Hyperkalemia and hyponatraemia associated with severe pre-eclampsia: a case report

To discuss a rare case of pre-eclampsia associated with hyperkalaemia and hyponatraemia, leading to premature delivery

**Objective**

**Literature Review**

- Pre-eclampsia (PET) is a multisystemic disorder complicating 2-8% of pregnancies and accounting for 2.5% direct maternal deaths. It is defined as gestational hypertension (≥140/90mmHg on two separate occasions thirty minutes apart), associated with proteinuria (≥300mg per 24 hours) after 20 weeks gestation.
- Hyponatraemia, defined as a serum sodium concentration below 135mmol/L, has been described as a rare complicating factor in severely pre-eclamptic patients.
- Severe hyponatremia, (sodium <125mmol/L) is associated with seizures, cerebral oedema and death.
- Hyperkalaemia affects up to 10% of pre-eclamptic pregnancies and is associated with disease severity, twin pregnancy and maternal age.
- In a retrospective review, hyponatraemia (sodium <130mmol/L) was noted in 9.7% of 332 pre-eclamptic pregnancies in one year. Postnatally hyponatraemia has resolved in all cases described in the literature to date excepting a case with fulminant hepatic failure requiring dialysis to resolve.

**Case Report**

- A 24-year-old primiparous woman of low obstetric risk at booking developed abnormal uterine artery Dopplers at 20 weeks, intrauterine growth restriction at 24 weeks and severe early-onset pre-eclampsia at 27 weeks of gestation.
- From 28 weeks of gestation she deteriorated clinically with signs and symptoms of pre-eclampsia. Firstly, she developed progressive hyponatraemia of up to 119mmol/L, which was refractory to treatment with fluid restriction, intravenous sodium chloride and sodium bicarbonate.
- The onset of hyperkalaemia developed in parallel (up to 6 mmol/L) and was associated with the severity of her hyponatraemia.
- Delivery via caesarean section was performed and intravenous hydrocortisone was administered for 24 hours post-delivery.
- Postnatally, her electrolytes and blood pressure resumed to pre-pregnancy levels. Addison’s disease was excluded.
- Hyponatraemia and hyperkalaemia resolved postnatally.

**Discussion**

- Hyponatraemia in pregnancy is most commonly caused by excess ADH as in the following mechanisms:
  1. True volume depletion secondary to increased fluid losses prompts ADH release.
  2. Reduced tissue perfusion, either due to a reduced circulating volume or systemic vasodilation prompts ADH release.
  3. Primary ADH release in siADH
  4. Reduced ADH release by vasopressinase.
- Hyperkalemia is caused by:
  - Impaired renal elimination of potassium
  - Increased potassium shift from cells.
  - Medication-induced e.g diuretics
  - PET associated hyperkalaemia has been associated with the use of both intravenous and oral labetalol in non-PET patient (6).
  - There is an association between pre-eclamptic hyponatraemia and severity of PET, twin pregnancy, advanced maternal age, in vitro fertilization conception and HELLP (hemolysis, elevated liver enzymes, low platelets) syndrome (4).

**Conclusion**

- We describe a rare case of pre-eclampsia associated with severe hyponatraemia and hyperkalaemia.
- The mechanisms accounting for this are hypothetical but include oral labetalol therapy and mildly impaired renal function.
- Although case reports have described hyponatraemia with pre-eclampsia, most likely secondary to increased anti-diuretic hormone release, none of these cases have described co-existing hyperkalaemia.
- Therefore this is the first case of its kind to our knowledge.

**Take home message**

- Hyponatraemia complicates approximately 10% of pre-eclamptic pregnancies.
- Hyponatraemic pre-eclampsia is associated with more severe pre-eclampsia, HELLP syndrome, advanced maternal age, twin pregnancy and in-vitro fertilization.
- All patients with PET should be monitored for the development of electrolyte imbalance including hyponatraemia and hyperkalaemia.
- Delivery is warranted in cases of hyponatraemia and hyperkalaemia with PET refractory to replacement therapy.

**References**