration in three children.⁶ Six children, however, had a true decrease in plasma sodium concentration unassociated with ketoacidosis, dehydration, or lipaemia. All had had considerable polydipsia due to increased thirst, for a period of two days to four weeks before diagnosis.

Hyperglycaemia promotes both an osmotic diuresis and an osmotic shift of water from within the cells to the extracellular fluid.^{1,4} This in turn decreases the plasma sodium concentration by dilution. It is likely that the large fluid intake by these six children was sufficient to prevent appreciable dehydration thus minimising the osmotic shift of water. Presumably, this explains why the children seemed clinically well at diagnosis, despite both hyperglycaemia and hyponatraemia.

Hyponatraemia in the diabetic child at diagnosis in the absence of signs of either dehydration or ketonuria seems relatively common. Insulin treatment should be started but since these children are neither dehydrated nor vomiting, an intravenous saline solution is not required.

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