Initial Management of Paroxysmal Supraventricular Tachycardia with ST-Segment Depression in Primary Care Setting

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ABSTRACT

Introduction: Supraventricular tachycardia (SVT) is a common cardiac emergency that can be life-threatening. Symptoms may include palpitations, chest pain, and difficulty breathing. **Case:** A 50-year-old male with sudden palpitations since one hour ago, accompanied by chest pain and breathing difficulty. A 12-lead ECG revealed an SVT rhythm with ST-segment depression in leads II, III, aVF, and V2-V6. After initial interventions, which included carotid massage, Valsalva maneuver, and oral bisoprolol, the rhythm converted to sinus rhythm within an hour. ST-segment depression may occur during SVT, which warrants evaluation for ischemic etiology. **Conclusion:** The report focuses on the initial management of SVT in a primary care setting with constraints on equipment and referral facilities. It's important to note that changes in the ST segment in SVT do not always indicate myocardial infarction, and further examination is necessary to confirm the diagnosis.

Keywords: Bisoprolol, supraventricular tachycardia, Valsalva maneuver.

ABSTRAK

Pendahuluan: Takikardia supraventrikuler (SVT) adalah keadaan darurat jantung yang dapat mengancam jiwa. Gejala SVT dapat termasuk jantung berdebar, nyeri dada, dan kesulitan bernapas. **Kasus:** Laki-laki berusia 50 tahun dengan keluhan jantung berdebar sejak satu jam, disertai nyeri dada dan sulit bernapas. Pada EKG 12-sadapan didapatkan ritme SVT dengan depresi segmen ST pada sadapan II, III, aVF, dan V2-V6. Setelah intervensi awal termasuk pijat karotis, manuver Valsalva, dan bisoprolol oral, ritme SVT menjadi ritme sinus dalam satu jam. Depresi segmen ST dapat terjadi selama SVT, yang memerlukan evaluasi etiologi iskemia. **Simpulan:** Laporan ini berfokus pada tata laksana awal SVT di layanan kesehatan primer dengan keterbatasan peralatan dan fasilitas rujukan. Penting untuk diperhatikan bahwa perubahan segmen ST pada SVT tidak selalu mengindikasikan infark miokard, dan pemeriksaan lebih lanjut diperlukan untuk memastikan diagnosis. **Kevin Fernando Suhardi, Franky Kurniawan, Lusiani. Tata Laksana Awal Takikardia Supraventrikular Paroksismal dengan Depresi Segmen ST di Layanan Primer.**

Kata Kunci: Bisoprolol, takikardia supraventrikular, manuver Valsalva.

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INTRODUCTION

Supraventricular arrhythmia is a common type of tachyarrhythmia with a significant impact on the quality of life. This type of arrhythmia originates in the atrium or atrioventricular (AV) node or above the level of the bundle of His. One of the most common types of supraventricular arrhythmia is supraventricular tachycardia (SVT).[1] The prevalence of SVT is 2.25 per 1,000 individuals, and the incidence is 35 per 100,000 person-years; it is more common in females, with a ratio of 2:1 in all age groups.[2] Data on the prevalence of SVT in Indonesia is not available.

SVT is characterized by tachycardia with a narrow QRS interval of 100 ms, a frequency of 150-220 beats per minute, and a regular ventricular response on the electrocardiogram (ECG).[3] SVT can be classified into two subtypes: paroxysmal and persistent. Paroxysmal SVT (PSVT) is characterized by sudden onset and rapid termination of regular tachycardia, while persistent SVT refers to a continuous or prolonged episode that may require medical intervention to restore normal sinus rhythm. Three mechanisms behind SVT are atrioventricular nodal reentrant tachycardia (AVNRT), atrioventricular reentrant tachycardia (AVRT), and atrial tachycardia (AT).[4] Almost 80% of AVNRT or AVRT cases can be detected through ECG.[5] About 60% of PSVT cases are due to AVNRT, while around 20% are due to AVRT. Atrial tachycardia is rare. AVNRT is more common in adults over 20 years of age, while AVRT is more prevalent in the pediatric population due to congenital abnormalities of accessory circuits.[6]

