TREATMENT OF SVT BY ADENOSINE INJECTION *VIA* SUBCLAVIAN CATHETERIZATION

Jakchai Tittabut*, Watchareewan Khuanpet, Patumwan Panyapiang, Sathaporn Chaikaew, Pinmanee Sirimo and Prisana Burirat

Chiang-Dao Hospital, Chiang-Mai province, Thailand

ABSTRACT: The cases of 51-year-old female and 40-year-old male diagnosed with Supraventricular Tachycardia (SVT) were presented. Treatment using peripheral adenosine bolus failed to normalize the SVT. To prevent other cardiac complications due to a long distance referral system and delayed treatment. Prompted infraclavicular approach was done followed by a bolus of 6 mg Adenosine injection *via* subclavian catheter direct to the heart. After adenosine administrations, their vital signs were stable. ECGs were returned to Normal Sinus Rhythm within seconds. After the treatment, their clinical outcomes were satisfied.

Keywords: Supraventricular Tachycardia, SVT, Adenosine injection, subclavian catheterization

INTRODUCTION: Supraventricular tachycardia (SVT) is one of arrhythmia that has an impulse originated above the ventricle. SVT requires only atrial and/or atrioventricular (AV) nodal tissue for its initiation and maintenance. It is usually a narrow-complex tachycardia that has a regular, rapid rhythm. Manifestations of SVT are quite variable, patients may be asymptomatic or they may present with minor palpitations or more severe symptoms.

Paroxysmal supraventricular tachycardia (PSVT) is a regular, fast (160 to 220 beats per minute) heart rate that begins and ends suddenly and originates in heart tissue other than that in the ventricle[1]. PSVT is episodic, with an abrupt onset and termination. It is most common among young people and is more unpleasant than dangerous[2]. It may occur during vigorous exercise. The fast heart rate tends to begin and end suddenly and may last from a few minutes to many hours. It is almost always experienced as an uncomfortable palpitation. It is often associated with other symptoms, such as weakness, light-headedness, shortness of breath, and chest pain. Usually, the heart is otherwise normal. The diagnosis can be confirmed by doing an electrocardiogram (ECG)[3].

History

Because symptomatic severity depends on the presence of structural heart disease and on the hemodynamic reserve of the patient, individuals with PSVT may present with mild symptoms or severe cardiopulmonary complaints. Palpitations and dizziness are the most common symptoms reported by patients with SVT. Chest discomfort may be secondary to a rapid heart rate, and it frequently subsides with the termination of the tachycardia.

Common presenting symptoms of PSVT and their frequency rates are as follows:

Palpitation - Greater than 96%
Dizziness - 75%
Shortness of breath - 47%
Syncope - 20%
Chest pain - 35%
Fatigue - 23%
Diaphoresis - 17%

Patients who are hemodynamically unstable should be resuscitated immediately with cardio-version. An ECG should be performed as soon as possible.

Physical Investigation

Pertinent findings are generally limited to cardiovascular and respiratory systems. Patients often appear quite distressed. Tachycardia may be the only finding in patients who are otherwise healthy and have significant hemodynamic reserve.

Patients who have limited hemodynamic reserve may be tachypneic and hypotensive.

*To whom correspondence should be addressed.
E-mail: chiangdao_hos@hotmail.com
Tel. +66 8 1980 3690, Fax. +665 3455 2499

Crackles may be auscultated secondary to heart failure. An S_3 heart sound may be present, and large jugular venous pulsations may also be visualized[4].

Other Tests

ECG findings allow classification of the tachyarrhythmia and they may allow a precise diagnosis. P waves may not be visible when present, they may be normal or abnormal depending on the mechanism of atrial depolarization[5].

Medical Cares

PSVT can result in heart failure, pulmonary edema, myocardial ischemia, and/or myocardial infarction secondary to an increased heart rate in patients with poor left ventricular function[6]. Most of the patients who present with PSVT have Atrioventricular Nodal Reentrant Tachycardia (AVNRT) or Atrioventricular Reentrant Tachycardia (AVRT). These arrhythmias depend on AV nodal conduction and therefore can be terminated by transiently blocking AV nodal conduction.

Synchronized cardioversion starting with low-energy levels (50 joules) can be used immediately in patients who are hypotensive, have pulmonary edema, have chest pain with ischemia, or are otherwise unstable[7].

