Section: Biochemistry

Case Series

SIADH COMPLICATED PRE-ECLAMPSIA: A RARE CASE SERIES

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ABSTRACT

Background: SIADH (Syndrome of Inappropriate Antidiuretic Hormone Secretion), though rare, can be fatal in pre-eclampsia for both mother and fetus. It causes severe hyponatremia which can increase the risk of maternal mortality. We report three cases of women aged 20, 25 and 29 years who were admitted to JIPMER, Puducherry hospital after being diagnosed with pre-eclampsia and in whom hyponatremia was detected after preliminary blood investigation. The cases were investigated to find out the reason of hyponatremia.

Materials and Methods: For a period of six months, all cases who were admitted with pre-eclampsia in JIPMER Gynaecology and Obstretics Department and in whom hyponatremia was found in the third trimester were studied. Pre-analytical errors were ruled out and work up for hyponatremia was done.

Results: Of the 56 pre-eclampsia reports we reviewed over six months, only 7 had hyponatremia. On work up, 4 cases were ruled out as pseudohyponatremia. Other three were found to have SIADH.

Conclusion: Severe hyponatremia is an early indicator of SIADH in pre-eclampsia. Thus proper work up of hyponatremia in pre-eclampsia is necessary to reduce mortality.

Keywords: SIADH, Pre-eclampsia, Hyponatremia.

INTRODUCTION

Pre-eclampsia is one of the leading complications of pregnancy, responsible for 18% maternal mortality that arises after 20 weeks of pregnancy and is characterised by elevated blood pressure and proteinuria. If left untreated, it may result in seizures, at which point, it is known as eclampsia.

In pre-eclampsia patients, serum magnesium levels have been low in many cases.[1]

However, hyponatremia in pre-eclampsia is rare and the mechanism has not been widely explored.[2,3] The workup of such cases to understand the precise mechanism of hyponatremia in pre-eclampsia is necessary to properly manage the case as these conditions may predispose to convulsions, maternal mortality and fetal damage.[2]

Here, we have reported three cases of women aged 20, 25 and 29 years who were admitted to JIPMER hospital after being diagnosed with pre-eclampsia. Hyponatremia was detected after preliminary blood investigation. The cases were investigated to find out the causes of hyponatremia in these cases for proper and timely management.

CASE DESCRIPTION

Of the 56 pre-eclampsia reports we reviewed over six months, only 7 had hyponatremia. On work up, 4 cases were ruled out as pseudohyponatremia. Other three were found to have SIADH.

Case 1

A 20 year old female, G2P0A1, was diagnosed with early onset pre- eclampsia at 30th week of gestation and shifted to ICU at 34 weeks' gestation, with headache and preterm premature rupture of membrane (PPROM). Her blood pressure was 160/98mm Hg in both arms and urine dipstick test revealed proteinuria 3+. Her biochemical parameters were initially normal after admission but investigations after being shifted to ICU (highlighted in bold[Table 2]) revealed serum Sodium was 108mEq/L, urine osmolality was 450mosm/kg, while

serum osmolality was 254mOsm/L. Patient was given restricted volume (50ml/hr) of normal saline (0.9% isotonic sodium chloride) infusion and magnesium sulphate injections i.m.. After 24 hours of NaCl infusion and oral fluid restriction serum sodium rose to 121mEq/L and finally became normal after 48hours.

Her biochemical findings are summarized in [Table 2].

Table 1: Summary of Patient Findings

Case 1	**Case 2**	**Case 3**
20 year old female G2P0A1	25 year old female, G1P0A0	29 year old female, G3P1A1
Early onset pre-eclampsia at 30th week of gestation	Early onset pre-eclampsia at 30th week of gestation	Early onset pre-eclampsia at 32th week of gestation
Shifted to ICU at 34 weeks'gestation,with headache and preterm premature rupture of membrane (PPROM)	Shifted to ICU at 33 weeks' gestation, with headache, blurring of visionand shortness of breath	Shifted to ICU at 33 weeks' gestation, with persistent headache, whole body oedema and preterm premature rupture of membrane
BP:160/98 mm Hg in both arms	BP: 158/94 mm Hg in both arms	BP: 154/96mmHg in both arms
Restricted volume(50ml/hr) of normal saline (0.9% isotonic sodium chloride) infusion and magnesium sulphate injections i.m. given		
Serum sodium normal after 48 hours	Serum sodium normal after 72 hrs	Serum sodium normal after 48 hours

