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Hyponatremia as an underdiagnosed postpartum headache emergency: a case report

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Abstract: Postpartum headache is a common emergency department complaint with a wide differential diagnosis. Distinguishing primary from secondary, more sinister, causes of headache is an important skill for the emergency physician.

We present a case of a 31-year-old G1P1 woman who presented at five days postpartum with a 48-hour headache and an otherwise uncomplicated pregnancy. She had several precipitating postpartum risk factors, including use of hypotonic fluids in labour, oxytocin to augment labour, changes in food and drink patterns, and was mildly hypertensive. Urgent investigations in the emergency department found her to be severely hyponatremic (sodium: 121 mmol/L (normal: 136-144)) without evidence of preeclampsia. She was admitted to hospital to correct her electrolyte imbalance. This case highlights the importance of remaining vigilant in differentiating concerning causes of postpartum headache, as well as identifying key precipitants which may put women at risk for postpartum hyponatremia.

Keywords: Headache; Hypertension; Hyponatremia; Postpartum

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1. Introduction

Headache is one of the most common presenting complaints encountered in the postpartum period, with reported incidence ranging between 11% to 80% (1). The differential diagnosis is broad and includes several life-threatening causes. Further, the postpartum period is characterized by sleep changes, irregular food intake, dehydration, and hormonal fluctuations which can all be associated with headaches (2-4). With limited time and resources in the emergency care setting, it can be challenging to determine which headaches are particularly worrisome and warrant further urgent investigation. Despite its high incidence, there are no guidelines and little to no research describing appropriate investigations and treatment of women with postpartum headaches (5). It is imperative to keep a broad differential and be vigilant in recognizing warning signs of a more insidious cause.

2. Case presentation

A 31-year-old G1P1 woman presented at five days postpartum with a 48-hour frontal-pressure headache. She described it as 4/10 in intensity, non-radiating, with associated nausea (no vomiting), blurred vision without diplopia, and no photophobia or phonophobia. Review of systems was otherwise negative, including no fevers, chest pain, dyspnea, or orthopnea. She had an uncomplicated pregnancy, with no history of hypertension in pregnancy, preeclampsia, or gestational diabetes. She was induced for post-dates at 40 weeks and four days with an artificial rupture of membranes and initiation of oxytocin with hypotonic fluids. She subsequently had a spontaneous vaginal delivery with no postpartum hemorrhage. Upon discharge home she was medically stable, with blood pressure 129/82 mmHg. Her past medical history is significant only for migraines, but she said this headache is different. She had been taking acetaminophen and ibuprofen for perineal pain, and that morning took rizatriptan, which is prescribed as needed for her migraines. She takes no other medications regularly.

At the emergency department, her initial vitals were as follows: blood pressure 148/91 mmHg, heart rate 76 beats/min, respiratory rate 18 breaths/min, temperature 36.3 degrees Celsius, and oxygen saturation 97% on room air. Her physical examination was unremarkable, with Glasgow coma scale score of 15, normal cranial nerve examination including pupils equal and reactive bilaterally, power 5/5 in upper and lower extremities, reflexes 1+ bilaterally, neck supple, with normal gate and cerebellar testing. There was no temporal or scalp tenderness nor jaw claudication. Cardiac, respiratory, abdominal exams and peripheral vascular were unremarkable, and her uterus was firm.

Her initial electrolyte investigations demonstrated her to be significantly hyponatremic, with a sodium: 121 mmol/L (normal: 136-144), potassium: 4.2 mmol/L (normal: 3.5-5.5), chloride: 88 mmol/L (normal: 98-109), bicarbonate: 22

Table 1 Differential diagnosis of postpartum headache

Primary headache
Migraine headache
Tension-type headache (includes benign ordinary headache)
Orgasmic headache
Secondary headache
Post-dural puncture headache
Preeclampsia/eclampsia*
Cerebral venous sinus thrombosis*
Stroke (ischemic or hemorrhagic)*
Ruptured aneurysm or malformation*
Hypertensive encephalopathy or bleeding
Cerebral arterial thrombosis or embolism
Posterior reversible encephalopathy syndrome (PRES)
Postpartum cerebral angiopathy*
Pituitary apoplexy*
Pseudotumor cerebri
Subarachnoid hemorrhage*
Meningitis*
Hyponatremia and other electrolyte disturbances*

*: Life-threatening
Diagnoses adapted and expanded from Stella et al. (5)

mmol/L (normal: 22-29), anion gap: 11 (normal: 8-16), plasma osmolality: 253 mmol/kg (normal: 278-305), creatinine: 44 umol/L (normal: 44-80), uric acid: 122 umol/L (normal: 143-339), thyroid stimulating hormone (TSH): 1.71 munits/L (normal: 0.40-5.00) and normal liver function tests. Complete blood count showed hemoglobin of 125 g/L (120-160), white blood cells: $5x10^9$/L (4.8-10.8), and platelets: $281x10^9$/L (130-400). Urinalysis demonstrated white cells and trace blood but no protein, urine osmolality was 134 mmol/kg (normal: 50-1200), urine sodium was 33 mmol/L, urine potassium was 12.7 mmol/L, and urine chloride was 29 mmol/L. Electrocardiogram demonstrated normal sinus rhythm.

She was given one dose of furosemide 10 milligram (mg) intravenous (IV) in the emergency department and initiated on nifedipine extended release 30 mg twice daily. She was seen by an obstetric medicine specialist and admitted with a diagnosis of severe hyponatremia with mild postpartum hypertension (no preeclampsia). She remained in hospital briefly to correct her hyponatremia with fluid restriction (<1.5 L/day) and monitor her blood pressure (target <140/90 mmHg). Target sodium was no more than an 8-point increase/day, which normalized naturally with fluid restriction. Blood pressure was well-controlled on nifedipine extended release 30 mg twice daily, and she was discharged without further complication.