Individuals aged over 65 years are at a higher risk of experiencing SVT compared to younger individuals. A cohort study in the United States on 1,754 patients who underwent catheter

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ablation for SVT found that the mean patient age was 45 years with a standard deviation of 19 years.[7] The majority were female (62%). The study also observed that the incidence of AVNRT increased with age, while AVRT decreased. Other risk factors for SVT include a history of myocardial infarction, heart failure, heart surgery, and heart valve disease.[8]

The management of SVT and other tachyarrhythmias has undergone rapid changes in the last decades. The 2020 Advanced Cardiac Life Support provided clear guidelines for treating stable and unstable SVT patients.[9] However, even with trained medical staff, the optimal treatment of SVT may not always be achieved because the availability of drugs and equipment, such as cardioversion and adenosine, which are the mainstays of SVT management, may be limited, particularly in primary care. This case report reviews the initial management of SVT in primary care with limitations of cardioversion equipment and adenosine.

CASE

A 50-year-old male arrived at the emergency department due to sudden heart palpitations that started an hour ago. The symptoms began suddenly before he went to sleep and were accompanied by chest pain like being crushed by a heavy object. The pain spread to his neck and left arm and lasted for more than 15 minutes. He also experienced shortness of breath, sweating, and weakness. The patient had a similar experience two years ago, but not as severe. The patient was previously diagnosed with myocardial infarction and hypertension by a cardiologist. No history of angioplasty. Bisoprolol 2.5 mg, captopril 12.5 mg, and aspirin 80 mg were prescribed but have not been taken for over a year.

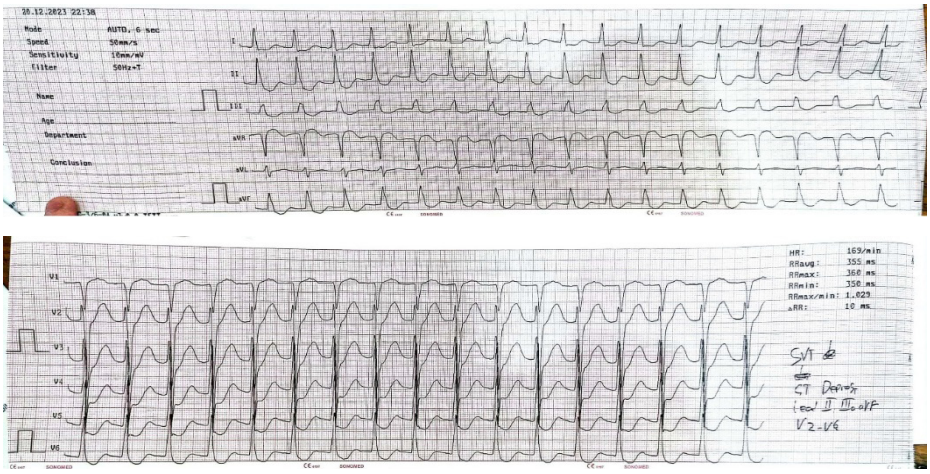


Figure 1. ECG during supraventricular tachycardia. The ECG shows a narrow-complex tachycardia with a heart rate of 169 beats per minute. There is also notable ST segment depression in Lead II, III, aVF, and V4-V6. ECG speed 50 mm/second.