When SVT is not terminated by vagal maneuvers, short-term management involves pharmacological treatment is then needed. There are various choices of antiarhythmic drugs as follow:

1.Adenosine: 6 – 12 mg rapidly bolus *via* venous line and immediately flushed with NSS due to its duration of action was short about 5 seconds. It has 95 % efficacy.

2.Verapamil: 5 – 10 mg IV slowly push in 3 – 5 minutes. The efficacy may be as same as Adenosine, but may cause hypotension, transient heart block, and cardiac conduction compression.

3.Diltiazem: 15 – 10 mg IV slowly push. It has the same efficacy as Verapamil with less side effects.

4.Digoxin: 0.25 – 0.5 mg IV. It should not be used as the first line drug.

5. Others i.e. Aramine, class Ic antiarrhythmic agents[8,9].

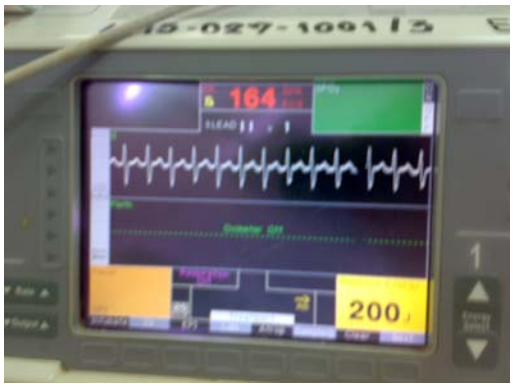
Adenosine is a short-acting drug that blocks AV node conduction. It terminates 90% of tachycardias due to AVNRT or AVRT by blocking electrical impulses in the heart. Typical adverse effects of adenosine include flushing, chest pain, and dizziness. These effects are temporary because adenosine has a very short half-life of 10-20 seconds[10].

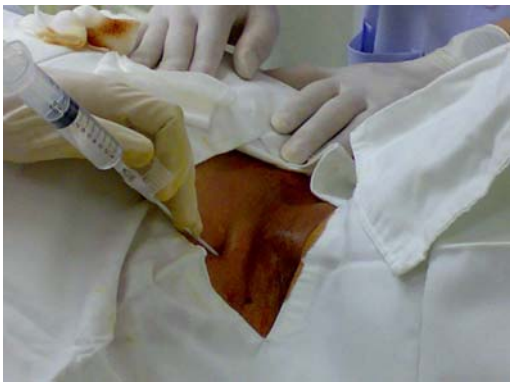
There has never been reported case of SVT treatment by given adenosine bolus *via* subclavian catheterization in the district hospital in Thailand.

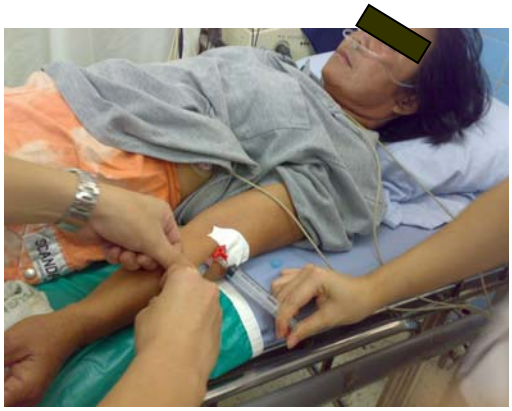
CASES REPORT:

A 51-year-old female with underlying PSVT has received conservative treatment from Maharaj Nakorn Chiangmai Hospital, a government cardiac center and Chiang-Dao Hospital, a district hospital, which distant from the center for 70 km, and take a referral time about one hour. She has a sudden onset of palpitation and breathlessness while worked as handicraft three hours prior to emergency room administration.

