BJOG: an International Journal of Obstetrics and Gynaecology
September 2004, Vol. 111, pp. 1020–1022
DOI: 10.1111/j.1471-0528.2004.00237.x

CASE REPORT

Severe hyponatraemia and pre-eclampsia

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Case report

A 31 year old primigravida booked at 15 weeks with blood pressure of 110/60 mmHg and no significant medical history. Fetal growth scans at 20, 24, 28 and 30 weeks showed symmetrical growth restriction. She was admitted at 31 weeks with elevated blood pressure (130/100 mmHg) and proteinuria (2+). Routine haematological and biochemical screens for pre-eclampsia showed an elevated urate level of 0.38 mmol/L. Ultrasound assessment of the fetus showed abdominal and head circumferences on the third centiles and oligohydramnios but normal middle cerebral and umbilical artery Doppler flow tracings. She was given intramuscular betamethasone 12 mg × 2 doses to accelerate fetal lung maturity.

At 32 weeks, her blood pressure fluctuated between 160/100 and 180/110 mmHg and urinalysis showed 2,050 mL urine volume with 160 mg of protein in 24 hours. She developed headaches and subsequent biochemical analysis showed hyponatraemia with serum sodium level of 128 mmol/L (Table 1). Fundoscopy was normal. However, she rapidly developed progressive oedema, epigastric pain, hypereflexia and significant proteinuria (310 mg/24 hours). Antihypertensive treatment was initiated with Labetolol 200 mg qds, methyldopa 750 mg qds and nifedipine 10 mg daily. The serum sodium levels fell steadily to 119 mmol/L over the next 20 days, and the urine sodium and potassium levels were less than 20 mmol/L. The urine and plasma osmolality were 348 and 256 mmol/L, respectively. Thyroid function tests, electrocardiogram and fetal cardiotocograph were all normal.

She was delivered by caesarean section at 33 weeks of gestation because of deteriorating hyponatraemia, worsening symptoms of pre-eclampsia and fetal growth restriction. A female infant was delivered weighing 1.53 kg with the following cord blood gas analysis results: venous pH 7.22, p_aO_2 4.8, BE −7.1 and arterial pH 7.32, p_aO_2 6.5, BE 0.9. The baby had hyponatraemia with serum sodium of 125 mmol/L, which resolved spontaneously within 72 hours in the special care unit. Within six hours of delivery, the maternal hyponatraemia had improved to 132 mmol/L. Postnatal chest X-ray was normal. The histology of the placenta after delivery showed excess syncitial knots in the placental villi with accelerated maturation, but there was no villitis, funisitis or chorioamnionitis. She was discharged 15 days postpartum, and the baby at 29 days old. Her blood pressure and blood biochemistry were normal at four weeks postpartum and all antihypertensive drugs were discontinued.

Discussion

This case report showed severe hyponatraemia complicating pre-eclampsia in a patient with normal urine sodium and potassium excretion and urine osmolality but with decreased plasma osmolality. Hyponatraemia can be classified according to the presence of hypovolaemia, normovolaemia or hypervolaemia.[1]

Hyponatraemia with hypovolaemia (deficit of total body water or large deficit of extracellular sodium) can be as a result of renal or non-renal causes. Renal causes are associated with urinary sodium concentration of >20 mmol/L. These include mineralocorticoid deficiency, sodium-losing renal disorders and diuretic excess. Non-renal causes are associated with urinary sodium concentration of <10 mmol/L. These include profuse vomiting, prolonged diarrhoea, extensive burns and excessive sweating. Hyponatraemia with normovolaemia (normal or slight excess of total body water) are associated with urinary sodium concentration of >20 mol/L. This includes glucocorticoid deficiency, hypothyroidism, syndrome of inappropriate antidiuretic hormone secretion (SIADH) and inappropriate intravenous fluid therapy (such as 5% dextrose). Hyponatraemia with hypervolaemia (excess of extracellular sodium or larger excess of total body water) can be as a result of renal or non-renal causes. Renal causes are associated with urinary sodium concentration of >20 mmol/L. These include acute and chronic renal failure. Non-renal causes are associated with urinary sodium concentration of <10 mmol/L.

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Table 1. Blood investigation results.

