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# RESEARCH ARTICLE

## MULTIDISCIPLINARY MANAGEMENT ON PAROXYSOMAL SUPRAVENTRICULAR TACHYCARDIA IN PREGNANCY-BALANCING MATERNAL HEMODYNAMIC STABILITY AND FETAL SAFETY

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## ABSTRACT

Pregnancy is known to be associated with an increased incidence of arrhythmias; more benign forms of arrhythmias, such as premature atrial and ventricular contractions, demonstrate a higher burden during pregnancy compared with prepregnancy. Proarrhythmic mechanisms of pregnancy are presumably related to cardiovascular, autonomic, and hormonal changes. Increased circulating catecholamines , chronotropic effects of relaxin, mechanical effects of atrial stretch, increased ventricular end-diastolic volume caused by intravascular volume expansion, and hormonal and emotional changes all contribute to proarrhythmia . Perhaps the normal but mild hypokalemia of pregnancy or the physiological rise in heart rate serves to arrhythmias. Supraventricular tachycardia (SVT) is a tachyarrhythmia characterized by a heart rate above 120 beats per minute (BPM). Patients with SVT present with the following symptoms:-palpitations, shortness of breath, chest pain, hemodynamic instability, or possibly asymptomatic. The increase in cardiac output and the increase in resting heart rate during pregnancy predispose pregnant women to SVT. The management of SVT in pregnancy, although remarkably similar, varies slightly based on the trimester of pregnancy. Atenolol and verapamil are effective methods of treating SVT, which can be used during the second and third trimesters. Both medications are contraindicated in the first trimester. At the same time, intravenous adenosine can be used in all three trimesters, including labor. Electrical cardioversion is an effective treatment method for hemodynamically unstable or drug-refractory patients, which has proven to be safe in all three trimesters, including labor but can result in pre-term labor in the third trimester. Non-fluoroscopic ablation proved to be the only treatment method that definitively resolved SVT without recurrence.



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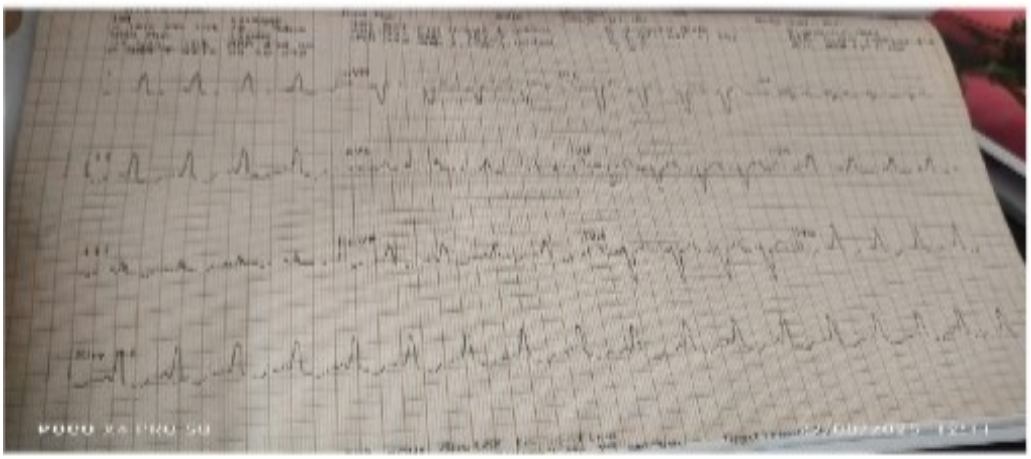
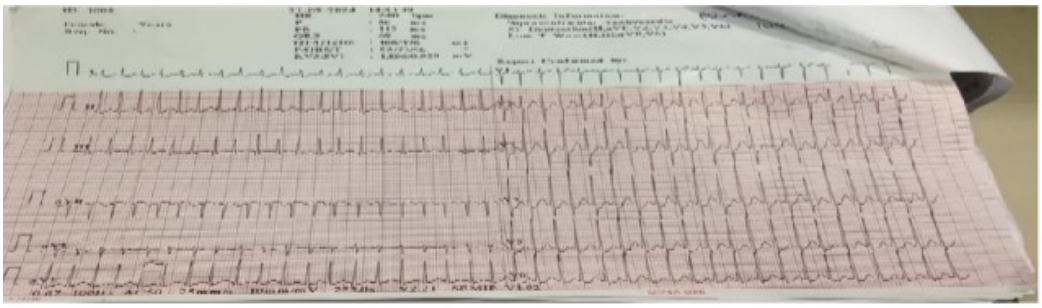
# INTRODUCTION