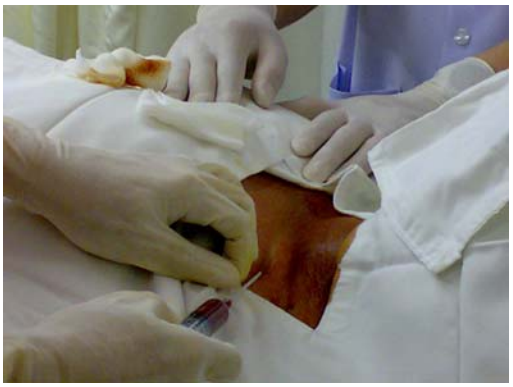
Examination revealed blood pressure of 127/75 mmHg with heart rate 158 bpm, respiration of 20 bpm, and temperature of 37 C. She was slightly hypersthenic shape, afebrile, good consciousness. Heart rate was tachycardia, 158 bpm. 12 – leads ECG and long running lead II ECG was shown narrowed complex SVT with regular tachycardic rhythm 150 – 160 bpm. She was symptomatically treated followed by Advanced Cardiac Life Support (ACLS) clinical practice guideline and specifically treated with 6 mg adenosine bolus *via* peripheral vein for two times, the SVT did not returned to the NSR, to prevent other cardiac complications due to a long distance referral system and delayed treatment, prompted infraclavicular approach subclavian catheterization was done, followed by a bolus of 6 mg adenosine *via* subclavian catheter direct to the heart. Patient's vital signs were normally stable, ECG was returned to NSR within seconds. After that, patient was admitted for two days in the internal department for resting, oxygenation, and ECG monitoring. During admission, patient's vital signs and ECG was normal.

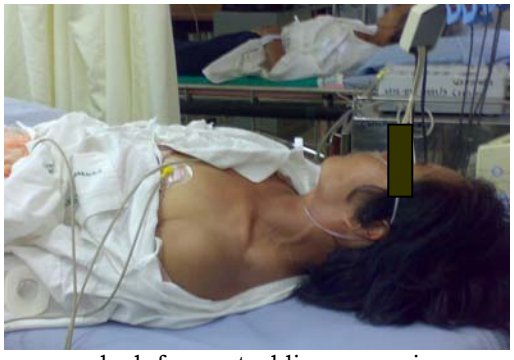




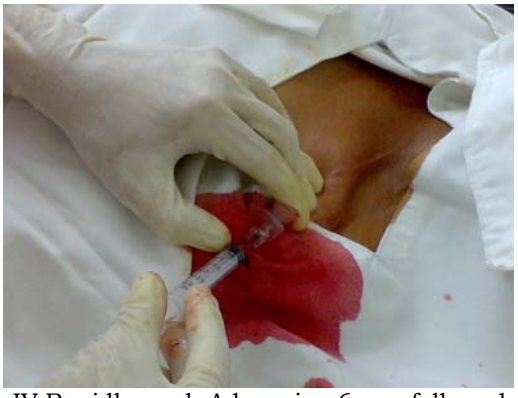


IV Rapidly push Adenosine 6 mg, followed by rapidly bolus NSS. EKG still shown SVT 2nd dose, IV Rapidly push Adenosine 6 mg , followed by rapidly bolus NSS.
EKG not turn to NSR yet



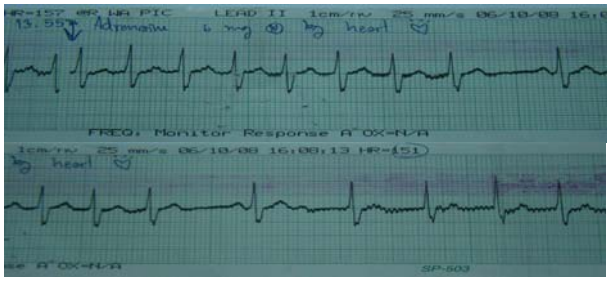


look for central line accessing

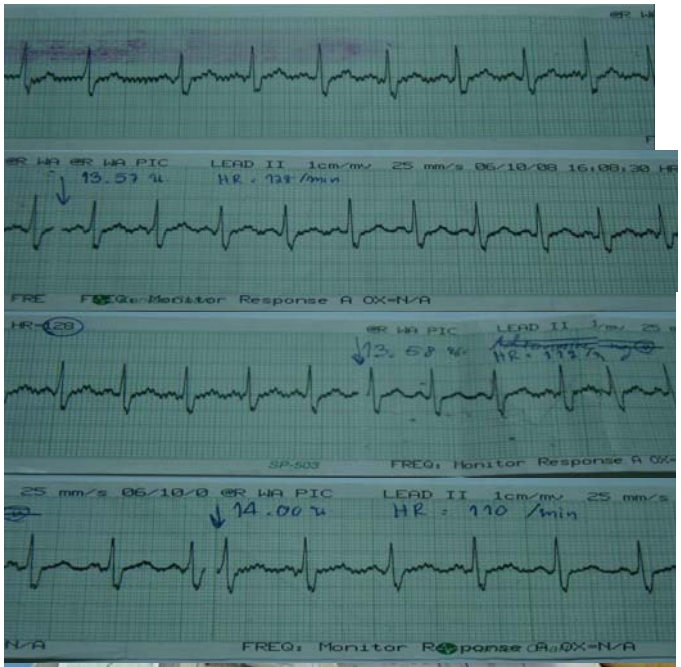


IV Rapidly push Adenosine 6 mg, followed by NSS bolus.

