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The Association of Chronic Hyponatraemia with Severe Preeclampsia and Eclampsia in A Type 1 DM Patient – Case Report and Literature Review

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Abstract

Hyponatraemia in pregnancy (defined as serum sodium < 130 mmol/L) is not uncommon in the presence of preeclampsia (hyponatraemia is reported in about 9% of cases), even more so in those with preeclampsia with severe features and twin gestations.

It is regarded more as a complication of preeclampsia and in the reported cases patients were not diagnosed with chronic hyponatraemia from the first trimester. Chronic hyponatraemia diagnosed at the beginning of the pregnancy is a rare condition.

We report a case of chronic unexplained hyponatraemia diagnosed in the first trimester of the pregnancy that was later complicated by severe preeclampsia and eclampsia, with a background of type 1 diabetes and hypothyroidism. This is a rare situation and we want to highlight the high risk of patients with type 1 diabetes and chronic hyponatraemia to develop severe preeclampsia and eclamptic seizures during the pregnancy.

Keywords: Hyponatraemia; Preeclampsia; Type 1 Diabetes Mellitus; Eclampsia

Case Report

We present the case of a 37year old patient, BMI 23 with moderate chronic hyponatremia (Na 128 mmol/l) diagnosed at 9 weeks gestation. She was booked in our unit and she had all the antenatal care and the delivery in the same hospital. We collected the data from her digital notes and our laboratory results.

The endocrinology team didn`t find a specific cause for her chronic hyponatraemia. She was asymptomatic but she would occasionally drink more than 2l (up to 4l) of water per day. The serum osmolarity, urinary sodium and urinary osmolality were normal and the synacten test was normal as well. She was treated with fluid restriction and the serum sodium (Na) levels were quite stable at around 130 mmol/l during the pregnancy with fluid restriction only. She was advised to not exceed 1.5l of fluids per day.

She had a background of type 1 diabetes on insulin pump and hypothyroidism well controlled with levothyroxine. She was never diagnosed with hyponatremia before pregnancy.

At 31weeks gestation she required increased doses of insulin in order to control her blood sugar levels. The blood pressure (BP) was normal. Fetal growth and amniotic fluid were in normal range throughout the entire pregnancy.
At 35+4weeks she attended the hospital for a persistent severe headache lasting for 3 days. The BP was 150/88mmHg with proteinuria: protein creatinine ratio (PCR) was 51. The diagnosis of preeclampsia (PET) was made. She was treated with Labetalol (100mg twice daily) and the BP was controlled (below 135/85 mmHg). However, the headache persisted and the Na was124 mmol/l. The endocrinology team advised for fluid restriction to 1l/day and the Na recovered slowly over the next 2 days up to 130 mmol/l. The headache resolved. She was discharged on Labetalol 100mg twice daily and advised to continue the fluid restriction.

Only 10 days later, at 37+0weeks gestation, she attended the hospital secondary to a fall on her right-side during walking. The BP was found to be high at 148/93mmHg and she had ++proteinuria. The BP raised further to 155/97 mmHg within the next hour and then to 161/94 despite further doses of Labetalol. She was treated with Nifedipine 10 mg, and the BP temporarily settled. However, 3 hours later the BP was 173/96mmHg with normal reflexes and no clonus, just a mild headache. The Na was 129 mmol/l. We decided to start magnesium sulphate (MgSO4) infusion to prevent eclamptic seizures and to increase the dose of Labetalol. We administered a bolus of MgSO4 4g followed by an infusion at a rate of 1g/h. We measured the BP continuously via arterial line and the Na levels by ABG (arterial gas) and laboratory. We repeated the Na levels 2 hourly. The levels were between 128-130mmol/l.

