



## Maternal Hyponatremia and Cardiac Arrhythmia in Peripartum Period: A Case Report and Review of Literature

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### Abstract

Hyponatremia is a common and sometimes unrecognized electrolyte imbalance in labor and hospital admissions. Pregnant women are at greater risk of this complication because of several reasons e.g. lower baseline plasma sodium level in pregnancy, altered secretion of Anti Diuretic Hormone (ADH), inappropriate fluid management in labor or increased oral intake and the anti-diuretic effect of oxytocin. We present a case which illustrates complications associated with hypervolemic hyponatremia as a result of positive fluid balance in labor. A caesarean section was required for failure to progress in first stage, chorioamnionitis, hyponatremia and acute kidney injury. She developed narrow complex Supra Ventricular Tachycardia (SVT) immediate post-delivery which was terminated with adenosine.

### Introduction

In the literature, and national guidance framework, there is a paucity of guidance for the fluid management of laboring women. This group is at a high risk of dehydration, and fluid overload if careful management of fluid intake, both oral and intravenous, is not undertaken. This can lead to risk of acute kidney injury, electrolyte imbalances [1] and cardiac arrhythmias associated with fluid overload, and well as neonatal risks, including transient neonatal tachypnoea [2], jaundice, hyperbilirubinemia, fluid overload and seizures [3].

### Case Presentation

Ms X, a 26 year old primiparous Caucasian woman was low risk with a BMI of 27 kg/m<sup>2</sup>. Her pregnancy was uneventful and she presented to our Midwifery Led Unit (MLU) following spontaneous rupture of membranes at 40+8 weeks. Following a delay in the 1<sup>st</sup> stage of labor at 5 cm cervical dilatation, she was transferred to the Consultant Led Unit (CLU) for oxytocin augmentation.

In terms of fluid intake and output record in labor; she had 4L of intravenous Hartmann's in labor in consultant led unit, but prior oral intake in labor was unknown. She had required 9 h of oxytocin infusion in labor with titration rates of 8 to 12 ml/hr according to uterine contractions. Her total urine output in 9 h was recorded as 700 ml. She subsequently developed pyrexia in labor. In view of poor urine output and pyrexia in labor, she had septic screen and bloods taken, including FBC, CRP, U&E's and lactate. This showed her sodium level as 120 mmol/l and creatinine 139 μmol/l. Nine hours prior to deterioration, her creatinine level was 72 μmol/l; however sodium was recorded low at 123 mmol/l. There were no preceding sodium levels to compare to.

Following review of blood results, identification of Acute Kidney Injury (AKI), suspected chorioamnionitis and delay in the 1<sup>st</sup> stage of labor at 8 cm cervical dilatation, the decision was made to expedite delivery by caesarean section. Blood loss at delivery was 800 ml and fetal weight as 4215 g. Neonatal Apgar reported as 7 at 1 min and 5 at 10 min. Cord gases were 7.29 BE - 3.7 (arterial) and 7.31 BE - 4.6 (venous). Baby required neonatal special care input with IV antibiotics and sodium replacement in view of neonatal hyponatremia.

Immediately following delivery, she developed narrow complex supra ventricular tachycardia and required cardio version with IV adenosine. She was subsequently transferred to Intensive Therapies

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Unit (ITU) for cardiac monitoring, and strict fluid monitoring in light of hyponatremia and AKI. She required multidisciplinary care with further input from intensive care team, medical and renal teams for ongoing management.

At time of SVT, her GCS remained 15 with normal blood pressure and oxygen saturations. Her spot sodium test showed sodium of 117 mmol/l and potassium of 3.2 mmol/l. She developed second episode of SVT in ITU 6 h later, which required further cardio version with IV adenosine.

She required potassium and magnesium replacement in ITU. Her cortisol and thyroid function tests were reported as normal. Her initial urine creatinine was 2.48 mmol/l with urine sodium as 17.8 mmol/l and urine sodium to creatinine ratio as 7.2. She made full recovery and was discharged home with baby on day [4].

