Dialysis for severe hyponatraemia in preeclampsia

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Summary: Severe hyponatraemia is a rare complication of preeclampsia. In the case presented, the rapid recovery of liver function test abnormalities and thrombocytopenia were accompanied by acute renal failure, relative oliguria and progressive hyponatraemia contributing to confusion and ileus. Dialysis was instigated and the patient promptly recovered. Renal function recovered fully.

Keywords: hyponatraemia, preeclampsia dialysis, pregnancy

INTRODUCTION

Hyponatraemia in the setting of preeclampsia remains a rare but life-threatening complication with six published cases.^{1–6} The cause is variably attributed to dilutional hyponatraemia^{7–9} or the syndrome of inappropriate antidiuretic hormone (ADH).³ We describe briefly a case seen at Royal Prince Alfred Hospital for Women and Babies, Sydney, Australia where progressive hyponatraemia with neurological signs occurred despite urgent delivery for preeclampsia. Ultimately, dialysis was required to correct severe hyponatraemia.

CASE REPORT

The case was a 34-year-old woman who had an emergency caesarean section for fulminant preeclampsia and breech presentation at 30 weeks. She was a primipara with a prior medical history of treated hypothyroidism, and early pregnancy blood pressure readings of 110/70 mmHg. At 26 weeks she was noted to have intrauterine growth restriction on fundal height estimation and Doppler ultrasound (third centile estimated fetal weight). She had an increase in blood pressure (140/100 mmHg) and was treated with low-dose clonidine with good blood pressure control. No intravenous fluids were administered. There was an increase in transaminases (alanine transaminase [ALT] 304 mmol/L, and aspartate aminotransferase [AST] 146 mmol/L) and intramuscular betamethasone was given to enhance fetal lung maturity. After 48 hours in hospital around the time of planned delivery, she suddenly developed severe right upper quadrant pain, liver distention and hypertension 170/100 mmHg. Her serum glucose concentration was normal (minimum 4.9 mmol/L) and her coagulation profile showed an abnormal international normalized ratio (1.8 max) within the next 24 hours only. She had no vomiting. She was treated with undiluted magnesium sulphate, intravenous

sodium nitroprusside and urgent delivery. A female infant weighing 900 g with APGAR scores of 6 at one minute and 8 at five minutes was born.

She initially progressed well with a resolution of the marked rise in her transaminases (peak ALT 2800–1437 mmol/L and AST 3920–1441 mmol/L) within the next three days. Her platelet count reached a nadir of $33 \times 10^9/L$ on the morning after delivery and subsequently recovered. Over the next five days, she developed progressive hyponatraemia with a serum sodium reduced to 113 mmol/L, associated with confusion, muscular irritability and hyperreflexia. She was mildly hypokalaemic, 3.0 mmol/L, and her creatinine rose from 58 to 496 $\mu\text{mol/L}$. Her serum albumin concentration was 28 g/L on day 2 postdelivery. Normal pituitary function was confirmed with a high normal thyroid-stimulating hormone on day 2 (3.212 mIU/L; reference range 0.5–4.0 mIU/L). She was managed with a maximum of two litres of normal saline per day in the first 48 hours postdelivery and had free access to oral fluids until day 2 when fluids were restricted to 1000 mL initially and then 600 mL per day. Due to the risk of a postpartum seizure, she was again treated with intravenous undiluted magnesium sulphate, but this was ceased when she rapidly developed a pseudo-ileus requiring decompression with a nasogastric tube. Despite reasonable urine output 80–190 mL per hour, she remained very oedematous, and ultimately ultrafiltration by haemodialysis was required to increase her free water clearance and correct the severe hyponatraemia. On day 6 she increased her urine output spontaneously to 600 mL per hour and her electrolyte abnormalities and hypertension slowly resolved over the next few days. She completed two, four-hour hemodialysis sessions via an internal jugular double lumen vascular catheter.

DISCUSSION

This case demonstrates the sometimes severe nature of hyponatraemia, which may complicate the already difficult management of the pre-eclamptic patient. This patient developed a rapid deterioration in liver function tests in the setting of a rapid rise in blood pressure with normal blood glucose and

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no vomiting, suggesting preeclampsia rather than acute fatty liver of pregnancy. As with other complications of preeclampsia, delivery does not always provide immediate full recovery and management of the ongoing morbidity becomes problematic. She developed a progressive renal failure and severe hyponatraemia despite a strategy of minimal intravenous fluid that typifies the general management strategy at this institution. The importance of careful fluid balance cannot be over-emphasized in these patients. Central venous pressure monitoring was not used, but the sodium concentration was measured every 6–8 hours with a correction rate of 0.5 mmol/L per hour in the first 24 hours and then 0.33 mmol/L per hour in the next 24 hours. The slow rate of correction was used to prevent the risk of central pontine myelinolysis. The use of ultrafiltration with rapid recovery confirmed the mechanism of increased total body water. The use of intravenous loop diuretic such as frusemide was considered, but given the rapidly progressive confusion, the option of closer control of hyponatraemia via ultrafiltration was chosen. These patients have an inappropriate antidiuretic hormone (ADH) response in the setting of progressive hyponatraemia. The mechanism of this aberrant ADH response remains unclear but the apparent activation of ADH in response to decreased plasma volume (due to endothelial leak) remains a rare but life-threatening complication of severe preeclampsia.¹⁰ Other contributors identified in the literature include water intoxication^{11,12} and sodium depletion,¹³ although these mechanisms were not relevant in this case. The management of hyponatraemia in the postpartum state needs to take account of the lower serum sodium concentration (~ 5 mmol/L) in pregnancy¹⁴ with a lower serum osmolality and heightened ADH response. The normal postpartum diuresis requires an appropriate atrial natriuretic peptide response and a normal renal response to effect the necessary diuresis, especially in those patients fluid-overloaded from preeclampsia. The

investigation of persistent hyponatraemia requires an assessment of fluid balance, renal function and eventually ADH response if ongoing. The extreme hepatic pain experienced in this particular case, as well as the hepatic dysfunction, may well have been a contributor to the profound hyponatraemia.

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