3. Discussion

Headache is a common complaint encountered in the emergency department during the postpartum period (2,5). The differential diagnosis for headache in pregnancy is broad and includes both primary and secondary causes (Table 1). Primary causes of postpartum headache refer to headaches that are unrelated to any underlying medical condition and are typically benign (4,6,7). These are the most common cause of postpartum headaches, and are often related to factors such as hormonal changes, stress, dehydration or sleep disturbances (3,4,7). In contrast, secondary causes of postpartum headache are those that arise as a result of an underlying medical condition or pathology, such as preeclampsia/eclampsia, cerebral venous thrombosis, meningitis, or intracranial hemorrhage (4). These headaches may present with more severe symptoms (i.e., neurologic deficits, seizures, fever, neck stiffness) and require urgent medical evaluation and intervention. A reasonable diagnostic approach begins with a good history and physical exam. Within the immediate postpartum period, key risk factors to assess for include hypertension with proteinuria, seizures or neurologic deficits (5). If preeclampsia (Table 2) or eclampsia (seizures) is diagnosed, it is a medical emergency and the patient should be treated as an inpatient with antihypertensives and/or magnesium sulfate (8-10). If there are focal neurologic deficits at any point in time, cerebral imaging should be performed urgently (11,12). Of course, a history of dural puncture also necessitates consideration of a blood patch, and anesthesia should be consulted urgently (13-15). If there are none of these findings, tension or migraine headache may more reasonably be favoured (5).

This case is unique as it highlights the relationship between postpartum headache and hyponatremia, which is often underdiagnosed. The postpartum period is characterized by significant hormonal fluctuations (i.e., oxytocin which has an antidiuretic effect), as well as changes in fluid balance and electrolyte regulation (16,17). Specific risk factors include excess hypotonic fluids used during labour, oxytocin to augment labour, and changes in food and drink consumption. These factors can contribute to the development of hyponatremia in postpartum women, as demonstrated in this case (18,19). In turn, hyponatremia can manifest with various symptoms, commonly including headaches (20,21). In a patient with significant clinical symptoms from hyponatremia (i.e., sodium <130 mmol/L and seizures or loss of consciousness), emergency department management includes immediate treatment with intravenous hypertonic saline. This should raise serum sodium by 2-4 points and help to reduce cerebral edema. Further management involves fluid restriction (<30 ml/hour), careful monitoring of electrolytes so as not to have greater than 12-point increase in sodium/day, consideration of a small dose of intravenous furosemide (20 mg) if clinical evidence of fluid overload, and consultation from obstetric medicine specialists (22). As discussed in this case, the correction of the patient's hyponatremia, along with blood pressure management, led to the resolution of her symptoms and highlights the importance of considering electrolyte imbalances as a possible cause of headaches in the postpartum period.

The relationship between postpartum hyponatremia and hypertension is poorly understood. Due to the common post-

Table 2 Preeclampsia diagnostic criteria and investigations

Definition	Maternal diagnostic investigations
Gestational hypertension with new-onset proteinuria or one/more adverse conditions (defined as a maternal end-organ complication or evidence of uteroplacental dysfunction)	Maternal testing should include, in addition to gestational age and the presence of chest/pain dyspnea: • Oxygen saturation (with blood pressure) • Platelet count • Serum creatinine • AST or ALT

Definition and investigations adapted from society of obstetricians and gynaecologists of Canada (SOGC) guidelines (8)
AST: Aspartate aminotransferase; ALT: Alaine aminotransferase

partum physiologic changes that occur within the cardiovascular system and fluid shifting, blood pressure usually spikes in the first three to seven days after giving birth (23-25). These changes alone put postpartum women at higher risk of developing hyponatremia. It is thought that severe hyponatremia may be due to increased vasopressin sensitivity, placental overproduction of vasopressin and placental decreased production of vasopressinase (26). All of these changes combined resolute in inappropriately increased levels of antidiuretic hormone, thus contributing to hyponatremia (27,28). To our knowledge, no studies have looked at the relationship between postpartum hypertension and hyponatremia, although one found the incidence between preeclampsia and hyponatremia to be 9.7% (19). Currently, the diagnostic evaluation of women with preeclampsia does not include electrolytes (Table 2), although based on this relationship it may be considered as an addition in future guidelines. Regardless, vigilance and heightened awareness for hyponatremia in the postpartum period is key to early diagnosis and intervention.

This case demonstrates a unique presentation of postpartum headache which is often misdiagnosed and can be fatal. Emergency physicians see postpartum headache as a frequent presenting complaint and play an important role in differentiating benign from sinister causes. It is important to be vigilant when considering the differential diagnosis for postpartum headache, and aware of risk factors patients may have that can make them further susceptible to hyponatremia. Expedited workups, including electrolytes, and emergency department imaging may be necessary if a more insidious cause is suspected. Increased understanding and knowledge about this unique diagnosis will raise suspicion and enhance care of patients susceptible to postpartum hyponatremia.

4. Conclusion

This case demonstrates that extreme vigilance must be used when considering a broad differential diagnosis for women presenting with postpartum headaches, including primary versus secondary causes. Risk factors for identifying postpartum hyponatremia must be identified early to facilitate expedited investigations and treatment.

5. Declarations

5.1. Acknowledgement

None.

5.2. Authors' contribution

ML: Conceptualization, methodology, original draft preparation, writing – reviewing and editing; BK: Conceptualization, visualization; RV: Conceptualization, data curation, supervision.

5.3. Conflict of interest

None.

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