Immediately after the MgSO4 infusion was started she started to have the first episode of eclamptic seizures. She didn`t respond to intra venous (IV) Labetalol (50mg) with BP persistently over 170/90 mmHg and she had a second eclamptic episode. We administered the second bolus of MgSO4 of 4g and the seizures stopped. She received a second dose of IV Labetalol (50mg) as the BP was still high, at 176/96mmHg. The Na level was 130mmol/l at that moment (checked on arterial blood gas sample) and the capillary blood glucose levels were normal (5.6 mmol/l) so we concluded that the cause of the seizures was eclampsia and not hypoglycaemia or hyponatraemia. She responded finally to IV hydralazine (5mg). The BP came down to 145/85mmHg, and we performed an emergency caesarean section (CS). The baby was delivered in good condition (Apgars 9 at 1min and 9 at 5min) and the BP was under control after delivery with Labetalol (200mg 4 times daily) and Nifedipine MR 10mg twice daily. The blood glucose levels were well controlled. The Na levels were between 127-129mmol/l post- delivery and with fluid restriction were corrected to 130 mmol/l. One week after delivery her Na was 131 mmol/l and the proteinuria increased (PCR 263) with secondary hypoproteinaemia (serum albumin 24 g/l).

Discussion

Hyponatremia is the most common electrolyte abnormality. The chronic hyponatremia in pregnancy poses diagnostic and therapeutic challenges. The physiological changes in pregnancy affect water and Na homeostasis, however usually the Na levels don`t decrease below 130 mmol/l [1].

If associated with preeclampsia, there is a significant rate of complications. In one of the case series reported by Morton & al, acute kidney injury was found in in 34.1% of cases, HELLP syndrome in 17.1%, intrauterine growth restriction in 36.4%, intrauterine death in 2.3%, the use of magnesium sulphate in 44.2%, and admission to an intensive care unit in 28.7%. Obviously the moderate/severe hyponatraemia was associated with higher risk than mild hyponatraemia. Urgent delivery was required in 71% of cases within 24 h of diagnosis of moderate/severe hyponatraemia. [2] However, no case of eclampsia was described.

In almost all cases, hyponatraemia resolved quickly after delivery without requirement for fluid restriction or intravenous saline [2]

The pathogenesis of hyponatremia associated with preeclampsia is not well known. A non-osmotic stimulation of vasopressin release in the case of a hypervolemic state is believed to be the main mechanism. Fluid restriction is the best treatment [4].

Chronic hyponatremia from the first trimester of the pregnancy is rare and an early diagnosis and treatment are very important. In the setting of preeclampsia, the hyponatremia is associated with increased risk of maternal seizures. Fetal reported complications were: fetal hyponatremia, jaundice, tachypnea, seizures, and polyhydramnios [3].

The treatment of chronic hyponatremia can be very challenging in the setting of preeclampsia. Demeclocycline and conivaptan are contraindicated in pregnancy, and furosemide is a class C drug, so best to be avoided. Fluid restriction may not be effective, and worsening hyponatremia should be an indication for expedite delivery [3]

Type 1 Diabetes is a risk factor for preeclampsia in pregnancy as well.

In our case the hyponatremia was chronic, diagnosed from the first trimester and no definite cause was found by the endocrinology team. The preeclampsia developed just at 35+4 weeks gestation as a mild form but progressed very quickly, being severe at 37+0w and the patient developed recurrent eclamptic seizures few hours after admission despite treatment and despite the fact that the hyponatremia was mild at the time of eclampsia (between 128-131 mmol/l).

Hyponatraemia in the context of PET has complications that may significantly compound the sequelae of severe PET. It was proposed that monitoring of serum sodium levels should be a common practice in the management of patients with preeclampsia to allow for prompt and appropriate correction of hyponatremia [5].

Conclusions

Hyponatraemia should be considered a risk factor for severe preeclampsia and it increases the risk of eclamptic seizures, even if it`s mild or moderate.

We advise prompt treatment and early MgSO4 infusion in cases with severe preeclampsia on a background of chronic hyponatremia. An expedited delivery should be considered rather earlier than later.

More research is needed in order to establish the best management for these patients with type1 diabetes, hyponatraemia and preeclampsia

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