Bradford Teaching Hospitals NHS
NHS Foundation Trust

Pre-eclampsia as a rare cause of severe hyponatraemia

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Introduction

Hyponatraemia is a common electrolyte abnormality with multifactorial aetiology. It is associated with significant complications and carries a mortality rate of above 50% when plasma sodium concentration falls below 115 mmol/L.

Pre-eclampsia toxaemia (PET) is a multisystem disorder that affects 2% to 5% of pregnancies and is responsible for up to 18% of maternal mortality.[1] Classically, it is defined as hypertension and proteinuria (≥0.3g over 24 hours), with onset after 20 weeks gestation. It may also lower the threshold for seizures and predispose to foetal damage.

Severe hyponatraemia is a very rare, fatal complication of PET and has been described in only a few patients in the literature to-date. Here, we present a case of severe hyponatraemia complicating PET in a primiparous woman which resolved promptly postpartum.

Case Report

A 23-year old healthy primigravida was admitted at 34+6 weeks' gestation following an episode of reduced foetal movements. She was hypertensive (BP 171/98 mmHg) and had 2+ proteinuria. Her sodium level was 133 mmol/L (NR: 135-145mmol/L) and urine protein-creatinine ratio was 229 mg/mmol (NR: 0-15). Cardiotocography was unremarkable. She was diagnosed with pre-eclampsia and started on labetalol and aspirin. She was discharged two days later with adequate BP control.

She was recruited into the PHOENIX trial, a multi-centre trial comparing outcomes between early induction (34–36+6weeks) versus expectant management (delivery: 37 weeks) in pre-eclampsia. She was randomised to the early induction arm and received IM betamethasone at 35+4 weeks' gestation. Serum sodium dropped to 126 mmol/L and two days later, reached a nadir of 114 mmol/L at 35+6/40. She had now developed marked oedema and was admitted for further investigations, as outlined in Table 1.

Table 1: Summary of investigations as at 35+6 weeks gestation

Test	Value	Reference Range
Urea and Electrolytes		
Sodium	114* mmol/L	135-145 mmol/L
Potassium	3.8 mmol/L	3.5-5.0 mmol/L
Urea	8.3* mmol/L	2.5-7.8 mmol/L
Creatinine	62 umol/L	49-90 umol/L
Liver Function Tests		
ALT	721* iu/L	<40 iu/L
Bilirubin	21 umol/L	2-21 umol/L
Alkaline Phosphatase	171* iu/L	30-130 iu/L
Albumin	21* g/L	35-50 g/L
Thyroid Function Tests		
Free T4	11.9 pmol/L	10-20 pmol/L
TSH	1.6 miu/L	0.2-4.0 miu/L
Full Blood Count		
Haemoglobin	122 g/L	115-160 g/L
White Blood Cells	12.0 x 10^9/L	4-11 x 10^9/L
Platelets	248 x 10^9/L	150-400 x10^9/L
Random cortisol	75 nmol/L (post IM betamethasone)	150-600 nmol/L
Urine		
Urine osmolality	445 mosm/kg	
Urine sodium	< 10 mmol/L	
Serum osmolality	255* mosm/kg	275-295 mOsm/kg

Isotonic sodium chloride was carefully administered. She was delivered by caesarean section at 36+1 weeks because of persistent hyponatraemia and worsening symptoms of pre-eclampsia as well as suspected acute fatty liver (ALT 1348 iu/L; NR <40 iu/L).

A male infant was born (Apgar score 9 at 10 minutes) – he had mild hyponatraemia – corrected by the paediatricians. Within 24 hours of delivery, maternal hyponatraemia had improved to 133 mmol/L. This is illustrated in Figure 1.

Recovery was complicated by intrapartum sepsis. She was discharged eight days later with a normal BP.

