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Case Series

Obstetrics and Gynaecology Section

Maternal Peripartum Hyponatraemia Leading to Early Neonatal Seizures: A Case Series

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ABSTRACT

Peripartum hyponatraemia is an underrecognised yet significant electrolyte disturbance that can adversely affect both the mother and the neonate. Labouring women are predisposed to hyponatraemia which can be attributed to lower baseline sodium levels, impaired renal water excretion, and/or the antidiuretic effects of oxytocin. As maternal and foetal electrolytes equilibrate across the placenta, affecting the milieu, maternal hyponatraemia may result in neonatal dyselectrolytaemia and rarely early-onset seizures. In this case series, the authors aim to highlight the importance of sodium homeostasis during pregnancy and labour, to ensure accurate fluid balance monitoring, increase awareness, and hence earlier detection of peripartum hyponatraemia to prevent unfavourable maternal and neonatal outcomes. Three parturients and their neonates who developed significant hyponatraemia were analysed for their course of labour, biochemical parameters, and clinical outcomes. Two patients (Case 1,3) were Robson class I, Case 2 Robson II A. Case 1 had a vacuum-assisted vaginal delivery, the second and third had spontaneous vaginal delivery. Case 2 received Entonox as labour analgesia. Cord blood gases were normal. The three parturients had varied clinical presentation with case 1 being asymptomatic, case 2 slightly drowsy but she was also on Entonox analgesia and case 3 had generalised tonic clonic seizures. All three neonates presented with convulsions on postnatal day 1 and responded to intravenous saline. Sepsis work-up, glucose, and calcium levels were normal. No neurodevelopmental delay was observed in neonates. A high degree of suspicion in oxytocin augmented labour and even in low-risk labouring women is required to prevent the occurrence of dyselectrolytaemia. Timely recognition and treatment can avert serious maternal and neonatal complications.

Keywords: Electrolyte imbalance, Labour, Maternal hyponatraemia, Neonatal seizures

INTRODUCTION

Peripartum hyponatraemia is defined as serum sodium levels below 130 mmol/L (in pregnancy and labour) [1]. Labouring women are predisposed to developing hyponatraemia primarily because baseline plasma sodium levels are lower than those of non-pregnant individuals, the ability to excrete water in the late pregnancy is impaired and due to the antidiuretic effects of oxytocin [2]. The excessive ingestion of water and hypotonic liquids during labour and administration of intravenous fluids further contributes to the same, leading to dilutional hyponatraemia [3,4]. Exchange of water and electrolytes takes place at the placental interface, so maternal hyponatraemia can lead to an electrolyte imbalance in the foetus, thereby causing neonatal hyponatraemia. Maternal hyponatraemia remains underrecognised, and hyponatraemia symptoms have been reported in 16% of infants, both among those with and without documented maternal symptoms [5]. There is a paucity of literature in the Indian context. The authors report three cases of severe hyponatraemia in mothers, where neonates also developed severe symptomatic hyponatraemia leading to seizures. Informed written consent was taken from the participants. The lessons learned, measures to prevent this catastrophe, and further management have been discussed. The importance of sodium homeostasis during pregnancy and labour and its implications, the clinical significance of accurate fluid balance monitoring during labour, and the need for awareness and timely recognition of symptoms of hyponatraemia to prevent unforeseen consequences have been highlighted. The available literature has been reviewed. Case details are presented below and summarised in [Table/Fig-1].

CASE SERIES

Case 1

A 29-year-old booked low risk primigravida presented at 40 weeks 2 days in labour. Labour progressed spontaneously and she had spontaneous rupture of membranes at 5 cm cervical dilatation. The cardiotocograph was reassuring throughout labour. In second stage, patient had insufficient uterine contractions, frequency of contractions was less than three in 10 minutes, since the provider felt that syntocinon was required, it was added as per low dose protocol. Patient had a vacuum-assisted vaginal delivery in view of failure of descent of head beyond +2 station and maternal exhaustion. She delivered a male baby weighing 3.040 kg. The baby did not have spontaneous respiratory efforts and required positive pressure ventilation with bag and mask for 30 seconds. Paired cord blood gas analysis was normal. There was respiratory distress in the form of tachypnoea and mild intercostal retractions. On neurological examination, the neonate was slightly lethargic (depressed higher mental functions). However, tone and primitive reflexes were normal. At seven hours of life, the baby had multifocal clonic seizures and was found to have hyponatraemia (123 mmol/L). Sodium correction (1 mL/kg of 3% NaCl) was administered, after which the seizure did not recur. By day 2, serum sodium levels normalised, and neurological examination was unremarkable. Although mother was asymptomatic, her sodium level when checked was found to be low (122 mmol/L). She was given 3% saline over 3-4 hours. Both mother and baby were discharged in satisfactory condition on the 5th postnatal day.