Following cardiology review, she had an EP (Electrophysiology) study which confirmed the suspicion of an accessory pathway which has now been ablated.

## Discussion

Hyponatremia is broadly characterized as euvolemic, hypervolemic or hypovolemic hyponatremia. In pregnancy, the most common manifestation is hypervolemic hyponatremia (also referred to as dilutional hyponatremia or water toxicity). This is caused due to the woman taking on more fluid than she is able to excrete, resulting in a relative drop in serum concentration of sodium. Acute hyponatremia may cause cerebral edema in severe cases, but most cases are asymptomatic. Early signs and symptoms of hyponatremia are often difficult to identify. These include lethargy, agitation, headache, confusion and altered mental state.

Accepted values for normal sodium levels in pregnancy are 130 mmol/L to 140 mmol/L [5,6], due to the dilutional state induced by pregnancy, particularly in the third trimester [1,4,7]. This is due to increased water retention - a result of physiologically increased ADH secretion in pregnancy [8]. Oxytocin, another hypothalamic neuropeptide, has a similar chemical structure to ADH and increases the risk of fluid retention [9]. Compounds with this effect as well as over-judicious administration of fluids, both IV and oral can lead to hypervolemic hyponatremia. In many cases hyponatremia is mild and asymptomatic [1,10].

Previously reported cases outline that administration of hypotonic fluid (e.g. 5% dextrose) significantly increase the risk of dilutional hyponatremia [2,11,12], so there has since been a shift in trends towards more physiological fluids (e.g., Hartmann's solution for fluid resuscitation, and normal saline for oxytocin administration). One Swedish observational study demonstrated that 26% of women who received more than 2500 mL fluid in labor (both oral and intravenous) were hyponatremic in the postpartum period [1,13,14]. Interestingly, in this same group there was no statistically significant relationship between oxytocin augmentation and hyponatremia.

Hyponatremia has been reported in cases of pre-eclampsia in pregnancy. It's hypothesized that release of vasopressin in hypervolemic state in cases of deranged circulating volume in pre-eclampsia and ascites may be related to this pathology. It's important to keep this in mind while investigating for causes of hyponatremia in labor [15,16]. Our case did not have problem with blood pressure or proteinuria in pregnancy or labor and did not exhibit any signs and symptoms of Pre-eclampsia.

It's important to keep a strict fluid balance while being admitted to maternity wards especially for women undergoing induction of labor and in established labor. We have introduced a local guidance to guide fluid management in labor, recognition of hyponatremia and treatment since this case. We would encourage maternity units to devise their local guidance to increase awareness and management of fluid balance in labor to avoid a potentially serious complication of unrecognized hyponatremia for mothers and neonates.

Maternal Hyponatremia risks the cases of neonatal hyponatremia, which can cause fetal hyperbilirubinemia and jaundice, feeding difficulties, respiratory distress and convulsions. It's important to inform pediatrician about maternal hyponatremia in labor so that a neonatal management plan is devised in a timely manner.

Electrolyte disturbances can cause conduction problems in heart. There have been case reports of atrioventricular block with severe hyponatremia which improved following correction of hyponatremia [17].

Electrolyte disturbances can also cause changes in sino-atrial node and pulmonary vein electrical activity leading to atrial fibrillation [18]. An ECG should be offered to women with severe electrolyte disturbance to rule out consequent cardiac arrhythmias.

Pregnancy itself can predispose to arrhythmias without underlying heart disease because of increasing heart rate, circulating plasma volume changes and autonomic and hormonal changes and increased catecholamine.

To our knowledge, this is first case report of occurrence of supra ventricular tachycardia in a woman with concomitant severe hyponatremia. Her subsequent investigations revealed the presence of an accessory pathway which needed further management. This could have triggered her SVT with other biochemical changes in immediate postpartum period.

Treatment of SVT in pregnancy includes vagal maneuvers first and it fails to terminate tachycardia, adenosine is usually used as first line. Metoprolol or verapamil can be used in pregnancy too.

