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The Safety of Cardioversion in Supraventricular Tachycardia Patient with Pregnancy

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ABSTRACT

Background. Supraventricular tachycardia is a type of tachyarrhythmia characterized by sudden changes in heart rate and increases rapidly. In supraventricular tachycardia, abnormalities that occur include components of the conduction system and occur in the upper part of the HIS bundle. This case report describes a case of supraventricular tachycardia suspected of AVNRT in a pregnant woman accompanied by hypokalemia, hypocalcemia and prolonged QT Interval along with cardioversion which was performed as the management of this case. **Case presentations**. A woman, Mrs Y, age 24, a housewife, having her address within the city of Palembang. The patient came to the emergency department of the Moh. Hoesin Hospital in Palembang with the chief complaint of chest palpitations. On physical examination, there was a grade 2/6 murmur, no shortness of breath, the patient was 7 months pregnant. He had a history of heart disease SVT 8 years ago. ECG examination showed sinus rhythm with low atrial rhythm in leads II, III, AVF. On laboratory examination, the blood calcium level was 8.3 mg/dL. Management of this patient includes pharmacological and non-pharmacological management. On the 14th day of hospitalization, 100 joules of cardioversion were performed and the patient responded well. The patient's condition improved on the 20th day of treatment. **Conclusion**. The patient experienced a good and effective response to 100 joules of cardioversion which was performed on the 14th day of treatment. Maintenance treatment in the form of drugs in the form of diltiazem due to the condition of the patient who is 27 weeks pregnant. The next management is planning the birth process according to the patient's hemodynamics and preventing the occurrence of SVT in subsequent pregnancies.

1. Introduction

Supraventricular tachycardia (SVT) is a type of tachyarrhythmia characterized by a sudden increase in heart rate, ranging from 150 to 280 per minute. SVT triggers originate in tissue above the ventricular level (e.g. SA node, AV node, or HIS bundle). There are three basic mechanisms for the occurrence of SVT, namely automation, triggered activity, and reentry. Most SVTs have a normal QRS complex. This disorder often occurs in pregnancy due to increased heart burden, especially in trimesters 2-3, fever, stress, physical activity, heart failure, or valve abnormalities.[1-3]

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common variant of SVT tachycardia in daily practice, with a prevalence rate of 1:1 for male and female. AVNRT is divided into three types, namely the typical type (slow-fast), slow, and slow. In the typical type (slow-fast) AVNRT the prevalence is 80-90% of cases. In the fast-slow type AVNRT the prevalence is 10% of cases. In the late type of AVNRT the prevalence is 5% of cases.[2-5]

Atrioventricular reciprocating tachycardia is the presence of a large giant re-entrant loop in the normal

conduction system along with accessory pathways, which occurs in patients with the WPS syndrome. When tachyarrhythmias occur, the hallmarks of the WPW syndrome are lost because they combine with normal pathways to form a large re-entry circuit.[2-5]

SVT with aberrancy is a variant of SVT with impaired impulses to the ventricular fibers caught in a different form than usual, conduction disturbances due to the recovery/refractory period of the bundle branch occurring at the same time, as seen in the BBB pattern.[2]

The incidence and prevalence rates by age group for SVT are about 35 cases in 100,000 population annually, with a prevalence of 2.25 cases in 1000 population. SVT can occur at any age, but is more common in young adults without heart disease.[1-3]

Ectopic atrial rhythm is an action potential disturbance because the action potential is generated by atrial cells faster than the sinus (SA) node in other words, this ectopic focus takes over the pacemaker of the heart and this rhythm is not a rescue rhythm when the rate > 100 is called Atrial tachycardia is a variant of SVT with a focus originating from atrial cells, this picture is rarely found in ASD surgery patients who cause atrial scarring so that it is over-reactive when there are 3 forms of P wave morphology called multifocal atrial tachycardia.[1-4]

Prolonged QT interval or prolonged QT interval (LQTS) is a corrected prolonged QT interval (QTc) on the surface of the ECG and is associated with a trigger for TdP, a polymorphic form of ventricular tachycardia that can cause sudden death. LQTS can be congenital or acquired.[4.5]

Hypocalcemia is plasma calcium less than 8.8 mEq/L. Hypocalcemia can occur due to inadequate intake, increased calcium requirements in pregnancy, especially from the 2nd trimester to the 3rd trimester of pregnancy. Meanwhile, hypokalemia is a plasma potassium level of less than 3.5 mEq/L. Hypokalemia can result from inadequate intake, movement of potassium into cells, or renal or non-renal potassium loss. 6-8 This case report describes a case of supraventricular tachycardia suspected of AVNRT in a pregnant woman accompanied by hypokalemia, hypocalcemia and prolonged QT Interval along with cardioversion measures performed as the management of this case.