Supraventricular tachycardia (SVT) is a tachyarrhythmia that involves atrial tissue or atrioventricular junctional tissues This tachyarrhythmia is characterized by a heart rate above 120 beats per minute (1,2).The speed and functionality of theconduction pathways determines the type of supraventricular tachycardia (1). There are approximately four types of SVT; however, atrioventricular reentrant tachycardia (AVRT) and atrioventricular nodal reentrant tachycardia (AVNRT) account for the two most common forms .At presentation, the electrocardiogram (ECG) may display narrow QRS complexes with P waves that may be subtle or possibly buried within the ST-segment, inverted P waves, or bizarre QRS complexes (1,2). Additionally, SVT can consist of multiple P wave morphologies, short PR intervals, or undetectable P waves.SVT can be paroxysmal, defined as an abrupt onset and cessation of arrhythmia .Many pregnant patients presenting with this arrhythmia have no previous congenital heart disease or cardiac structural abnormalities.

Heart rate progressively increases during pregnancy, resulting in a 10%-25% increase from prepregnancy values. Cardiac output rises early in the first trimester and peaks in the second trimester, increasing by 45% . Estrogen and relaxin which stimulate nitric oxide production, increases peripheral arterial compliance and decreases vascular resistance. Normal pregnancy is associated with decreased parasympathetic and increased sympathetic activity at rest. Increased sympathetic activity may contribute to abnormal automaticity, reentry, or triggered activity.

**Electrocardiographic Changes of Pregnancy:** Electrocardiographic changes have been observed in pregnancy-prolonged maximum P-wave duration is observed (4). The PR interval may shorten, and a leftward shift in the QRS axis may be seen. Prominent Q waves in the inferior leads and flat and/or inverted T waves in leads III and V1-V3 are more frequent in pregnancy.QT interval is expected to be shorter; however, QT and QTc intervals are longer in the second and third trimester of pregnancy compared with





**Clinical spectrum and management of acute arrythmias**

| S No | Obstetric score | Complaints | ECG | Echocardiogram | Management | Obstetric outcome | Neonatal outcome | Maternal outcome |
|---|---|---|---|---|---|---|---|---|
| 1 | G4P2L2A1 | Palpitations | SVT | Normal | Tab Metaprolol 25 mg BD | LSCS | Alive,term | Discharged |
| 2 | P1L1 | Chest pain, palpitations | wpw syndrome and narrow complex tachycardia | No structural abnormality | INJ Adenosine iv stat ↓ Inj Amiodarone infusion ↓ Synchronised Cardioversion ↓ Reverted to normal sinus rhythm ↓ Tab Amiodarone 100 mg bd | Labour natural | Alive term | Discharged |
| 3 | G4P2L2A1 | Palpitations | SVT | NORMAL | Tab Metaprolol 25 mg BD | LSCS | Alive,term | Discharged |
| 4 | G2P1L1 | Palpitations | Supraventricular tachycardia | NORMAL | Carotid massage ↓ Inj adenosine iv stat ↓ Tab Metaprolol 25 mg BD | Labour Natural | Alive ,term | Postnatal uneventful |
| 5 | G2P1L1 | Giddiness palpitations | SVT | S/P DOUBLE VALVE REPLACEMENT | INJ Amiodarone infusion ↓ Tab amiodarone BD | Spontaneous expulsion | | Discharged |

nonpregnancy, although they are still within normal range.Maximum QTc and the T-peak to T-end interval, an indicator for dispersion of repolarization and sympathetic activation , increase by the third trimesterpatients with an underlying propensity for repolarization abnormalities, pregnancy could be a period of vulnerability for cardiac arrhythmias.