## Learning Points

1. Accurate fluid balance is important in labor as women are at high risk of dehydration as well as fluid overload.
2. Hyponatremia is a potentially life-threatening complication for women and their neonates. It's important to recognize, diagnose and treat accordingly.
3. Neonatologist should be informed of maternal hyponatremia for timely management of potential neonatal hyponatremia.
4. Multidisciplinary management of these cases is important such as medical specialist, anesthetist, ITU specialist, nephrologist and cardiologist.

## References

1. Moen V, Brudin L, Rundgren M, Irestedt L. Hyponatremia complicating labour--rare or unrecognized? A prospective observational study. BJOG. 2008;116(4):552-61.
2. Singhi SC, Chookang E. Maternal fluid overload during labour; transplacental hyponatraemia and risk of transient neonatal tachypnoea in term infants. Arch Dis Child. 1984;59(12):1155-8.

3. Valerio E, Fantinato M, Giovannini IAB, Baraldi E, Chiandetti L. Severe asymptomatic maternal antepartum hyponatremia leading to neonatal seizures: Prevention is better than cure. *Matern Health Neonatol Perinatol.* 2015;1:25.
4. Lind T. Fluid Balance during Labour: A Review. *J R Soc Med.* 1983;76(10):870-5.
5. Nelson-Piercy C. *Handbook of Obstetric Medicine.* 5<sup>th</sup> ed. CRC Press.2015.
6. Abbassi-Ghanavati M, Greer LG, Cunningham FG. Pregnancy and laboratory studies: A reference table for clinicians. *Obstet Gynecol.* 2009;114(6):1326-31.
7. Hytten FE, Klopper AI. Response to a water load in pregnancy. *J Obstet Gynaecol Br Commonw.* 1963;70:811-6.
8. Lindheimer MD, Davison JM. Osmoregulation, the secretion of arginine vasopressin and its metabolism during pregnancy. *Eur J Endocrinol.* 1995;132(2):133-43.
9. Li C, Wang W, Summer SN, Westfall TD, Brooks DP, Falk S, et al. Molecular mechanisms of antidiuretic effect of oxytocin. *J Am Soc Nephrol.* 2008;19(2):225-32.
10. Smith JG, Merrill DC. Oxytocin for induction of labor. *Clin Obstet Gynecol.* 2006;49(3):594-608.
11. Bilek W, Dorr P. Water intoxication and grand mal seizure due to oxytocin. *Can Med Assoc J.* 1970;103(4):379-82.
12. McKenna P, Shaw RW. Hyponatremic fits in oxytocin-augmented labors. *Int J Gynaecol Obstet.* 1979;17(3):250-2.
13. Williams DM, Gallagher M, Handley J, Stephens JW. The clinical management of hyponatraemia. *Postgrad Med J.* 2016;92(1089):407-11.
14. Guideline for the Prevention, Diagnosis and Management of Hyponatraemia in Labour and the Immediate Postpartum Period. GAIN. 2017.
15. Anglim B, Levins K, Busmann N, Imcha M. Severe hyponatraemia associated with pre-eclampsia. *BMJ Case Rep.* 2016;2016bcr2016215036.
16. Magriples U, Laifer S, Hayslett JP. Dilutional hyponatremia in preeclampsia with and without nephrotic syndrome. *Am J Obstet Gynecol.* 2001;184(2):231-2.
17. Kottwitz J, Akdis D, Duru F, Heidecker B. Severe hyponatremia leading to complete atrioventricular block. *Am J Med.* 2016;129(10):e243-4.
18. Lu YY, Cheng CC, Chen YC, Lin YK, Chen SA, Chen YJ. Electrolyte disturbances differentially regulate sinoatria node and pulmonary vein electrical activity: a contribution to hypokalemia- or hyponatremia-induced atrial fibrillation. *Heart Rhythm.* 2016;13(3):781-8.