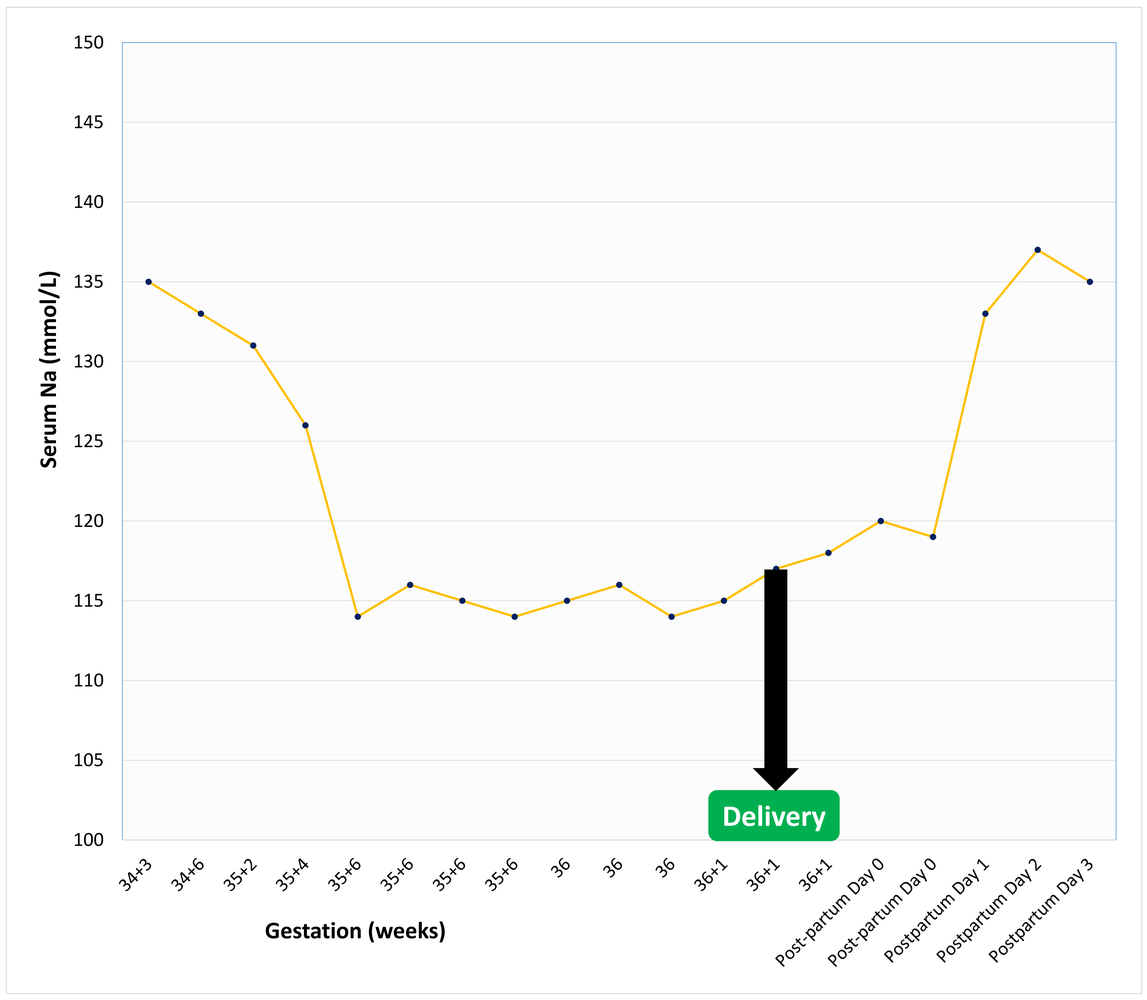

Figure 1: Variation in serum sodium level (NR: 135-145 mmol/L) during admission and post-partum. There is a net improvement in serum sodium level after delivery at 36+1 weeks gestation.

Discussion

Pregnancy involves physiological changes affecting water/ sodium homeostasis. However, most women with PET do not develop hyponatraemia. A recent review of 332 pregnancies complicated by PET found hyponatraemia to occur more frequently in older age and twin gestations;[2] both features were absent in our patient. However, she had features of severe pre-eclampsia, including uncontrolled hypertension and impaired hepatic function.

We postulate that this was a case of hyponatraemia with hypervolaemia (excess extracellular sodium and total body water) as a result of impaired free water clearance secondary to pre-eclampsia. SIADH was discounted because of low urinary sodium and oedema.

We draw attention to severe hyponatraemia as a biomarker of severe pre-eclampsia and as a rare indication for urgent delivery. This requires multidisciplinary management and continuing postpartum care to ensure favourable maternal/ neonatal outcomes.

References:

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2. Razavi A.S., Chasen S.T., Gyawali R. et al. (2017) Hyponatraemia associated with pre-eclampsia. J. Perinat. Med., 45(4): 467-470

BES2017 EP-048
Clinical biochemistry
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Poster presented at: SfE BES 2017 Harrogate
PosterSessionOnline.com