Severe Hypernatraemia in postpartum eclampsia: a case report

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Abstract:

Eclampsia is one of the most dangerous acute complications of pregnancy, with significant morbidity and mortality. Hypernatraemia in eclampsia is rare and when it occurs, could lead to critical and potentially life-threatening complications. Hypernatraemia is a disorder of the homeostasis of body water and sodium with a net increase in serum sodium. The most dreaded clinical manifestations of hypernatraemia whether in eclampsia or not are that of the central nervous system due to brain cell shrinkage which are more in acute and severe form of the disorder. We report a case of a 26-year-old primigravida female with severe acute hypertonic (hypervolemic) hypernatraemia secondary to postpartum eclampsia. This study highlights the clinical presentation, serial biochemical investigations and treatment modalities of the patient with a review of pertinent literature. We concluded that hypernatraemia in any form may occur in eclampsia and adequate attention need to be drawn to the fluid and electrolyte management of these patients to prevent unfavorable outcome.

Keywords: Severe hypernatraemia, postpartum, eclampsia, Venous Blood Gas Analysis

Introduction

Severe hypernatraemia in postpartum eclampsia represents a rare critical and potentially life-threatening complication that demands prompt recognition and intervention. Eclampsia is one of the most dangerous acute complications of pregnancy, with significant morbidity and mortality for both the mother and the infant.[1] It is described as the occurrence of one or more generalised tonic-clonic convulsions in women with hypertensive disease of pregnancy that are unrelated to other medical disorders. In advanced nations, the reported incidence of eclampsia ranges from 1.6 to 10 per 10,000 pregnancies, whereas it ranges from 50 to 151 per 10,000 deliveries in developing nations. Furthermore, low-resource countries have significantly higher rates of maternal and neonatal mortality and morbidity.[2] Severe hypernatraemia, defined as a serum sodium concentration exceeding 155 mmol/L, further exacerbates the complexity of the clinical scenario. This condition is particularly alarming due to its association with neurological sequelae and increased morbidity.

Although, with the establishment of guidelines for magnesium sulphate prophylaxis for women presenting with severe pre-eclampsia, eclampsia may be regarded a preventable condition in many circumstances.[3] However, eclampsia occurs at a rate of 2.0% in women with severe preeclampsia who do not receive magnesium sulphate, and 0.6% in those who do.[3] Both pre-eclampsia and eclampsia are one of the leading causes of maternal and perinatal morbidity and mortality in Nigeria with a varying prevalence that depends on the geopolitical zones. In a recent study done in Abuja, the prevalence was 3.6% with case fatality rate of 3.9%.[4] While in Sokoto the prevalence was 12% and case fatality rate of 28%.[5]

The development of hypernatraemia in postpartum eclampsia is multifactorial, involving a dysregulation of fluid and electrolyte balance (usually hyponatraemia),[6] renal dysfunction, and potentially, inadequate management of eclampsia. Severe hypernatraemia poses a threat to neurological, cardiovascular, and renal systems, leading to a range of complications such as cerebral edema, seizures, and organ failure. Recognizing the interplay between eclampsia and hypernatraemia is crucial for timely diagnosis and appropriate management to prevent further morbidity and mortality. This case reports highlights the serial electrolytes findings and modality of management of this patient from the establishment of diagnosis to successful discharge.

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Case report:

The patient is a 26-year-old primary school teacher, booked (elsewhere) primigravida at EGA of 36 weeks who presented at the labour ward with a referring blood pressure of 150mmHg systolic and 100mmHg diastolic as well as 3+ of proteinuria. She also presented with epigastric pain and haemoptysis of 1 week duration. Epigastric pain was said to be burning in nature, radiates to the back, no associated aggravating nor relieving factor. No history of vomiting and diarrhea. She was said to be a known peptic ulcer disease patient, although no endoscopy was done. She also presented with 1 week history of intermittent heamoptysis and cough. No history of dysnoea, no orthopnea and no paroxysmal nocturnal dysnoea. No history of head ache, no blurring of vision, no dizziness, no convulsion, no facial swelling but there was associated severe bilateral leg swelling up to the sacrum.

There was no labour pain, no bleeding per vagina, no draining of liquor, she still perceives fetal movement, and no maternal family history of twin gestation. Index pregnancy was spontaneously conceived, confirmed by ultrasound in a secondary institution at 16th week of gestation. She was booked at a secondary institution at 29th week and had 2 doses of TT and SP for IPT. She was regular on antenatal clinic visit and routine medications. She was noticed to have elevated BP at 35 weeks and was placed on Aldomet and Nifedine 10 mg daily.

With the above history and following examinations (repeat BP 160/110mmHg) and investigations (urinalysis – 3+ proteinuria and USS-Twin gestation), a diagnosis of severe pre-eclampsia in a primigrava with twin gestation at 36 weeks gestation was made. Venous blood gas (VBG) and electrolyte analysis was requested and this revealed Respiratory Alkalosis with moderate hypokalaemia and low normal sodium as shown in table 1.