# METHODOLOGY

This case series obtained from the Department of obstetrics and gynaecology, government kap viswanatham medical college and mgmgh, trichy 2024-2025

**Inclusion Criteria:** Patient presented with supraventricular tachycardia. Availability of complete clinical data

**Case series**

**Case -1**

A 23 year-old, G3P2L1, prev 2 LSCS,with past history of gestational hypertension . Booked and immunised at kulumani PHC.mother was apparently normal and had regular antenatal visits at kulumani PHC. At 5 months of gestation she had complaints of palpitations for which ECG done showed Supraventricular tachycardia, she was conscious,her blood pressure was 100/70 mmHg. Echocardiogram showed no structural abnormality. Serum Electrolytes done and found to be normal. As per cardiologist opinion, Patient was started on Tab Diltiazem 30 mg BD. She was on antiarrhythmic drugs throughout her antenatal period.A term alive boy baby delivered by Emergency repeat cesarean section at 37 weeks of gestation.postoperative period patient hemodynamically stable

and continued on tab diltiazem bd.Postop period- As per cardiologist advice TAB DILTIAZEM 30 mg BD continued.

**Case 2**

A 22 year-old, P1L1, Labour natural, PND-4 referred from Perambalur GH as Newly diagnosed GTHN with complaints of chest pain, palpitations. Antenatal history- primigravida, booked and immunised at Perambalur, admitted in Perambalur GH at 38 weeks of gestation in view of gestational hypertension. She delivered an alive term boy baby by labour natural at Perambalur GH. On the Postnatal day 4, she had chest pain and palpitations -ECG taken showed supraventricular Tachycardia H R 240/min, spontaneously reverted to sinus rhythm. Patient was on continuous monitoring. Again within half an hour, supraventricular tachycardia seen & patient started on Adenosine 6 mg IV STAT and referred to MGMGH.Our team of obstetricians, cardiologist and anaesthetist were kept ready- At MGMGH,on receiving the patient ,pt conscious, BP-90/60 mmHg,Heart rate showed 240 bpm ECG shows narrow QRS complex Tachycardia Ventricular bigemini/ SVT Heart rate 240 / mt. BP-90/70 mmHg. Echocardiogram done-structurally normal.SVT reverted with Adenosine 12 mg.Again patient developed SVT. DC shock 100 J given and reverted. Serum electrolytes done-normal.Patient was started on Inj AMIODARONE 150 mg iv bolus (in 100ml of 5% dextrose) followed by 360mg IV infusion and then continued with Tab Amiodarone 100 mg BD. She was diagnosed with WPW Syndrome- wolff-parkinson-white syndrome. She was discharged with antiarrhythmic drugs Tab Amiodarone 100 mgBD on postnatal day 24

**Case 3**

A 30 year-old, G4P2L2A1/ prev 2 LSCS, Booked and immunised at Thennur PHC.regular Antenatal visits at PHC, At 22 weeks of gestation patient had complaints of palpitations, patient got admitted at MGMGH, ECG showed supraventricular tachycardia Heart rate 220 beats per minute-Cardiologist opinion obtained- Echocardiogram done and found to be normal- no structural abnormality.Serum electrolytes -normal.Patient spontaneously reverted to normal sinus rhythm. Patient was started on Tab Metaprolol 25 mg Bd and was continued throughout the antenatal period. Baby delivered by repeat cesarean section at 38 weeks of gestation and postoperative period was uneventful. Mother was advised to continue tab Metaprolol 25 mg BD and discharged on 13th postoperative day.

**Case -4**

A 23 year-old, G2P1L1, previous NVD, Booked and Immunised at Sirugambur PHC. Regular Antenatal visits at PHC At 26 weeks of gestation, patient was referred fromSirugambur PHC with complaints of chest pain and palpitations to our hospital.on examination pt conscious, Heart rate-230/mt,BP-90/60 mmHg.ECG showed HR-230 / mt, supraventricular tachycardia.Cardiologist called over given,Carotid massage given which failed. Then Inj.Adenosine 6mg iv given - rhythm reverted to normal.Serum electrolytes done and found to be normal.Patient was on continuous ECG monitoring and started on tab metoprolol 25 mg bd.Patient was advised tab Metaprolol 25 mg Bd and continued throughout her antenatal period.she delivered an alive term baby by Labour natural.postnatal period was uneventful.mother was advised to continue antiarrhythmic drugs tab Metaprolol 25 mg BD and regular follow up with the cardiologist.