Case 2

A 31-year-old primigravida was admitted at 40 weeks of gestation to the labour room for induction of labour in view of oligohydramnios. Labour was induced with intracervical 0.5 mg Dinoprostone gel six hours apart total two doses followed by augmentation with low dose syntocinon. Continuous electronic foetal monitoring was done. The patient was on Entonox analgesia in active labour. She had a spontaneous vaginal delivery with right mediolateral episiotomy and delivered a female baby weighing 2.850 kg. At five hours of age,

Variables	Case 1	Case 2	Case 3
Period of gestation	40 weeks 2 days	40 weeks 0 days	39 weeks 4 days
Robson's Class	I	II A	I
Route of delivery	Vacuum assisted vaginal delivery	Spontaneous vaginal delivery	Spontaneous vaginal delivery
First stage duration (h)	6 h and 15 min	14	8.5
Second stage duration (h)	2.5	1	0.5
Intrapartum fluid administration			
Oral (mL)	300	Not quantified	800
Intravenous (mL)	500	2150	nil
Postpartum Maternal Sodium levels (mmol/L)	122	124	113
Cord blood gas	7.269	7.187	Not done
ABG pH			
pO2 (mm Hg)	31	19	
HCO_3^- (mmol/L)	14.9	14.2	
Base excess (mmol/L)	-12	-14	
VBG pH	7.261	7.301	
pO2 (mm Hg)	26	31	
HCO_3^- (mmol/L)	14.4	14.4	
Base excess (mmol/L)	-13	-12	
Apgar score	6,9,9	7,9,9	8,9,9
Resuscitation details	Bag and mask ventilation for 45 seconds	Bag and mask for 60 seconds	No resuscitation required
Symptoms in newborn	Multifocal clonic seizures	Generalised seizures	Generalised seizures
Time of onset of symptoms	7 Hours of life	5 Hours of life	4 ½ Hours of life
Sodium levels in the newborn at symptom onset (mmol/L)	123	124	118
Serum calcium, random blood sugar at the time of seizure	Normal	Normal	Normal
Sepsis work-up	Negative	Negative	Negative
Recurrence of seizures after sodium correction	No	No	No
MRI Brain	Not performed	Normal	Not performed
Neurodevelopmental follow-up at 1 year (DASII scale)	Normal	Normal	Normal
Transient tachypnoea of newborn	Yes	No	Yes
Neonatal jaundice	No	No	No

[Table/Fig-1]: Clinical details.
ABG:Arterial blood gas; VBG:Venous blood gas; DASII:Developmental assessment scale for Indian infants

baby had one episode of generalised clonic seizures. Differential diagnosis of hypoxic ischaemic encephalopathy, dyselectrolytaemia, and meningitis/sepsis was considered. Cerebrospinal fluid analysis was normal. Serum electrolyte analysis revealed hyponatraemia (Na+ 124 mmol/L), and sodium correction was given immediately. There was no seizure recurrence thereafter. Mother also appeared drowsy in the immediate postpartum period, which was initially attributed to Entonox and labour stress. However, when the neonate was diagnosed to have hyponatraemia, the mother was also investigated, and her serum sodium levels were 124 mmol/l. She was given salt rich diet and salt capsules after which the serum sodium levels gradually improved. Both mother and baby were discharged on the fourth postnatal day in good condition. MRI of the brain was performed on the child for follow-up, which was normal.

Case 3

A 30-year-old primigravida at 39 weeks four days presented to the labour room in active labour. Her antenatal medical records had been normal. Labour progressed spontaneously with spontaneous rupture of membranes at 8 cm cervical dilatation and drained clear liquor. She had a vaginal delivery in the left lateral position with a first-degree perineal tear. At 12 minutes postpartum, patient had generalised tonic clonic seizure. Immediate measures were taken to maintain a patent airway and circulation. Provisional diagnosis of eclampsia was made, and Inj. Magnesium sulphate was given according to the Pritchard regimen [6], although her blood pressure was normal. Further investigations revealed a serum sodium concentration of 113 mmol/L. She was given 30 mL of 3% saline over 15-20 minutes, followed by an infusion at 20 mL/hour. Injection Levetiracetam was given to prevent further seizures and magnesium sulphate was discontinued. Her other lab reports and fundus examinations was normal. Computerised Tomography (CT) scan of head was done which showed mild effacement of bilateral parieto temporal cortical sulci suggestive of oedema. Patient was maintaining vitals but was slightly disoriented. She was managed conservatively, responded to the symptomatic treatment in the intensive care unit, and was discharged in satisfactory condition.