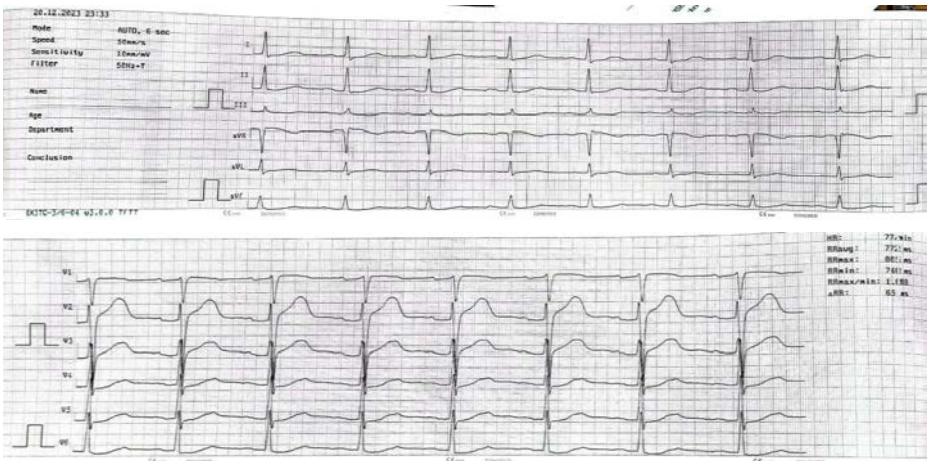


Figure 2. ECG after 1 hour of carotid massage, Valsalva maneuver, and oral bisoprolol shows sinus rhythm with persistent ST depression in V4-V6. ECG speed 50 mm/second.

On physical examination, blood pressure is 158/106 mmHg, pulse is 170 beats/minute and regular, respiratory rate is 24 breaths/minute and regular, SpO2 is 94% on room air, and temperature is 36.8°C. On cardiac examination, the ictus cordis was visible and palpable 1 finger lateral to the left midclavicle line, S1 and S2 were regular, there was no murmur, there was no S3 gallop, and JVP was at 5-2 cmH2O. A 12-lead ECG examination was done 10 minutes after arrival, showed SVT rhythm with a heart rate of 169 beats per minute, normoaxis, narrow QRS with a duration of 0.08", and ST depression in II, III, aVF, and leads V3-V6, normal T wave, and normal QT interval **(Figure 1).** The diagnosis was stable SVT with possible acute myocardial infarction.

The initial treatment included oxygen at 3 L/minute through a nasal cannula and Ringer's lactate infusion at 20 drops/minute. Carotid sinus massage was performed twice for 10 seconds each, but there was no response. Medication was then given, consisting of a 10 mg oral bisoprolol, 5 mg sublingual ISDN, and 160 mg aspirin loading dose. To stimulate the vagus nerve, the patient was instructed to exhale through a closed nose and mouth for 30-60 seconds and to cough and strain frequently. After an hour, the patient's heart palpitations and chest pain were reduced. Blood pressure 110/80 mmHg, pulse 70 beats per minute, respiratory rate 20 breaths per minute, and oxygen saturation 98% with a nasal cannula at 3 lpm. Heart examination revealed normal S1 and S2 sounds, no murmurs, and no gallop S3.

The second ECG examination showed a normal sinus rhythm, with a rate of 77 beats/minute, and normal P waves. The P-R interval was 0.20 seconds, with rSr' waves in lead III and ST depression in leads V4-V6. T waves and QT interval were normal **(Figure 2)**. After the condition was stabilized, the patient was transported by ambulance to an advanced referral health facility with ICCU facilities to receive further evaluation and treatment from a cardiologist.

DISCUSSION

The risk of SVT increases with age. AVNRT is the most common type of SVT in the geriatric population, accounting for almost 80% of cases. The risk factors for SVT in this population are coronary heart disease, heart failure, and cardiomyopathy.[10] Symptoms of SVT include palpitations, chest pain, shortness of breath, lightheadedness, dizziness, and loss of consciousness. Some patients may experience no symptoms. The most common symptom is palpitation, a sensation of heart-pounding or fast beating, fluttering, or racing.[11] Occasionally, patients may experience chest pain and shortness of breath, or "air hunger," due to decreased cardiac output or as part of other comorbidities, such as acute coronary syndrome and heart failure.

