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Peripartum Hyponatraemia: A Rare Occurrence?

Yeung E and Fleming T*

The Gold Coast University Hospital, Australia

Abstract

Background: Hyponatraemia is a recognised, but underreported complication of the peripartum period. This presentation endeavours to bring to light the susceptibility of labouring women and their babies to hyponatraemia by presenting an overview of sodium metabolism, in addition to a case report and review of the current literature.

Case study: A 33 years multiparous lady in spontaneous labour developed confusion four hours postnatal and was found to have serum sodium of 114mmol/L. She had not received oxytocin or intravenous fluid supplementation intrapartum, and reports consumption of 2.5 litres of water in the preceding six hours of labour.

Conclusion: There are a number of factors which predispose the pregnant woman to a dilutional hyponatraemia, including the elevated fluid volume, iatrogenic fluid supplementation, "encouraged" polydipsia and alterations in Anti Diuretic Hormone (ADH).

Keywords: Anti Diuretic Hormone; Hyponatraemia; ADH secretion; Atrial baroreceptors; Anti diuretic effect

Introduction

Hyponatraemia is a recognised, but underreported complication of the peripartum period [1]. Broadly, hyponatraemia is stratified into dilutional and non-dilutional, where hypervolaemic hyponatraemia is most common during the perpuerium. There are a number of factors which predispose the pregnant woman to a dilutional hyponatraemia, including the elevated fluid volume, iatrogenic fluid supplementation and "encouraged" polydipsia [1]. The most important factor, however, is likely to be due to alterations in anti Diuretic Hormone (ADH), ADH simulants, and the physiological responses to this hormone [2].

This report endeavours to bring to light the susceptibility of labouring women and their babies to hyponatraemia by presenting an overview of sodium metabolism, in addition to a case report and review of the current literature.

Pathophysiology

The major stimuli to ADH secretion are usually hyperosmolality and the depletion of circulating blood volume. These responses are mediated by cranial osmoreceptors, and arterial and atrial baroreceptors. ADH secretion can also be provoked by common labour phenomena, such as pain, nausea and opioid analgesics [2].

In addition to this, oxytocin, in both its endogenous and synthetic forms, has an anti diuretic effect. Oxytocin is structurally and functionally related to ADH, and is able to bind to renal ADH receptors to produce a similar action [2].

While these factors increase the likelihood of the development of hyponatraemia, there are other physiological adaptations that make a pregnant woman more susceptible to the adverse impact of this condition. Specifically, circulating oestrogen and progesterone inhibit the maternal brain's sodium pump, thereby exacerbating neurological symptoms. A relative hypernatraemia occurs in brain cells, causing an influx of water into the brain tissue. In severe cases this results in cerebral oedema and subsequent hyponatraemic encephalopathy [2].

Symptomatic acute hypernatraemia is a medical emergency. However, rapid correction of hyponatraemia in itself also presents a risk for congestive cardiac failure and of central pontine myelinolysis or osmotic demyelination due to the inability of the brain to adapt to rapid osmotic changes [3].

Chronic hyponatraemia in most cases is asymptomatic and usually related to pre-existing medical conditions including liver cirrhosis and heart failure [3]. Therefore, this entity is not seen frequently in the pregnant population.

Commonly, hyponatraemia is asymptomatic. Symptoms are not usually seen with sodium levels above 125 mmol/L [1]. Early symptoms of hyponatraemia such as nausea, weakness and apathy, are easily missed as they are frequently seen in normal labouring women. More advanced symptoms include unresponsiveness, pulmonary oedema, respiratory arrest and seizures.

As neonatal sodium and water equilibrates across the placenta, the fetus is also at risk in the presence of maternal hyponatraemia [4]. Neonatal hyponatraemia is associated with hyperbilirubinaemia, respiratory distress, feeding difficulties and convulsions [4,5].

Case Study

A 33 year old multiparous lady presented in spontaneous labour at 41 weeks gestation. There was a history of two previous spontaneous vaginal deliveries, no prior medical issues, and an uncomplicated antenatal course this pregnancy through the Midwifery-led model of care. Labour progressed adequately to full dilatation; second stage was completed in twenty minutes and an unremarkable physiological third stage having declined oxytocin or ergometrine-oxytocin (Syntometrine®) for active management. Total estimated blood loss for the birth was 400 ml, there were no perineal lacerations, and paracetamol was given for afterbirth discomfort. It was calculated that she had consumed 2.5 L of water during the preceding 6 hours, and no intravenous fluid had been administered.