**Case -5**

A 28 year-old,G2P1L1/Prev vacuum/GA-14weeks H/O Right CVA-MCA infarct@2009,H/0 closure of Mitral valvulotomy done@2010/ H/0 Double valve replacement done with TTK CHITHRA DISC PROSTHESIS&Tricuspid sector-annuloplasty done@2014/Acute pulmonary edema (recovered)/Acute prosthetic valve-thrombus-Thrombolysis done/Embolus at bifurcation of CFA&DFA-Transfemoral embolectomy done 2025 Prev H/0 admission at MGMGH at 9weeks of gestation and patient was advice for termination in view of S/P DVR on Anticoagulants. But patient was not willing for termination and hence discharged. Pt readmitted with complaints acute onset breathlessness, ECG showed supraventricular tachycardia, heart rate 270 beats per minute, BP-90/60 MMHg. Echocardiogram done -cardiologist opinion obtained. patient started on Inj Amiodarone 150 mg iv bolus followed by 360 mg iv infusion over 6 hours and then 540 mg iv infusion over 18 hours and then continued on oral anti arrhythmic drugs.Patient had Spontaneous expulsion.she was continued on antiarrythmic drugs and anticoagulants during her admission period. Patient was discharged with antiarrhythmic drugs and advised to have regular follow up in cardiology department.

# DISCUSSION

## REVIEW OF LITERATURE

**First Trimester:** The first trimester consists of the first 13 weeks of pregnancy. During the first trimester of pregnancy, numerous physiological changes occur within the body. As mentioned previously, the blood volume nearly doubles increasing cardiac output (9). The fetus's safety should be heavily considered during this trimester as organogenesis takes place within the fifth to tenth week of gestation (1,9). The fetus is extremely vulnerable to congenital disabilities, and the mother is highly susceptible to spontaneous abortion during this period (21). In cases in which the patient is hemodynamically unstable, the immediate benefit of the treatment outweighs the possible teratogenic effects. Based on a thorough examination of the literature, vagal maneuvers are considered the best initial treatment for SVT (9). This non-pharmacological treatment is well-tolerated in the first trimester. Vagal maneuvers consist of carotid sinus massage, Valsalva, and facial ice immersion. This form of treatment should be used in hemodynamically stable patients. Although vagal maneuvers are considered extremely safe during pregnancy, it may not be the most effective treatment. In a study conducted by Jian-Ming Li *et al*., SVT was terminated with Valsalva maneuver in 3% of patients (22). If vagal maneuvers do not resolve the SVT, pharmacological treatments must be considered.Adenosine is the first-line pharmacological agent to treat SVT. Adenosine is the first-line pharmacological agent to treat SVT. Intravenous (IV) adenosine is unlikely to enter fetal circulation because it has a tremendously short half-life of 10 seconds. This medication is typically administered intravenously with 6 mg to 12 mg. This medication has been successful in 84% of cases (23). Beta-adrenergic blockers, such as propranolol and metoprolol, are anti-arrhythmia drugs that can be first-line for outpatient

treatment and second-line for acute treatment (12). Digoxin has been used in the first trimester of pregnancy and is relatively safe (23). If the patient's arrhythmia is drug-refractory or if the patient is hemodynamically unstable, electric cardioversion can be used to terminate SVT. Electric cardioversion has been proven to be safe in all stages in life-threatening circumstances.

**Second Trimester:** According to the American College of Obstetricians and Gynecologists (ACOG), the second trimester occurs between the 14th and 27th week of gestation . During this stage of pregnancy, the fetal heart rate can be auscultated, and fetal malformations can be detected through ultrasound, and fetal movement is detected by the mother . Lack of fetal movement can be the mother's first indication of a fetus's problem, unlike the first trimester. There are fewer limitations in treatment during the second trimester because organogenesis is completed by this pregnancy stage (8). The risk of spontaneous abortion and fetal loss is substantially lower than in the first trimester (21). Electrical cardioversion is safe to use in the second trimester. It continues to be reserved for patients who fail to respond to medications or become hemodynamically unstable. Electrical cardioversion is generally successful between 50 J to 100 J Catheter ablations are generally indicated for treatment-refractory SVT. This procedure is safe to administer during the second trimester of pregnancy.