Table 2: Biochemical Report of Case 1

Biochemical Parameters						
Sodium (136-146 mEq/L)	137	125	119	**108**	121	136
Potassium (3.5-5.5mEq/L)	4.18	4.2	4.05	**4.02**	4.1	4.08
Urea (17-43mg/dl)	18	20	17	**18**	22	20
Creatinine (0.6-1.2mg/dl)	0.76	0.73	0.8	**0.83**	0.78	0.79
Total Protein (6.6-8.3g/dl)	5.5	5.3	4.7	**4.2**	4.8	4.9
Total Cholesterol (<200mg/dl)	167	170	168	**165**	171	170
Triglyceride (<150mg/dl)	155	160	156	**158**	155	157
Random Glucose (<140mg/dl)	115	120	116	**122**	125	127
Serum Osmolality (285-295mOsm/Kg)	287	280	270	**254**	272	283
Cortisol (12-50µg/L in 3rd trimester)	32			**38**		
TSH (0.38-4.04 µIU/ml in 3rd trimester)	3.210					
Urine protein (Dipstick)				**3+**		
Urine osmolality (50-1200 mOsm/Kg)				**450**		352
Spot Urine Sodium (15-267mEq/L)				**42**		28

Case 2

A 25 year old female, G1P0A0, was diagnosed with early onset pre eclampsia at 30th week of gestation and shifted to ICU at 33 weeks' gestation, with headache, blurring of vision and shortness of breath. Her blood pressure was 158/94mm Hg in both arms and urine dipstick test revealed proteinuria 3+. Her biochemical parameters were initially normal after admission but investigations after being shifted to ICU (highlighted in bold[Table 3] revealed serum Sodium was 114 mEq/L, urine osmolality was 420mosm/kg while serum osmolality was 252mOsm/L. Patient was given restricted volume (50ml/hr) of normal saline (0.9% isotonic sodium chloride) infusion and magnesium sulphate injections i.m..After 48 hours of NaCl infusion and oral fluid restriction serum sodium rose to 126mEq/L and finally became normal after 72 hours.

Her biochemical findings are summarized in [Table 3].

Table 3: Biochemical Report of Case 2

Biochemical Parameters						
Sodium (136-146 mEq/L)	135	121	**114**	119	126	136
Potassium (3.5-5.5mEq/L)	4.25	4.22	**4.11**	4.05	4.2	4.22
Urea (17-43mg/dl)	26	22	**18**	19	20	20
Creatinine (0.6-1.2mg/dl)	0.8	0.75	**0.84**	0.81	0.77	0.85
Total Protein (6.6-8.3g/dl)	4.8	4.9	**3.9**	4.1	4.6	5.1
Total Cholesterol (<200mg/dl)	163	173	**164**	166	170	174
Triglyceride (<150mg/dl)	157	155	**160**	152	158	150
Random Glucose (<140mg/dl)	120	125	**115**	122	130	124
Serum Osmolality (285-295mOsm/Kg)	287	270	**252**	260	275	285
Cortisol (12-50µg/L in 3rd trimester)	40			42		
TSH (0.38-4.04 µIU/ml in 3rd trimester)	2.673					
Urine protein (Dipstick)			**3+**			
Urine osmolality (50-1200 mOsm/Kg)			**420**			372
Spot Urine Sodium (15-267mEq/L)			**39**			30

Case 3

A 29year old female, G3P1A1, was diagnosed with early onset pre-eclampsia at 32nd week of gestation and shifted to ICU at 33 weeks' gestation, with persistent headache, whole body oedema and preterm premature rupture of membrane (PPROM). Her blood pressure was 154/96mm Hg in both arms and urine dipstick test revealed proteinuria 3+. Her

biochemical parameters were initially normal after admission but investigations after being shifted to ICU (highlighted in bold[Table 4]) revealed serum Sodium was 113mEq/L, urine osmolality was 477mosm/kg while serum osmolality was 266mOsm/L. Patient was given restricted volume (50ml/hr) of normal saline (0.9% isotonic sodium chloride) infusion and magnesium sulphate injections im. After 24 hours of NaCl infusion and oral fluid restriction serum sodium rose to 125mEq/L and finally became normal after 48hours.