Two hours post-partum, the patient's husband alerted staff to her unusual behaviour and confusion. On immediate review, she was normotensive, normocardic and saturating above 98%. Gross

*Corresponding author: Tina Fleming, The Gold Coast University Hospital, Australia, Tel: +61 7 5519; 8211; E-mail: Tina_Fleming@health.qld.gov.au

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neurological examination was normal, however she was not oriented to time or place. Urgent investigations were requested demonstrating a significant hyponatraemia, with maternal sodium concentration of 114 mmol/L, normal potassium 3.7 mmol/L, and reduced osmolality 236 mmol/kg. Urinary osmolality was normal at 739 mmol/kg, and urinary sodium was 52 mmol/L. Other investigations conducted revealed normal renal, liver, thyroid and adrenal function.

In view of the rapid development of her symptoms, a diagnosis of acute hypoosmolar hyponatraemia was made. Rapid correction of her hyponatraemia was commenced with a hypertonic saline solution (3%) at a rate no faster than 2 mmol/L to minimise the risk of the development of osmotic demyelination syndrome or central pontine myelinolysis [6]. She was transferred to the intensive care unit (ICU) for close observation of her neurological state and monitoring of sodium levels every 2 hours. Nine hours following initiation of treatment, the patient's biochemical parameters improved, with sodium correcting to 122 mmol/L, serum osmolality at 250 mmol/kg and urine osmolality 340 mmol/kg. At this stage symptoms of confusion and disorientation had improved. She was discharged from ICU after 24 hours. Her neurological symptoms completely resolved over the course of the next 48 hours along with daily improvements in her serum biochemical profile and she was discharged home on day 4 post-partum.

Discussion

Maternal hyponatraemia is an important yet underreported complication of the peripartum period. As discussed above, specific physiological changes as well as management practices during the intrapartum and postpartum period can contribute to maternal and neonatal hyponatraemia and its associated complications in its most severe form.

The largest study of this topic undertaken is a prospective observational study published in 2008 assessing the rate of symptomatic and asymptomatic hyponatraemia in the term labouring population [1]. It found 21 cases of hyponatraemia (defined as $\text{Na} < 130$ mmol/L) out of 287 labouring women (7.3%), and that this correlated significantly with total fluid volume administered during labour, but not with epidural analgesia or oxytocin administration. Other associations with reduced sodium borne out by this study were prolonged second stage, and delivery by instrument or caesarean section. None of the participants of this trial developed signs of severe hyponatraemic encephalopathy.

While severe hyponatraemia is a rare occurrence, it seems that mild hyponatraemia is more common than we have previously acknowledged. As parturients are at an elevated risk for the development of hyponatraemia due to several physiological factors, it is important that those managing labour are alert to this potential complication.

A search of international literature revealed five other reported cases of severe hyponatraemia in parturients resulting in neurological sequelae [7-11]. The interesting issue in our case is that the fluid loading that contributed to this patient's hyponatraemia was purely via oral intake of water. In the five other cases, fluid supplementation was with oral intake of water and electrolyte drinks as well as with intravenous fluid and incidental additional fluid via required oxytocin infusion. The resulting neurological sequelae of the other cases ranged from acute confusional state to grand mal seizures.

In the case presented, the patient consumed electrolyte-free water which, in its relatively low volume compared with other presented cases, increased her susceptibility to developing hypervolaemic hyponatraemia.

A differential that may be considered in women with hyponatraemia and neurological symptoms is preeclampsia. There have been 9 cases of hyponatraemia complicating preeclampsia reported in literature [12]. It has been postulated that the increase of fluid in the interstitium and subsequent decrease in effective circulating volume leads to the physiological response of ADH leading to hyponatraemia. This however, is a rare entity.

Taking into account this information, there are two issues which warrant consideration. Firstly, there may be value in a low volume, high concentration of oxytocin infusion for patients requiring induction or augmentation of their labour. Secondly, while fluid restriction of labouring patients is not routinely recommended in Australia, it may be prudent to advise careful monitoring of the fluid balance intrapartum in an attempt to prevent overload.

Only two criteria are required to develop hypervolaemic hyponatraemia: a source of electrolyte-free water, and an increase in activity of ADH to retain this water. While obstetric physiology dictates the altered hormone release and there is little we can do to resolve this, we can exercise caution in free fluid administration and consumption in the labouring patient.

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