At four and a half hours of life, baby had multifocal clonic seizures. The sodium at the time of seizures was 118 mmol/L. Sodium correction (3% Normal saline) was given, and there was no recurrence of seizure after sodium correction. Neurosonogram of the baby was done which was normal.

DISCUSSION

The UK Obstetric Surveillance System (UKOSS) has defined peripartum hyponatraemia as the presence of symptomatic hyponatraemia and at least one sodium measurement of <125 mmol/L identified during labour or within 48 hours of delivery, once other causes of hyponatraemia are excluded, such as preeclampsia, sepsis, and any drug overdose [5]. National Institute for Health and Care Excellence (NICE) defines Peripartum hyponatraemia as serum sodium levels below 130 mmol/L (in pregnancy and labour) [1]. Hyponatraemia can result from a relative sodium deficiency, excess

water, or both, and from the antidiuretic effects of both natural and synthetic oxytocin [2]. It can be dilutional or non-dilutional, with the former being more common during labour [7].

Pregnancy is a state of hypervolemia and positive water balance. Changes in maternal osmoregulation, increased oestrogen and progesterone, increased Renin-Angiotensin Aldosterone System (RAAS) activity, increased aldosterone and deoxycorticosterone, Atrial Natriuretic peptide (ANP) secretion, and Arginine Vasopressin (AVP) control cause sodium retention and fluid overload [7]. As a result, there is an accumulation of 6.5-8.0 litres of fluid by the third trimester [8]. A labouring woman may need additional intravenous fluids to correct dehydration, pyrexia, or as a measure of foetal resuscitation; the latter is being challenged recently in the literature [9]. Oral intake of liquids and intravenous administration of hypotonic fluids during labour lead to changes in plasma osmolality, which, in turn, can cause dilutional or hypervolemic hyponatraemia. In the present-series, case 2 received over two litres of intravenous fluids in addition to oral liquids (juice, water) with adequate urine output; cases 1 and 3 received around 800 mL of fluids. Case 3, who was only on oral fluids and did not receive any intravenous fluid, still had critically low sodium levels and presented with maternal and neonatal seizures. Labouring mothers should be encouraged to maintain oral hydration with isotonic fluids rather than plain water or hypotonic fluids. The risk of dilutional hyponatraemia is directly proportional to the amount of fluid intake, with the risk being 1% in those receiving < 1 litre vs 26% in those receiving >2.5 litres [10]. Intake between 1-2.5 litres increases the incidence to 5% [2]. NICE recommends that a positive fluid balance of 1500 mL or more, or clinical signs and symptoms of hyponatraemia in a pregnant female, could indicate a risk of hyponatraemia [1]. Likewise, serum sodium levels should be monitored in those on dextrose or insulin infusions [2].

Hyponatraemia remains underrecognised as it may initially present as non-specific symptoms like anorexia, nausea, lethargy, headache, which get masked due to labour and spontaneously resolve. However, occasionally as cerebral oedema sets in, it may have grave outcomes. Patients might present with agitation, seizures, depressed reflexes, focal neurological deficit, Cheyne-Stokes respiration, and even coma and death [2]. It may mimic preeclampsia, which needs to be ruled out, as in case 3.

During normal labour, painful uterine contractions, stress, nausea, vomiting and dehydration stimulate the secretion of Antidiuretic Hormone [3]. Oxytocin, the hormone responsible for uterine contractions during labour is a nonapeptide hormone with a similar chemical structure to vasopressin differing only at two amino acid loci [8]. It has been found to have slight antidiuretic properties. Exogenous oxytocin has the same mechanism of action as endogenous oxytocin, so when used for the augmentation or induction of labour, it can cause hyponatraemia [2,8,11]. In present case series, oxytocin augmentation was required in case 1 (second stage) and in case 2 in accordance with standard guidelines [12]. However, case 3 (Robson I) was not on oxytocin augmentation. Despite the optimum duration of labour in cases 1 and 3, neonates of both parturients had neonatal seizures.

Glucose solutions, which were used as a vehicle for oxytocin in the past or still in a few institutes or high infusion rate of oxytocin lead to hyponatraemia [13,14]. The use of isotonic saline for the preparation of oxytocin infusion has been shown to prevent hyponatraemia [8]. It was quite surprising for us to observe maternal seizures in case 3 who came in active labour and no oxytocin was given externally. In case 2, oxytocin was also given at a low dose and prepared in normal saline, so the outcome was unexpected. Eclampsia was also initially thought in case 3; it was only later, on investigation reports and correlation with neonatal convulsions, that the diagnosis of hyponatraemia was made.

