

# Cardiac Dysrhythmia- An Unusual Complication Of Severe Preeclampsia

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## Abstract

We report a novel association between postpartum paroxysmal supraventricular tachycardia and severe pre-eclampsia in a 22 year old Indian primipara without pre-existing heart disease. The patient was successfully treated with intravenous adenosine. The mechanism by which maternal placental syndromes like severe pre-eclampsia increases the risk for cardiac arrhythmia is probably due to the persistence of altered myocardial geometry and hypomagnesemia in these women.

## INTRODUCTION

Paroxysmal supraventricular tachycardia (PSVT) is one of the most common arrhythmias encountered in pregnancy. It is seen mostly in the third trimester of pregnancy and the postpartum period and has a frequency of 24/100000 pregnancies [1]. Patients with maternal placental syndromes like severe preeclampsia are more prone to develop premature cardiac failure and dysrhythmias [2]. We report a rare case of postpartum paroxysmal supraventricular tachycardia in a young primiparous lady without pre-existing heart disease whose antenatal period was complicated by severe preeclampsia.

## CASE REPORT

A 22 year old second gravida with one previous abortion presented to the Obstetric outpatient department of a tertiary care hospital in India at 33 weeks and 2 days gestation with complaints of headache of 2 days duration. There was no history of blurring of vision, vomiting, epigastric pain or decreased urine output. She perceived fetal movements well.

She has been married non-consanguineously for 3 years. Two years prior to the present pregnancy, she had a spontaneous miscarriage during the first trimester. One year later, she underwent evaluation for secondary infertility and conceived after the first cycle of ovulation induction with clomiphene citrate. She had regular antenatal care from 12 weeks of gestation. All her blood pressure recordings were normal. The ultrasound scan done at 20 weeks showed a morphologically normal fetus. She was suspected to have intrauterine growth restriction from 32 weeks of gestation

and an ultrasound done for assessing growth showed a 1.4 kg fetus with normal amniotic fluid index and umbilical artery doppler.

At 33 weeks and 2 days gestation, her blood pressure was found to be 170/110 mm of Hg in the right upper limb in sitting position. The urine dipstick method showed 4+ albuminuria. Ultrasound scan done then showed a single live fetus with an estimated weight of 1.4 kg with absent end diastolic flow on umbilical artery Doppler. The preeclampsia workup like platelet count, creatinine and liver enzymes were normal.

She was started on anti-eclampsia prophylaxis with 4 gram loading dose of magnesium sulphate followed by 1 gram/hour infusion (Modified Pritchard's intravenous regime). The first dose of betamethasone was given to hasten fetal lung maturity. Four hours after the first dose of steroid, electronic fetal monitoring showed category III non-reassuring fetal heart rate pattern and hence she was delivered by emergency lower segment caesarean section under spinal anaesthesia, a 1.22 kg girl baby with I minute Apgar score of 9. The baby was admitted in nursery for preterm care.

She was monitored in labour room in the immediate postpartum period and received Magnesium sulphate prophylaxis for 24 hours. As her blood pressure was persistently high even after delivery, she was started on 2.5 mg of ACE inhibitor, Enalapril twice a day. As the blood pressure was still uncontrolled, 100 mg of labetalol was also added twice daily on the second postoperative day. Her

sutures were removed on postoperative day 6 and she was discharged on postoperative day 8 with blood pressure under control. Her baby was in the nursery at the time of her discharge.

She came to the emergency department on postoperative day 11 with complaints of giddiness and palpitation of 4 hours duration. On examination, she was found to have tachycardia with a heart rate of 180 beats per minute and her blood pressure was 120/70 mm of Hg. Bedside ECG was suggestive of supraventricular tachycardia. A Cardiology opinion was sought and she was administered 12 mg of adenosine intravenously with which her arrhythmia resolved. The antihypertensives were tapered and stopped as her blood pressure was normal. Her thyroid function tests and serum electrolytes were normal. Echocardiography ruled out any underlying cardiac lesion. She was started on beta-blockers and was planned for cardiac electrophysiological studies followed by radiofrequency ablation later.

### **DISCUSSION**

The arrhythmogenic effect of pregnancy has already been established in literature. The increased prevalence of tachyarrhythmias in pregnancy is attributed to the normal pregnancy related haemodynamic, autonomic and hormonal changes and to the increased surveillance. The mild hypokalemia associated with pregnancy, elevated levels of plasma catecholamines, increased adrenergic receptor sensitivity of myocardium due to hyperoestrogenic state, intravascular volume expansion leading to atrial stretch and increased end-diastolic volumes contribute to this effect [3]. This case report highlights the novel association between cardiac dysrhythmias and severe preeclampsia.