2. Case Presentation

A woman, Mrs Y, 24 years old, a housewife, having her address in the city of Palembang. The patient came to the emergency department of Moh.Hoesin Hospital Palembang with the chief complaint of chest palpitations that had been getting worse since ±13 hours before admission to the hospital. On physical examination, there was a grade 2/6 murmur, no shortness of breath. Physical examination revealed no carpopedal spasm and no Trousseau's sign. The patient had a history of SVT heart disease 8 years ago. Based on family history, there was no history of diabetes mellitus, hypertension and heart problems in the family.

The ECG showed supraventricular tachycardia (SVT) with hidden P-wave Pseudo r' visibility in V1 and V3 so that it can be concluded that the suspected AVNRT is atypical type with slow-fast wave appearance. After the conversion of the patient's ECG wave, an EKG was repeated and the results were sinus rhythm with low atrial rhythm in leads II, III, AVF. Ultrasonography (USG) examination of the abdomen showed a single live intrauterine fetus, the placenta was located anteriorly, enough amniotic fluid, the impression was 27 weeks pregnant, a single live intrauterine fetus. On laboratory examination, the blood calcium level was 8.3 mg/dL. The patient was then diagnosed as supra ventricular tachycardia (SVT) stable, suspect atrioventricular nodal reentrant tachycardia (AVNRT) typical (slowly fast), G1P0A0 27 weeks pregnant.

Management of this patient includes pharmacological and non-pharmacological management. Pharmacological management includes diltiazem 30 mg/24 hours, metoprolol 5 mg dissolved in 0.9% NaCl intravenously over 10 minutes, and calcium tablets 500 mg/12 hours. Management of non-pharmacological form of O2 intranasal as much as 3 liters / minute, vagal maneuvers, low-oxalate diet and bedrest. The patient was treated in the cardiovascular intensive care unit (CVCU).

Follow-up

Following are the results of the follow-up carried out during the patient's intensive care at the cardiovascular care unit (CVCU), before cardioversion, after cardioversion and treatment on days 19-20 in the hospital (table 1-3).

Table 1. Examination results before cardioversion

Treatment	Day 14
Subjective complaints	Heart palpitations, chest pain and weakness.
General physical examination	Compos mentis examination, blood pressure 80/60 mmHg, pulse rate of 190 beats / minute, respiratory rate 22 breaths / min, temperature 36.6^{0}C, body weight 58 kg, height 156 cm, O^2 saturation of 99%.
Special physical examination	Heart rate 90 beats/minute, regular I/II heart sounds, grade 2/6 systolic murmur, punctum maximum ICS V, hardens during expiration, spreads to the left axilla, there is splitting of physiological inspiration.
Heart auscultation	
Abdomen	Convex, uterine fundus height tinggi umbilicus-processus xiphoideus (29 cm), right back, bottom of buttocks, fetal heart rate 155 beats/minute.
Investigation	Electrocardiography (ECG) Pre cardioversion: supra ventricular tachycardia, prolonged QT interval Cardioversion 100 Joule: supra ventricular tachycardia, prolonged QT interval Post cardioversion: supra ventricular tachycardia, prolonged QT interval
assessment with	SVTAVNRT suspect typical (slow-fast), *Low Atrial Rhythm*, hypocalcemia, hypokalemia, prolonged QT Interval, G1P0A0 34-35 weeks pregnant singleton live.
Therapy	Non-pharmacological: bed rest, vagal maneuver, cardioversion 100 Joules, diet high in calories and low in oxalate 1800 kcal Pharmacological: calcium tablets 500 mg/12 hours, KSR 600 mg/24 hours, metoprolol 5 mg in

Table 2. The condition of the patient after cardioversion

Treatment	Day 14-15
Subjective complaints	Heart palpitations.
General physical	Compos mentis examination, blood pressure 100/60 mm Hg, pulse rate of 181 beats / minute, respiratory rate 22 breaths / min, temperature 36.6^{0}C, body weight 58 kg, height 156 cm, O2 saturation of 99%.
Specific physical examination	
Cardiac auscultation	Heart rate 90 beats/minute, regular I/II heart sound, no murmur.
Abdomen	Convex, uterine fundal height umbilicus-processus xiphoideus (29 cm), right back, bottom of buttocks, fetal heart rate 155 beats/minute.
Lab examination	Electrocardiography (ECG) Sinus tachycardia, low atrial rhythm in Leads II, III, AvF
Diagnosis	SVT suspected AVNRT typical type (slow-fast), Low Atrial Rhythm, hypocalcemia, hypokalemia, prolonged QT Interval, G1P0A0 34-35 weeks pregnant single live.
Therapy	Non-pharmacological: bed rest, vagal maneuvers, diet high in calories and low in oxalate 1800 kcal Pharmacological: calcium tablet 500 mg/12 hours, KSR 600 mg/24 hours, metoprolol 5 mg in 100 cc NaCl 0.9% in 10 minutes intravenous, midazolam 2.5 mg intravenously in 5 cc 0.9% NaCl 5 min slow bolus.