SVT occurs due to an abnormal flow of electrical impulses in the heart. AVNRT is facilitated by two distinct electrical pathways with different conduction velocities within the AV node. These pathways are known as the fast pathway and the slow pathway. Re-entry between the fast and slow pathways can disrupt normal atrial and ventricular contractions, resulting in a narrow QRS tachycardia on the ECG. Due to the simultaneous activation of the atria and ventricles, P waves are not easily seen on the ECG. However, the P wave may be visible in AVNRT as a pseudo r' wave in lead V1 or a pseudo S' deflection in inferior leads.[1,5,11] These findings may refer to retrograde P waves visible after the QRS complex.

In AVRT, tachyarrhythmia involves both the AV node and an accessory pathway that connects the atria and ventricles outside the AV node. AVRT with a narrow QRS complex occurs due to anterograde conduction through the AV node and retrograde conduction through the accessory pathway; the electrical impulses travel down the AV node to the ventricles and back to the atria via the accessory pathway, as indicated by an inverted P wave after the QRS wave. This pathway can also cause pre-excitation of the ventricles, which can lead to a delta wave appearance, a widening QRS complex, and a shortened PR interval. The condition most often associated with AVRT is Wolff-Parkinson-White syndrome (WPW), although not all individuals with WPW will experience AVRT.[5,10]

Symptoms of SVT can range from no symptoms to severe, such as chest pain, shortness of breath, and loss of consciousness. In the emergency room, the primary survey (airway, breathing, circulation, disability) is conducted to monitor vital signs and provide initial treatment, such as oxygen (if needed) and IV line access. A complete history and physical examination are performed. Patients with tachycardia of over 150 beats/minute or pulse irregularities should be suspected of tachyarrhythmia; a 12-lead ECG should be performed. The primary survey, including the 12-lead ECG examination, should be possible even in primary health facilities. Other examinations at advanced facilities include complete peripheral blood examination, blood biochemistry, thyroid function examination (TSH, FT4), and transthoracic echocardiography. A 12-lead ECG examination can confirm SVT. ECG evaluation for tachyarrhythmias includes the following: (1) QRS duration; (2) Regularity; (3) Visible P waves; (4) Atrial rate versus ventricular rate; (5) RP interval.[5] ECG of typical AVNRT—the most common subtype of SVT—will show a heart rate >150 beats per minute, regular rhythm, and normal PR interval with a narrow QRS complex. P waves may not be visible. Atypical AVNRT can be identified by a visible p wave after the QRS complex or as a pseudo R' wave in lead V1 or pseudo S' deflection in inferior leads.[1] **Table** shows the differential diagnosis of other tachyarrhythmias. The ECG findings in this patient are consistent with typical AVNRT, as indicated by HR 169x/minute, regularity, an indiscernible P wave, and a narrow QRS complex.

If tachyarrhythmia is still present after the initial treatment, the ACLS 2020 Tachyarrhythmia Protocol by the American Heart Association suggests further acute management (**Scheme**).[9] The patient's ECG shows narrow-QRS tachycardia. This case complained of ischemic chest pain, confirmed by history and ECG findings consistent with myocardial infarction. Depending on the presence of hemodynamic instability, such as hypotension, signs of shock, acute changes in mental status, ischemic chest pain, and acute heart failure, a patient is categorized as stable or unstable. If a patient has even one of these conditions, it is considered unstable and will undergo synchronized cardioversion.