Maternal placental syndrome includes gestational hypertension, pre-eclampsia, eclampsia, placental abruption and infarction. It occurs as a result of defective placentation. In pre-eclampsia which is characterized by hypertension and proteinuria, the ischaemic placenta releases anti-angiogenic factors causing endothelial dysfunction leading to multisystem involvement [4].

The common complications of severe preeclampsia in the mother include eclampsia, pulmonary oedema, renal failure, thrombocytopenia, cerebral haemorrhage, hepatic failure and abruptio placentae leading to disseminated intravascular coagulation. Though there are many retrospective studies proving that women with preterm preeclampsia are more prone to develop premature cardiac failure and dysrhythmias one year after delivery [2], the association between cardiac

arrhythmias and severe preeclampsia in the puerperal period is less reported.

The association between pre-eclampsia and chronic cardiovascular disease was first described by Corwin and Herrick in 1927 which has been confirmed by several observational studies since then [5]. The ill-effects of preeclampsia on cardiovascular system does not end with the delivery of baby and placenta as once thought. A large retrospective study by Ray et al demonstrated a 61% increase in the relative risk for dysrhythmias and heart failure 1 year after delivery in women with maternal placental syndromes [2]. The risk was found to be higher if there was associated fetal compromise like intrauterine growth restriction, intrauterine death or preterm birth. They postulated that the higher prevalence of cardiac arrhythmias in women when compared to men later in life may be linked to these adverse pregnancy outcomes. Hence they recommended frequent blood pressure and weight measurements from 6 months postpartum and lifestyle modifications in women with maternal placental syndrome.

In a pre-eclamptic pregnancy, there is left atrial enlargement due to increased atrial natriuretic peptide and concentric and eccentric ventricular remodeling. This altered myocardial structure and geometry do not always revert to normal after delivery and this predisposes to premature cardiac disorders in these women. The unfavourable vascular and metabolic changes persist after a pre-eclamptic pregnancy in spite of the normalization of blood pressure [6]. Asymptomatic left ventricular dysfunction was significantly more in preterm pre-eclampsia (56%) when compared with term pre-eclampsia (14%) or matched controls (8%) at 1 year postpartum [7].

The other causes of paroxysmal supraventricular tachycardia like hyperthyroidism, underlying valvular heart disease, excessive caffeine intake, use of sympathomimetic drugs for tocolysis and electrolyte imbalance like hyperkalemia and hypomagnesemia were ruled out in our patient by relevant history and investigations. Among the various causes described, hypomagnesemia is interestingly associated with both cardiac arrhythmias and pre-eclampsia-eclampsia syndromes [8]. Evidence supports the use of Magnesium sulphate for anti-seizure prophylaxis in severe pre-eclampsia as well as for the treatment of eclampsia. Similarly, magnesium supplementation increases the chance of conversion of atrial fibrillation to sinus rhythm. Hence it is hypothesized that hypomagnesemia may be the reason for the association between cardiac arrhythmia and severe pre-

eclampsia. Unfortunately, the measurement of serum total magnesium is affected by a number of factors like low albumin levels, haemolysis, acidosis etc and this may be misleadingly normal. The measurement of ionized magnesium is not universally acceptable and available [8]. However, this association is only an observation and further research is recommended to confirm the relationship between hypomagnesemia, cardiac arrhythmias and maternal placental syndromes.

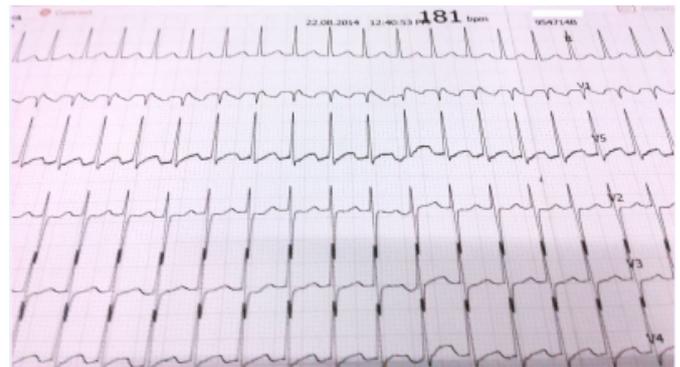
Our patient was successfully treated with adenosine which is an ultrashort acting endogenous purine nucleotide that temporarily depresses sinus node activity and slows atrio-ventricular conduction. Adenosine is the drug of choice for supraventricular tachycardia during pregnancy and lactation due to its rapid onset of action, short half-life (less than 10 seconds), fewer side effects and high efficacy [3]. Vagal manoeuvres like Valsalva and carotid massage can be tried to control supraventricular tachycardia before pharmacological intervention. If long term therapy is needed, beta-blockers, verapamil or digoxin may be used. Recurrent episodes warrant electrophysiological studies to delineate the accessory pathway followed by radiofrequency ablation under fluoroscopic guidance. But radiofrequency ablation during pregnancy is contraindicated due to radiation exposure.