**Third Trimester:** According to ACOG, the third trimester of pregnancy occurs between the 28th and 40th week of pregnancy. During the final trimester of pregnancy, the fetus's respiratory system becomes fully developed, and the fetus has increased viability. In a study conducted by **Li *et al*.,** it was determined that a large percentage of patients developed SVT during the third trimester (29). An arrhythmia's likelihood remains high due to major hemodynamic changes that impact the body (30).

Additionally, the increased heart during the third trimester predisposes patients to any arrhythmia (8). Adenosine is the first-line pharmacological agent to treat SVT. Intravenous (IV) adenosine is unlikely to enter fetal circulation because it has a tremendously short half-life of 10 seconds. This medication is typically administered intravenously with 6 mg to 12 mg. This medication has been successful in 84% of cases (23). Beta-adrenergic blockers, such as propranolol and metoprolol, are anti-arrhythmia drugs that can be first-line for outpatient treatment and second-line for acute treatment (12). Digoxin has been used in the first trimester of pregnancy and is relatively safe (23). If the patient's arrhythmia is drug-refractory or if the patient is hemodynamically unstable, electric cardioversion can be used to terminate SVT.

**During Labour**

SVT can present during labor**. Romem *et al*.** conducted a study that determined cardiac arrhythmias occur more often during labor (31). The cause of SVT during labor is multifactorial. Common triggers of SVT during labor are the catecholamine release, electrolyte disturbances such as hyperkalemia, and vasopressors administered to treat post-epidural hypotension (13). The cardiac output of a pregnant woman at term increases to approximately 10 liters at term (16). As previously stated, the increased cardiac output results in stretches on myocardial tissue developing into this tachyarrhythmia (9,10).

**Summary and Discussion**

Both mother and fetus are at risk when SVT occurs during pregnancy. Pregnancy may predispose to and exacerbate symptoms of SVT which are shortness of breath, palpitations, dizziness and presyncope. Clinical assessment of vital signs and 12-lead ECG investigation are mandatory for an accurate diagnosis of arrhythmia (7,9) .Echocardiography is essential to exclude structural and functional heart diseases as the presence of organic heart diseases is an important risk factor for arrhythmias during pregnancy. Early involvement of cardiologist is recommended to diagnose SVT and to detect any underlying etiology which can be life-threatening. Close collaboration between the cardiologist and the obstetrician is important throughout the pregnancy as well as puerperium to develop care strategies for potential recurrences of SVT(3,7,9). The acute management of SVT in pregnancy .. The decision must be taken with appropriate consideration of both maternal and fetal factors. Monitoring of both mother and fetus should be continued during acute treatment. In stable patients, noninvasive maneuvers like carotid massage or Valsalva maneuver with simultaneous positioning the patient in the left lateral position, administering 100 % oxygen and establishing intravenous access should be first attempted. In case of failure with physical procedures, first-line pharmacological treatment is adenosine, followed by low dose of β-blockers. Second choice is verapamil but only after the first trimester of pregnancy and only in acute circumstances. When drugs fail or in case of life-threatening symptoms such as shock and pulmonary edema, ECV is indicated.

**Conclusion**

Episodes of SVT occur with increased frequency during pregnancy particulary in third trimester.Potential risk factor for SVT in pregnancy is underlying congenital or structural heart disease . In most cases, there is no history of heart disease.
In a study of women with asymptomatic or mildly symptomatic WPW syndrome, half developed SVT for the first time and the other half experienced an increase in attack rate ( kounis, 1995).In some patients, acessory pathway ablation may be needed.Physical treatment like sinus carotid massage or Valsalva maneuvers followed by drug therapy is tried in hemodynamically stable patients. In cases of failure of above measure or when there is hemodynamic compromise, electrical cardioversion or invasive method like radiofrequency ablation is justified. SVT in pregnancy is associated with adverse fetomaternal outcomes.Prompt recognition and appropriate management are vital.A patient centered,evidence-based approach coupled with timely intervention ,ensures optimal outcomes and minimizes complications.

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