Vagal maneuver is the primary treatment for terminating narrow QRS SVT with stable hemodynamics.[3,8] This maneuver involves various techniques to stimulate the vagus nerve situated in the internal carotid arteries. Stimulating the vagus nerve activates the parasympathetic nerve, which can reduce the heart rate. Some techniques include the Valsalva maneuver and carotid sinus massage. Valsalva maneuver is considered safe with minimal risk and can be performed in primary health facilities. The Valsalva maneuver can effectively treat stable SVT with a 19% to 54% success rate.[12] The maneuver involves blowing into a 20 mL syringe to move the plunger. Currently, the modified Valsalva maneuver method is more commonly used. This technique involves positioning the patient in a semi-recumbent position followed by a supine position and passive leg strain after the Valsalva strain. Studies have shown that the modified Valsalva maneuver is more effective in terminating SVT than the standard Valsalva maneuver (OR 5.52; 95% CI 2.26-13.47; p<0.001).[13] During certain medical procedures, patients may be required to strain for 20-30 seconds or cough repeatedly. Additionally,

Table. Differential diagnosis of tachyarrhytmia.[1,4]

DIFFERENTIAL DIAGNOSIS	R-R REGULARITY	P-WAVE VISIBILITY	ATRIAL RATE > VENTRICULAR RATE	PRE-EXCITATION (DELTA WAVES)
Atrioventricular nodal re-entrant tachycardia (AVNRT)	Yes	Mostly no	No	No
Atrioventricular re-entrant tachycardia (AVRT)	Yes	Mostly no	No	Yes
Atrial tachycardia (AT)	Yes/No	Yes	No	No
Atrial fibrillation (AF)	No	No	Yes	No


carotid sinus massage can be performed by applying pressure unilaterally on the carotid sinus for 5 seconds. It is essential to ensure that the patient's neck position is extended and turned away from the side where pressure is applied.[3]

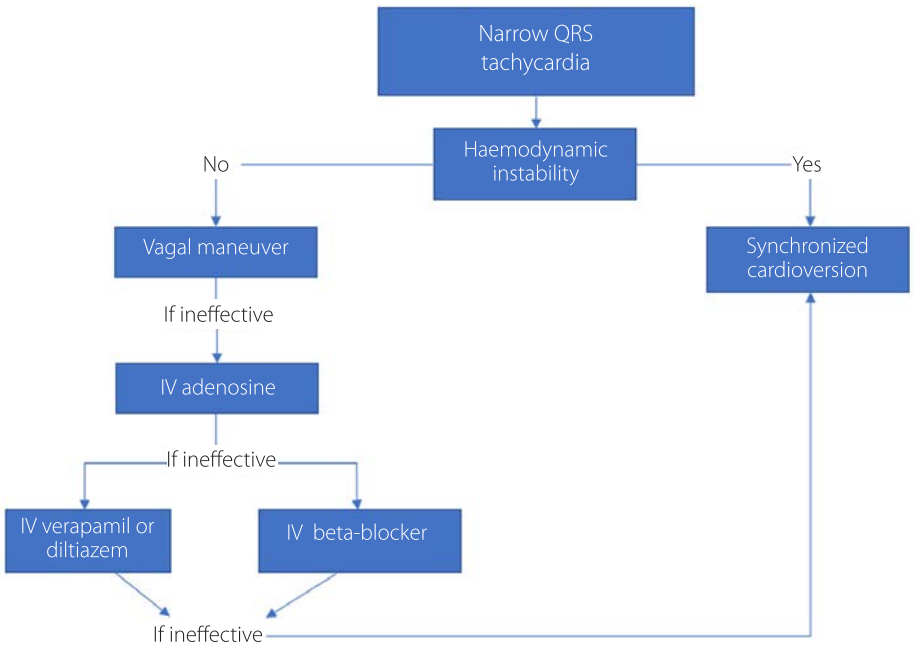
Adenosine is a drug commonly used to treat narrow QRS tachycardia or supraventricular origin, which does not respond to vagal maneuvers.[1,10,11] It is particularly useful if the ECG diagnosis of tachyarrhythmia is unclear. Adenosine works by attaching itself to the adenosine receptor A1R, found in abundance in AV nodal tissue and peripheral vasculature, thus mediating its electrophysiological effect. Stimulation of A1R results in a decrease in cAMP production, which in turn leads to hyperpolarization of target cells. In the SA node and AV node, adenosine causes bradycardia, sinus arrest, and AV block.[13] The initial dose of adenosine is 6 mg intravenously. It is recommended to follow with a flush of 20 mL of normal saline for 1 minute and elevate the hand to enhance absorption. If SVT does not convert within 1-2 minutes, a second dose of 12 mg of adenosine IV can be given.[14] However, adenosine can cause bronchospasm; it is contraindicated for patients with a history of asthma or chronic obstructive pulmonary disease (COPD).[9]

