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Pre-eclampsia related hyponatremia, a new challenge in obstetrics practice: A case report

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Background

The female body undergoes a series of physiological changes during pregnancy to support fetal and maternal well-being. An increase in the hormones **estrogen and relaxin** generates systemic vasodilation, serving as a **non-osmotic stimulus for ADH**. The placenta releases **vasopressinase**, degrading ADH and thus preventing hyponatremia. In pre-eclampsia (PE) the **defective placenta** fails to properly release vasopressinase, increasing the risk of hyponatremia.

Case Presentation

We present a case of a 38-year-old, para 3 woman, who presented with **non-severe PE** and early type I of Barcelona **intrauterine growth restriction (IUGR)** at 27 weeks of gestation.

At 28 weeks, she was found to have **moderate hyponatremia of 129 and 126mEq/L** on repeat testing.

Further studies revealed plasma osmolarity in 260mOsm/kg (low) and urinary sodium in 54mEq/L (high), findings suggestive of **syndrome of inappropriate secretion of ADH (SIADH)**.

A **water intake restriction** regimen of 500ml/day was initiated and her natremia normalized shortly thereafter. (Figure 1)

The **IUGR** progressed to type II at 30 weeks. A caesarean section was performed at 32 weeks. A live 1420g female newborn was delivered with a 7-8 APGAR score, and normal serum sodium at birth. She required **neonatal intensive unit care** given her **weight and several comorbidities** such as hyperbilirubinemia, intraventricular hemorrhage, swallowing disorder and several metabolic imbalances.

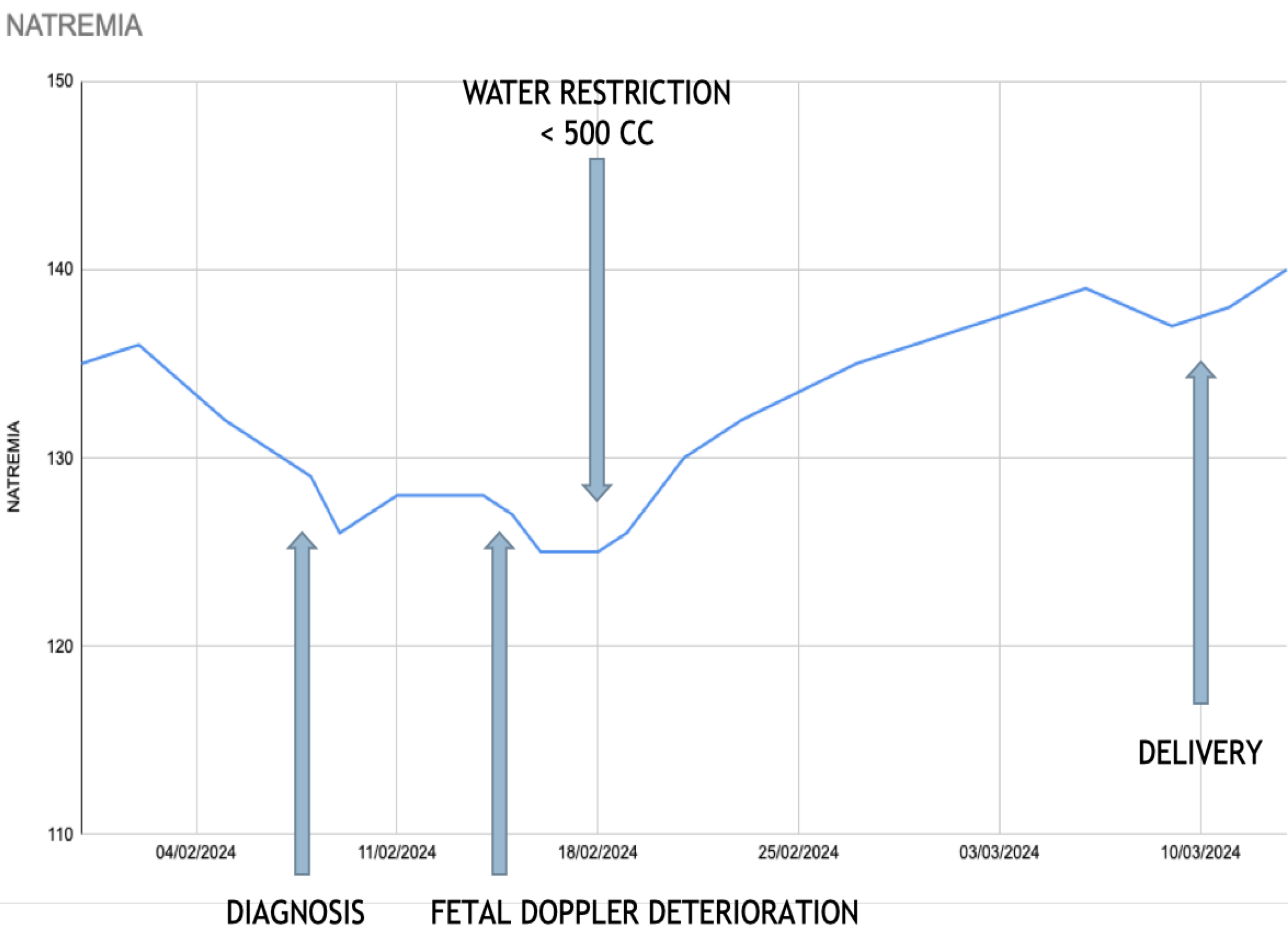


Figure 1. Timeline of natremia related to key events in patient’s management.

Conclusions

Hyponatremia secondary to SIADH and severe PE and IUGR has been previously reported, leading to adverse perinatal outcomes. This new association poses diagnostic and management challenges for the obstetrician.