Her biochemical findings are summarized in [Table 4].

Table 4: Biochemical Report of Case 3

Biochemical Parameters						
Sodium (136-146 mEq/L)	139	133	126	**113**	125	137
Potassium (3.5-5.5mEq/L)	3.9	4.1	4.05	**4.03**	4.12	4.08
Urea (17-43mg/dl)	22	25	17	**18**	21	23
Creatinine (0.6-1.2mg/dl)	0.72	0.76	0.73	**0.75**	0.79	0.74
Total Protein (6.6-8.3g/dl)	5.2	5.3	4.9	**4.2**	5.1	5.5
Total Cholesterol (<200mg/dl)	177	171	167	**170**	179	176
Triglyceride (<150mg/dl)	152	155	150	**158**	153	157
Random Glucose (<140mg/dl)	110	116	122	**128**	122	127
Serum Osmolality (285-295mOsm/Kg)	288	283	274	**266**	273	280
Cortisol (12-50µg/L in 3rd trimester)	27			**31**		
TSH (0.38-4.04 µIU/ml in 3rd trimester)	2.298					
Urine protein (Dipstick)				**3+**		
Urine osmolality (50-1200 mOsm/Kg)				**477**		322
Spot Urine Sodium (15-267mEq/L)				**46**		27

DISCUSSION

The precise mechanism of hyponatremia in pre-eclampsia is still debatable.[4] The causes are elicited in details below:

1. Pseudohyponatremia: It is an uncommonly encountered laboratory abnormality where serum sodium concentration is <135mEq/L but serum osmolality is normal (285-295mOsm/Kg). Electrolyte exclusion effect is the main cause for this inaccurate measurement of sodium and it is seen when samples are analysed by indirect ion selective electrode method which is the common working principle for analysis of electrolytes by auto-analysers. It happens when the samples do not have the normal 93% water phase and 7% solid phase. Instead the solid phase percentage is increased in these samples.This may happen if there is an increased cholesterol components in blood,[5] raised serum triglyceride or high levels of protein in serum.
2. Hyperglycemia: In hyperglycemia, since glucose is a largely extracellular molecule, there is serum hypertonicity. It leads to shifting of water to intravascular space causing dilution of extracellular ions like sodium (dilutional hyponatremia).[6,7] The proposed formula for corrected Na in hyperglycemia is: Na+{1.6(Glucose-100)/100}.
3. SIADH

Our cases had normal total serum cholesterol level and mildly raised triglyceride level and has hypoproteinemia. The patients are euglycemic as per their fasting blood glucose reports and bedside glucometer test. Hence pre analytical errors like electrolyte exclusion effect due to lipemia and hyperproteinemia, hyperglycemia induced pseudohyponatremia were ruled out.

Serum osmolality in each of the cases was low (<275mOsm/Kg) and urine sodium was >30mEq/L & urine osmolality was >100mOsm/Kg signifying euvolemic hyponatremia.

Differential diagnoses for euvolemic hyponatremia are psychogenic polydipsia, hypothyroidism, cortisol deficiency, renal failure and SIADH.

Normal TSH levels, serum cortisol, urea creatinine ruled out the former causes and SIADH was our diagnosis of exclusion.

The exact mechanism of SIADH in pre-eclampsia is still not fully understood.[8,9] However, it has been hypothesized that increased renal sensitivity to ADH coupled with non-osmotic ADH secretion in pre-eclampsia may cause hyponatremia.

Another reason may be defective placenta in such cases do not secrete sufficient Vasopressinase, the enzyme which inactivates ADH.

Other theories suggest that premature contractions and preterm labour stimulate the placental unit and uterine myometrium to produce pulsatile release of ADH and oxytocin under the influence of ovarian steroids. Frequency of release increases with progression of labour. If either the pulsatile nature or the amount of secretion of Oxytocin or ADH is altered as in pre-eclampsia, it can precipitate SIADH.[10]

CONCLUSION

Pre-eclampsia in pregnancy contributes to 10% maternal mortality and requires early management but when it is coupled with SIADH induced hyponatremia, chances of patient mortality increases further. Although occurrence of SIADH is rare in pre-eclampsia, it must be considered as a possible cause when hyponatremia is noticed in pre-eclampsia patients. Early detection through correlating history and clinical features with biochemical reports can

lead to timely treatment and can prove to be life saving for the patient.

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