Table 3. Follow-up treatment on 19-20 days

Treatment	day 19-20
Subjective complaints	No palpitations.
General physical	Compos mentis examination, blood pressure 100/60 mm Hg, pulse rate of 114 beats / minute, respiratory rate 22 breaths / min, temperature 36.5^{0}C, body weight 58 kg, height 156 cm, O2 saturation of 99%.
Special physical examination	Heart rate 90 beats/minute, regular I/II heart sound, no murmur.
Cardiac auscultation	
abdomen	Convex, uterine fundal height umbilicus-processus xiphoideus (29 cm), right back, bottom of buttocks, fetal heart rate 155 beats/minute.
Lab examination	Electrocardiography (ECG) Sinus tachycardia, low atrial rhythm in Leads II, III, AvF.
Diagnosis	SVTsuspected typical type AVNRT (slow-fast), Low Atrial Rhythm, hypocalcemia, hypokalemia, prolonged QT Interval, G1P0A0 34-35 weeks pregnant singleton live.
Therapy	
	Non-pharmacological: rest, diet high in calories and low in oxalate 1800 kcal Pharmacological: calcium tablet 500 mg/12 hours, KSR 600 mg/24 hours

3. Discussion

Supraventricular tachycardia is a type of tachyarrhythmia characterized by sudden changes in heart rate and increases fast. Changes in heart rate in infants with SVT generally range from 220 to 280 beats/minute. children older than 1 year are generally slower, which ranges from 180 to 240 x/minute. In supraventricular tachycardia, abnormalities that occur include components of the conduction system and occur in the upper part of the bundle of His. The ECG features of SVT mostly have a normal QRS complex.[2-4]

SVT is caused by several factors. The first cause can be idiopathic, which is often found in most patients, infants and children. Wolf Parkinson White syndrome occurs in 10-20% of cases and occurs only after conversion to sinus arrhythmias. This syndrome is a syndrome with a short PR interval and a wide QRS interval, caused by a direct connection between the atria and ventricles via an auxiliary pathway. Other causes can include congenital heart disease and pregnancy.

AVNRT is one of the most common types of SVT. Most of these types of AVNRT have no abnormalities in the heart. However, there are abnormalities in the heart that can cause AVNRT, including: mitral regurgitation, pericarditis, myocardial infarction. AVNRT arises because of the presence of a reentrant loop connecting the AV node and the atrial tissue. In patients with this type of tachycardia, the AV node has two conduction pathways, namely the fast conduction pathway and the slow conduction pathway. The slow conduction pathway, which lies parallel to the tricuspid valve, allows a reentrant loop as a pathway for new electrical impulses through the pathway, exiting the AV node retrogradely (i.e., backward from the AV node into the atria) and anterogradely (i.e., forward to or from the AV node). AV to ventricle) at the same time. Due to concomitant atrial and ventricular depolarization, P waves are rarely seen on the ECG, although atrial depolarization will occasionally produce a P wave at the end of the QRS complex in lead V1. [5-6]

AVNRT is divided into three types, namely the

typical type (slow-fast), slow, and fast-slow, the typical type AVNRT picture we see through the ECG manifestations P waves are lost in visibility and are seen as Pseudo r' at the end of QRS, RP very tight intervals, at V1, II, III, AvF. We can see the picture of fast-slow type AVNRT through ECG manifestations, namely the P wave is clearly visible without disappearing and there is a retrograde P wave before the QRS (QRS-PT).[7]

Based on history, the complaints of the patients was chest thumping is very strong and shortness of breath. Heart palpitations have been felt continuously since the last 13 hours. The patient vomited once and sweated profusely. This is consistent with the theory that the symptoms of Supraventricular Tachycardia (SVT) include palpitations more than 96%, dizziness 75%, shortness of breath 47%, fainting 20%, chest pain 35%, fatigue-23%, diaphoresis 17%, and nausea. 13%. Supraventricular tachycardia has a sudden onset and termination of regular palpitations. This patient has a history of heart disease SVT 8 years ago and the patient is in a state of fatigue due to the solid activity experienced by the patient which may be a trigger for SVT.