### CONCLUSION

We report a case of supraventricular tachycardia as an unusual complication of severe pre-eclampsia in the postpartum period in a lady without pre-existing heart disease. The possible mechanism by which maternal placental syndromes like severe preeclampsia increases the risk for cardiac arrhythmia is uncertain. It is postulated that hypomagnesemia and persistence of the altered myocardial geometry in these women predispose them to cardiac arrhythmia. The novel association between hypomagnesemia, cardiac arrhythmias and severe preeclampsia is an exciting area for future research.

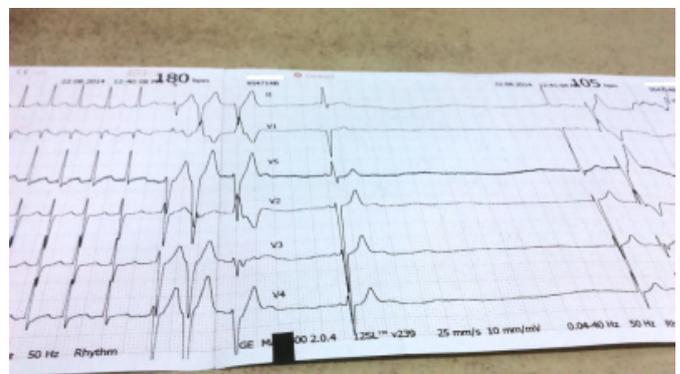
**Figure 1**

ECG showing supraventricular tachycardia



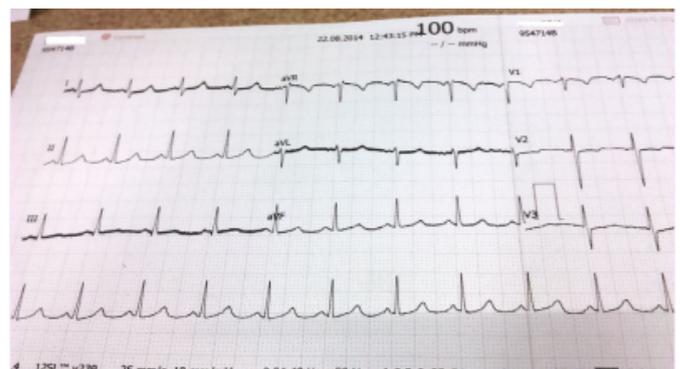
**Figure 2**

ECG while administering adenosine



**Figure 3**

ECG post-adenosine with a heart rate of 100 bpm.



### References

1. Jiang-Ming Li, Carol Nguyen, Jose A Joglar, Mohammed H Hamdan, Richard L Page: Frequency and outcome of arrhythmias complicating admission during pregnancy. *Clin.Cardiol* 2008,11:538-41
2. Ray JG, Schull MJ, Kingdom JC, Vermeulen MJ: Heart failure and dysrhythmias after maternal placental syndromes: HAD MPS study. *Heart* 2012, 98:1136-41.
3. Tan HL, Lie KI: Treatment of tachyarrhythmias during pregnancy and lactation. *Eur Heart J* 2001, 22: 458-64
4. Levine RJ, Karumanchi SA. Preeclampsia, a disease of the maternal endothelium: the role of antiangiogenic factors and

implications for later cardiovascular disease. *Circulation* 2011;123:2856-69

5. Corwin J, Herrick WW. Relation of hypertensive toxemia of pregnancy to chronic cardiovascular disease. *Journal of the American Medical Association* 1927; 88:457-9

6. Garovic VD, Hayman SR. Hypertension in pregnancy: an

emerging risk factor for cardiovascular disease. *Nat Clin Pract Nephrol* 2007;3:613-22

7. Melchiorre K, Sutherland GR, Liberti M, Thilaganathan B. Pre-eclampsia is associated with persistent postpartum cardiovascular impairment. *Hypertension* 2011; 58:709-15

8. Dacey MJ. Hypomagnesemic disorders. *Crit Care Clin* 2001 ;17: 155-73

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