	Day								
	1 (admission)	3	5	8	15	20 (delivery)	21	23	35 (discharge)
Na, mmol/L (130–140)	137	132	128	128	120	119	132	133	135
K, mmol/L (3.3–4.1)	3.8	3.7	4	4.1	3.8	3.8	3.6	4.1	4
Urea, mmol/L (2.4–3.8)	3.1	2.9	3	3.9	3.4	4	5.5	3.1	3.4
Cr, μmol/L (55–73)	67	82	73	85	72	79	75	82	80
Bilirubin, μmol/L (3–14)	10	11	8	8	10	7	8	8	10
Alkaline pH, iu/L (133–418)	95	118	139	112	120	122	130	132	134
Aspartate aminotransferase, iu/L (11–30)	27	28	29	29	35	38	37	36	30
Albumin, g/L (28–37)	26	24	23	23	23	23	26	25	25
Urate, mmol/L (0.21–0.38)	0.38	0.38	0.39	0.38	0.4	0.38	0.38	0.38	0.38
Hb, g/dL (11–14)	12.2	12	11.9	11.8	11.6	11.1	10.8	10.7	10.7
Platelets, $\times 10^9$ L (150–400)	249	292	265	254	276	244	250	238	239

From Refs. [7, 8].

These include cardiac failure, cirrhosis, nephrotic syndrome, inappropriate intravenous fluid therapy (such as normal saline). Drugs including tricyclic antidepressants, opiates, dopamine antagonists, diuretics, selective serotinin re-uptake inhibitors, chlorpropamide and chemotherapy agents (vincristine and cisplatin) have been reported to cause hyponatraemia, but these were ruled out in this case.

We postulate that in this case there was hyponatraemia with hypervolaemia (excess extracellular sodium and total body water) as a result of a non-renal cause and subsequent low urinary sodium of <20 mmol/L. Although it is possible to classify her hyponatraemia in this way, we still do not know the underlying mechanism and why she developed hyponatraemia when most women with pre-eclampsia do not. There was no significant medical history of thyroid, cardiac, liver, adrenal or renal failure. Nephrotic syndrome was excluded as a result of the mild proteinuria (310 mg/24 hours). SIADH was discounted because of the low urinary sodium (<20 mmol/L), normal urine osmolality and significant oedema. The criteria for the diagnosis of SIADH includes dilutional hyponatraemia, urinary osmolality greater than the plasma osmolality, renal sodium excretion 50–70 mmol/L, normal renal, thyroid and adrenal function and the absence of hypotension, hypovolaemia and oedema-forming states.[1] In this case, the blood investigations showed no other electrolyte or biochemical imbalance in spite of profound deteriorating hyponatraemia.

Previous published case reports have described dilutional hyponatraemia in pre-eclampsia with and without nephrotic syndrome[2,3] and transient SIADH in pregnancy.[4] Only one case report showed maternal HELLP syndrome and neonatal hyponatraemia.[5] Review of the literature suggests that this is the only case report that documents neonatal hyponatraemia with pre-eclampsia in the absence of HELLP syndrome. Clinical features of hyponatraemia include headache, lethargy, muscle cramps, confusion, convulsion and coma. Their occurrence depends on the absolute sodium concentration and its rate of fall. Note that hyponatraemia can precipitate maternal seizures, which can be difficult to differentiate from eclampsia. Death can occur in up to 50% of patients in whom the serum sodium concentration falls below 120 mmol/L within a period of 24 hours.[6] Care is necessary if hyponatraemia is treated rather than allowed to correct itself spontaneously. The suggested rate of correction of hyponatraemia is 2 mmol/L/h.[6] Neonatal hyponatraemia can cause jaundice, tachypnea and seizures if the serum sodium falls below 130 mmol/L.

In this case, the maternal and neonatal hyponatraemia resolved spontaneously within 24 and 72 hours, respectively, with no residual effects. We draw attention to severe hyponatraemia as a rare indication for urgent delivery in patients with pre-eclampsia. This requires multidisciplinary management and continuing care postpartum of both the mother and baby to ensure a good outcome.

Acknowledgements

The authors would like to thank Dr J. Milton for the assistance in the preparation of the table of results.

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Accepted 27 March 2004