Based on the physical examination and supporting examinations in the patient, a diagnosis of supraventricular tachycardia was established in that patient, where SVT is a type of tachydysrhythmia characterized by changes in heart rate that suddenly increase rapidly with the patient's heart rate being above 140 beats per minute. electrical impulses originating above the ventricles of the heart. Abnormalities in SVT involve components of the conduction system and occur in the upper part of the HIS bundle.[8,9]

Management obtained in patients in the ED is like an emergency cardiac situation, the gold standard ABC (airway, breathing, circulation) must be followed in the emergency management of SVT. A rapid examination of the patient's airway, breathing, and circulation should be performed, and all vital signs should be documented. Then a vagal maneuver was performed and there was no improvement. After that, given metoprolol 5 mg bolus in NaCl 0.9% 100 cc discharged in 10 minutes, and suppressed the occurrence of complex ventricular activity. It was also successful in 71% of patients in which it was combined with propranolol.[10-15]

Initial strategies for terminating PSVT are generally vagotonic maneuvers, such as carotid sinus massage. However, the clinician should evaluate the patient for the presence of a carotid bruit (abnormal sound) before attempting this maneuver, especially in a patient elderly. The Valsalva maneuver or possible immersion of the face in cold water can also be tried. This method serves to increase vagal tone, which can prolong AV nodal refractoriness to the point of AV block thereby ending the tachycardia. It should be noted that vagotonic maneuvers will not stop atrial tachycardia, but they can create temporary AV blocks, clarifying the underlying mechanism by allowing visualization of the P waves. [15-18]

If these efforts are unsuccessful in terminating the SVT, the next step in treatment is pharmacological intervention. In some cases of SVT, calcium channel blockers and -blockers may be useful. Metoprolol was given in this case considering the lower risk of IUGR because the patient was not in the first trimester of pregnancy, Verapamil was the most commonly used drug as an alternative to adenosine. Verapamil is particularly useful if adenosine is contraindicated or if the SVT ends quickly but recurs immediately. Amiodarone as antiarrhythmic therapy is avoided because it is a pregnancy category D drug. 8-10 Evaluation is still carried out by performing an ECG per day viewed from a monitor attached to the patient. The results showed an improvement in SVT where the HR decreased to normal which was <100 beats/minute and the QRS complex was normal and the p wave was starting to appear. [17,18]

From the anamnesis it is also possible to obtain an estimate of the etiology of supraventricular tachycardia suspected of AVNRT. Clinical manifestations in this case, including symptoms, physical examination, ECG recording and changes in fetal health are highly dependent on the degree and cause of this disease. The disease can be asymptomatic or have minimal symptoms for many years. Cardiac examination

revealed a grade 2 systolic murmur, echocardiographic examination revealed a minimal pericardial effusion, typical AVNRT (slow-fast).[19-20]

Initial management of this case was carried out by vagal maneuvers, but the patient's response was not effective, followed by pharmacotherapy given metoprolol, the patient's response was refractory, hemodynamic signs were found to be unstable, cardioversion was carried out starting from 30 Joules to 50 Joules and 100 Joules. In the next attack of SVT, a Vagal maneuver was performed on the patient, with a less effective response, a non-dihydropyridine CCB drug was administered, namely diltiazem.[6]

In patients with gestational age at term, maintaining the pregnancy may be considered. The choice of termination of pregnancy is highly dependent on the patient's maternal condition, namely hemodynamic stability, refractory to vagal maneuvers, refractory to pharmacotherapy, and refractory to cardioversion. In hemodynamically unstable patients who are refractory to pharmacotherapy or cardioversion, as well as fetal distress, an emergency abdominal delivery can be performed.[21]

The results of laboratory examinations in this patient showed the presence of hypocalcemia and hypokalemia. Hypokalemia was defined as plasma potassium less than 3.5 mEq/L. Hypokalemia can result from inadequate intake, movement of potassium into cells, or renal or non-renal potassium loss. The clinical picture of potassium depletion is highly variable, and its severity depends on the degree of hypokalemia. ECG changes due to hypokalemia do not match plasma potassium concentrations. Initial changes include flattening or inversion of the T wave, prominent U wave, depression of the St segment and lengthening of the QT interval. Severe potassium depletion can cause PR interval prolongation, low voltage, and widening of the QRS and increase the risk of ventricular arrhythmias. [23,24]

4. Conclusion

The patient had a good and effective response to 100 joules of cardioversion which was performed on the 14th day of treatment. Maintenance treatment in the form of drugs in the form of diltiazem due to the condition of the patient who is 27 weeks pregnant. The next management is planning the birth process according to the patient's hemodynamics and preventing the occurrence of SVT in subsequent pregnancies.

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