Beta-blockers and calcium channel blockers are recommended as the next step in treatment if IV adenosine and vagal maneuvers are not effective.[15] Beta-blocker works by blocking beta-1 receptors in the SA and AV nodes, leading to a negative chronotropic effect. The European Society Guideline (ESC) recommends using short-acting beta-blockers, such as esmolol and IV metoprolol, for treating SVT.[3] Both these drugs are cardioselective beta-blockers with negative intrinsic sympathomimetic activity (ISA). Bisoprolol is another drug that falls into this category. Despite limited evidence, bisoprolol has an excellent safety profile in hemodynamically stable patients. Recent studies also support the use of bisoprolol in treating supraventricular arrhythmia.[16] However, beta-blockers are not recommended for patients with acute heart failure.[9]

It is common to find ST-segment depression during SVT. Many reports have shown that changes in the ST segment can occur during SVT, but its significance is uncertain.[17,18] This condition can occur in patients with narrow-complex paroxysmal tachycardia without a history of coronary heart disease, especially young patients with no related risk factors. This is known as tachycardia-induced ST-segment depression. In this population, the presence of ST-segment changes with tachycardia has a low possibility of flow-limiting coronary stenosis in the absence of a positive myocardial scintigraphy. The non-ischemic mechanisms of this phenomenon are unclear.[17] This might be due to increased oxygen demand caused by rapid myocardium contraction and reduced diastolic function.[18] In some cases, ST-segment depression during SVT that persists for a few minutes following the termination of the episode may be related to coronary spasms.[19] However, all ST-segment changes accompanied by chest pain that persists even after termination of SVT must be evaluated for possible ischemic etiology.[9]

It is unclear whether SVT is associated with myocardial infarction in this case. The patient's history of myocardial infarction is not sufficient; further examination, such as cardiac biomarkers or cardiac angiography, is necessary. Ideally, treatment for SVT patients with myocardial infarction, especially STEMI, should be done simultaneously. SVT can be treated as soon as possible while preparing the cath lab for immediate revascularization.[17–19]

SVT is a potentially life-threatening condition. In cardiac emergencies, primary health facilities are required to provide first aid to reduce the risk of death. The difference between the management of SVT in a primary facility and the ideal management is the availability of cardioversion, adenosine, monitoring, and a cardiovascular unit. In limited facilities, the main focus is to rely on diagnosis and initial management. SVT diagnosis can be easily made by clinical examination and ECG.[20] Initial management, such as primary survey, administration of available oral medications (beta blockers or nitrates), and vagal maneuvers (including Valsalva maneuver and carotid sinus massage), can be done at primary settings and must be followed by referral to more complete facilities.[21]



Scheme. Algorithm of narrow-QRS tachycardia.[9]
Abbreviation: IV: intravena.

Conclusion

A case of a 50-year-old man diagnosed with paroxysmal supraventricular tachycardia received initial treatment at a primary health facility. The patient's symptoms included heart palpitations and chest pain, and an ECG suggested an AVNRT. Initial management of SVT involves techniques such as carotid massage, Valsalva maneuver, and oral bisoprolol, especially if medical equipment and referral facilities are limited. It's important

to note that changes in the ST segment in SVT do not always indicate myocardial infarction, and further examination is necessary to confirm the diagnosis.

Conflicts of Interest
The authors affirm no conflict of interest in this report.

Acknowledgment
None

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