Certain medical conditions, like uncontrolled hypothyroidism, liver cirrhosis, cardiac or renal disorder, are the vulnerable causes of hyponatraemia [8]. None of the patients in the present series had any underlying medical condition.

Moen V et al., who did a prospective study on 287 term pregnant females in Sweden, found a significant correlation of hyponatraemia with the fluid volume administered during labour, prolonged second stage, instrumental delivery, and emergency caesarean section for failure to progress. However, they found no correlation with oxytocin administration and epidural analgesia [10]. We observed varied case presentations viz., prolonged second stage and instrumental delivery in case 1, labour augmentation with low dose oxytocin in case 2 and spontaneous uneventful labour in case 3.

Maternal dyselectrolytaemia can lead to neonatal hyponatraemia. Understanding the basic physiology of sodium and water homeostasis and awareness may, in fact, help prevent hyponatraemia and facilitate timely detection to avoid unwanted consequences for both mother and the neonate. The neonate might get affected due to neonatal hyponatraemia while maintaining equilibrium across the transplacental interface. Foetal vasopressin levels have also been reported to increase at birth, possibly due to hypoxic stress or increased cranial pressure during contractions, further contributing to changes in plasma osmolality [8]. Neonates might present with feeding difficulties, vomiting, excessive lethargy, irritability, and, in severe cases, seizures or coma. However, other causes of seizures in a neonate, like hypoxic-ischaemic encephalopathy, ischaemic infarction, intracranial haemorrhage, intracranial infections, metabolic or electrolyte disorders, congenital malformations of the central nervous system, inborn errors of metabolism need to be ruled out before labelling peripartum hyponatraemia as the causative factor. [Table/Fig-1] provides a snapshot of the neonatal workup in current series.

Neonates are at a higher risk for seizures with a reported incidence of 1-5 per 1000 infants born at term [15]. Electrolyte disorders account for 10% of neonatal seizures at birth [16]. Some studies suggest increased incidence of jaundice and respiratory distress in newborns with hyponatraemia [7,17]. Two neonates in present series had tachypnoea (lasting for less than 12 hours), and none had clinically significant hyperbilirubinaemia.

Diagnosing hyponatraemia poses a challenge for clinicians, and a strong degree of suspicion is needed. Management of severe symptomatic hyponatraemia requires a multidisciplinary approach. Intravenous sodium correction is the treatment of choice if sodium levels are below 125 mmol/l. The amount of hypertonic saline required for correction is calculated by calculating the sodium deficit using the following equation:

Sodium deficit = total body water × (desired Na+ - actual Na+). Total body water is estimated as lean body weight times 0.5 for women [18]. Aggressive correction of serum sodium levels can also lead to neurological injury, including central pontine myelinolysis. Therefore, the sodium levels should not rise by >12 mmol/L in 24 hours [1]. A balanced, targeted approach is required. If sodium levels are between 125-130 mmol/L, then fluid is restricted to 80 mL/hour, sodium levels are repeated after four hours, and oxytocin can be continued. If sodium levels are below 125 mmol/L, fluids should be restricted to 30 mL/hour, oxytocin should be stopped, and a referral to an intensivist should be sought [8]. The neonatologist has to be informed in either case. A 20 mg i.v. Furosemide is recommended in cases with fluid overload, which helps reduce cerebral oedema and raises serum sodium levels by 2-4 mmol/L [1].

The basic aim in publishing this series was to increase awareness and emphasise the water and electrolyte homeostasis during labour and how even low-risk asymptomatic mothers can present with complications that can further lead to neonatal seizures. Some salient features which can help prevent hyponatraemia: strict peripartum fluid management record should be maintained, intake

of isotonic drinks instead of plain fluids should be encouraged during labour, normal saline should be used as vehicle for oxytocin if not contraindicated, labouring women should be encouraged to void every 2-4 hourly, electrolyte levels should be done for those on oxytocin infusion, anyone having sodium levels less than 130 mmol/l and/or having positive fluid balance >1500 ml, at any sign for imbalance and also for patients on Entonox as sometimes drowsiness because of low sodium levels can get masked in its effect as in case 2, hypotonic fluids should not be transfused to prevent dilutional hyponatraemia.

CONCLUSION(S)

Hyponatraemia is one of the common yet underrecognised electrolyte disorders during labour. Strict clinical vigilance and adherence to protocols are required to prevent unwanted consequences. It requires timely detection and management to have an excellent prognosis for both the mother and the neonate.

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