She was subsequently placed on MgSO4 by Parthard regimen, intermittent hydralazine if diastolic blood pressure is greater than 110 mmHg, tab Nifedipine 30mg daily, tab Aldomet 500mg tds, and tab slow K 600mg daily. She was subsequently scheduled for emergency caesarean section (eMCS). This was done about 12 hours on admission under total anaesthesia. She delivered live female twin with good APGA's scores for both twins. Patient recovered almost fully from anaesthesia around 8 hours post eMCS. She was placed on IVF Normal saline alternate with 5% D/S 1 litre 8 hourly.

Venous blood gas and electrolyte analysis done 6 hours post eMCS revealed improving Respiratory Alkalosis and moderate hypokalaemia and high normal sodium as shown in table 1.

Serum Liver function test only revealed marginally elevated alkaline phosphatase with normal clotting profile. Serum urea and creatinine were within reference ranges. She commenced graded oral intake after 24 hours of surgery. She had a total of 2 pints of blood post eMCS.

Her BP was however poorly controlled despite hydralazine, MgSO4, Nifedipine and Aldomet with a least BP of 150/98mmHg and highest of 180/116mmHg. She convulsed 28 hours post eMCS and a diagnosis of postpartum eclampsia precipitated by suspected sepsis was made. The convulsion was generalised tonic, clonic and lasted for about 1 minutes. A total of 3 episodes were recorded within 24 hours after which she never convulsed again but her GS scale

Table 1: Results Venous blood gas and electrolyte analysis and urine sodium at different days

	Values obtained at different days							
Analyte (in venous blood)	Admission day	6 hours Post eMCS	45 hours Post eMCS	24 hours following institution of correction of sodium	48 hours following institution of correction of sodium	72 hours following institution of correction of sodium	six days following institution of correction of sodium	References
pH	7.53	7.52	7.488	7.46	7.50	7.50	7.39	7.31 – 7.41
pO_2 mmHg	75	79	70	79	79	75	75	65 - 80
PCO_2 mmHg	30.2	32.	31.7	33	30	30.2	35	35 – 60
HCO_3 mmol/L	24.7	25.3	23.5	24.2	24.2	24.7	24.9	24 – 32
N+ mmol/L	136	144	167	156	147	136	136	136 – 145
K+ mmol/L	3.0	3.2	3.2	3.0	2.9	3.0	3.3	3.6 – 5.2
Cl- mmol/L	105	103	138	125	112	105	103	90 – 108
Lactic acid	1.14	1.23	1.84	1.2	1.2	1.14	1.08	0.36 – 1.7
RBG mmol/L			7.6	6.9	6.5	6.9	8.1	3.5 – 11.1
*Urine Sodium mmol/L			45	56	56	25	20	30 – 300

* spot urine sample

was 9/15. Patient was placed on fortified pap 300mls 3 hourly through NG tube, IVF D/S 500mls 12 hourly and IV ceftriaxone 1g daily.

Forty-two hours post diagnosis of postpartum eclampsia, consult was sent to Chemical Pathologist and Medical Microbiologists on account of persistent acid-base disorder, dyselectrolytaemia and query sepis. The Medical Microbiologist confirmed the diagnosis of Sepsis and she was placed on IV Meropenem 1g daily. A repeat Venous blood gas and electrolyte analysis was requested and this revealed a moderately compensated respiratory alkalosis, moderate hypokalaemia, severe hypernatraemia and hypercholraemia as shown in table 1. A diagnosis of acute severe hypervolaemic (hypertonic) hypernatraemia secondary to postpartum eclampsia was made.

A plan for correction was set in motion using the Adrogue-Madias formula[7]

$\Delta Na^+ = (\text{infusate } Na^+ - \text{Measured } Na^+) \div (TBW + 1)$[7] to estimate the effect of 1 L of any infusate on the patient's sodium concentration and thus calculate the total negative amount of sodium daily in the patient to achieve a concentration within reference limits.

Where infusate sodium = 0 (Na^+ in 5% D/W)

TBW = Weight x 0.5 = 60 x 0.5 = 30

Thus: $\Delta Na^+ = (0 - 167) \div (30 + 1)$

$\Delta Na^+ = -167 \div 31 = -5.4$ mmol/L

Patient was then placed on 5% D/W 1 litre 8 hourly with no any saline containing fluids. Continued on IV Meropenem 1g daily, tab aldomet 500mg tds, and tab Nifedipine 30mg daily, tab slow K 600mg daily. Also, continued on fortified pap with reduction in the salt composition of the pap.

Daily repeat of Venous blood gas and electrolyte analysis revealed gradually improving acid base status, serum sodium and chloride but worsening serum potassium. There is also a relative BP control as the highest systolic BP was 133mmHg and diastolic of 84mmHg. Also, the general status of patient was also improving with GCS rising from 9/15 to 13/15 within 3 days.

Patient regain full consciousness and became ambulatory after 14 days post diagnosis of postpartum eclampsia and was discharged for follow up in the clinic and physiotherapy clinic. The discharging medications were as follows. Tab Aldomet 500mg bd, tab Nifedipine 10mg daily, tab Slow K 600mg daily, haematinics 1 tabs daily

Discussion

Hypernatraemia is defined as serum sodium concentration above 145 mmol/L. It can be mild, moderate and severe. Since sodium constitute a great proportion of serum osmolality, hyernatraemia often denotes hyperosmolality. It is a rarely encountered electrolyte disorder in hospitalized patients, usually in elderly and critically ill.[8] Depending on various clinical settings, prevalence of hypernatremia varies widely.[9] The prevalence among in-patients was reported to be between 0.5% and 5.0%;[8,10] in the accident and emergency unit, hypernatraemia is rare with a prevalence of 0.2% to 1.0%, it is about 10 times higher at 2% to 6% in critically ill patients, whereas it is about 10% - 37.2% in intensive care units.[11,12] Several studies have demonstrated high mortality rate from 10-75% in patients with hypernatraemia that varies directly with acuteness and severity.[12,13]

Pre-eclampsia and eclampsia are one the most commonly encountered complications of pregnancy with high morbidity and mortality. Although, patients with eclampsia are considered critically ill, most authors have demonstrated lower serum sodium in pregnancy induced hypertension, preeclampsia and eclampsia as compared to normotensive pregnant and non-pregnant women.[14,15] This has being attributed to factors like reduced intrarenal production of cyclic GMP (cGMP), endothelin and prostaglandin E2 (PGE2) with resultant sodium retention, hypertension and thrombosis;[16] alteration in cell membrane sodium transport leading to extravascular accumulation of sodium with a reduced plasma sodium level;[16] decreased intravascular circulating volume with resultant activation of baroreceptors, release of antidiuretic hormone (ADH) causing water retention and altered natriuretic peptides levels;[17] as well as dilutional.[15] Other studies[18] reported no changes in the level of sodium in pre-eclampsia and eclampsia while some reported increase in the concentration of sodium in them.[19] Hypernatraemia, whether in eclampsia or general population has being attributed to negative fluid balance, increasing solute load from medications and nutrition,[20] low Glasgow Coma Scale (GCS) score, inadequate cation exchange (renal sodium handling) due to severe illness[12] and blood transfusion.[21] Index patient was placed on 5% Dextrose saline, an intravenous fluid containing 75mmol/1litre of sodium. Although this is justified judging with the level of serum sodium before the surgery. She was also on ceftriaxone (contains 3.6mmol/g of drug) and later meropenem (4.0 mmol/g of drug), fortified pap (usually contains varying percentage of table salt). She only had 2 days of negative fluid balance, a GCS of 8-9 and had 3 pints of blood transfused and urinary sodium of >20mmol/L. And hypernatraemia in this patient might be attributed to the above listed factors.

Clinical manifestations of hypernatraemia are usually related to its effect on the central nervous system due to brain cell shrinkage which are more in acute and severe form.[22] List of signs and symptoms of hypernatraemia includes thirst, nausea, vomiting, oliguria, orthostatic blood pressure changes, tachycardia, weakness, muscle spasticity, neuromuscular excitability, lethargy, hyperreflexia,

confusion, seizure, coma[23] etc. Although whether the seizure in the index patient was a sign of hypernatraemia or otherwise could not be ascertained. In the index patient, other signs and symptoms were masked with seizure and the semi-comatous state.

Determining the cause of hypernatraemia involves a thorough analysis of the patient's medical history, physical examination, and appropriate biochemical investigations. Several steps are employed in making correct diagnosis. Several diagnostic approaches are available. Most of them involves the following - exclusion of pseudohypernatremia; correction of serum sodium concentrations in case of hyperglycaemia; determination of the state of extracellular volume state using the history and physical examinations' measurement of urine sodium; measurement of urine volume; calculation of ongoing electrolyte-free water clearance (EFWC); measurement of urine osmolality; measurement of AVP or copeptin levels in patients with hypotonic polyuria; and lastly checking for concomitant electrolyte disorders (serum potassium and calcium). [24,25,26] EFWC was not calculated, neither did AVP/copeptin was measured in index case. We were able to made a diagnosis acute severe hypervolaemic hypertonic hypernatraemia secondary to postpartum eclampsia.

Treatment of hypernatraemia in eclampsia comes with its intricacies, trying to balance the need for fluid restriction strategy in eclampsia in order to prevent pulmonary oedema and the need for the fluid for correction of hypernatraemia. Management of hypernatraemia involves identifying and treating the underlying cause(s) and subsequently correcting the established hypernatremia while considering the onset, severity of symptoms (most especially neurologic) and volume status of the patient.[24] Probable cause of hypernatraemia in this patient include to negative fluid balance, increasing solute load from medications and nutrition, altered consciousness, severe illness and blood transfusion

Conclusion

Finally, severe hypernatraemia complicating postpartum eclampsia is a rare complex and potentially fatal condition that needs to be recognized and treated promptly. Managing the condition comprehensively by a multidisciplinary team, correcting electrolyte imbalances promptly, and closely monitoring the health of both the mother and the fetus are all necessary to reduce risks and improve outcomes. Judging with this index patients, a close monitoring of the fluid charts of patients is essential thereby balancing fluid restriction on one hand and IV fluid therapy on the other for correction while not forgetting the appropriate type of IV fluid to use.

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