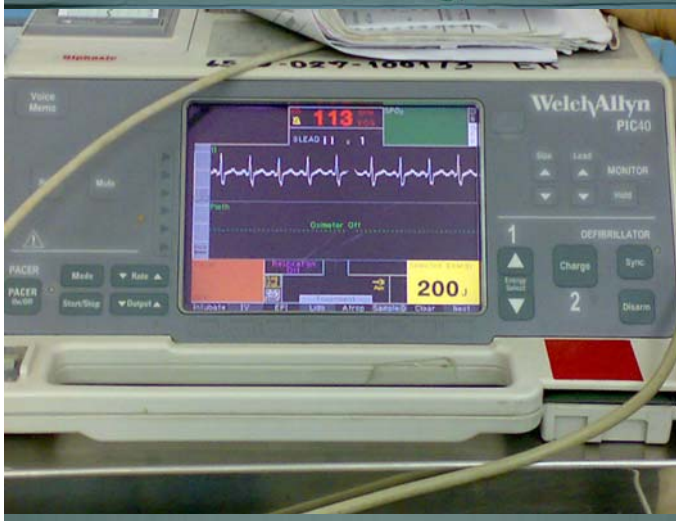
Subclavian catheterization was done with sterile infraclavicular approach, under local anaesthesia



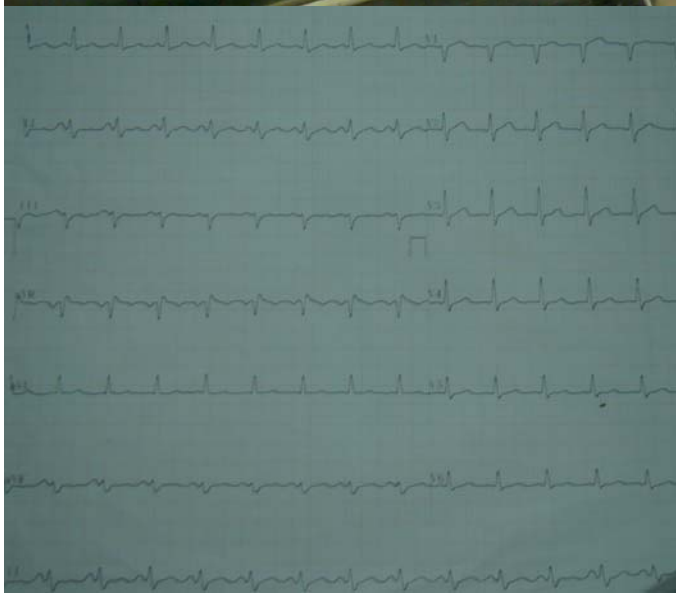
presented "pauses" after bolus



ECG showed the ventricular rate gradually decline the presented " P " wave



The monitoring lead presented sinus tachycardia with regular rhythm 113 bpm



On admission, 12 leads ECG presented normal sinus rhythm

The other case, a 40-year-old male without underlying disease, has abrupt onset of palpitation and fainting after taken a deep breath while playing football 1 hour prior to emergency room administration. He denied a history of palpitation and fainting like this, has smoked 5 cigarettes per day for 15 years and drink alcohol occasionally. Examination revealed blood pressure of 90/60 mmHg with heart rate 150 bpm, respiration of 20 bpm, and temperature of 37 C. He was sthenic, atheltic shape, afebrile, good consciousness. His heart sound was tachycardic and diminished intensity of his radial pulse. 12 – leads ECG shown SVT with regular rhythm 150 – 160 bpm. Symptomatically and specifically treatment was done too, and two bolus of peripheral line adenosine injection was done with failure to normalize the SVT too.

Physician decided to give a 6 mg adenosine *via* infraclavicular approach subclavian catheterization to prevent cardiogenic shock, after a 6 mg adenosine bolus, the heart rate slow down, blood pressure gradually risen to the normal value and the ECG was returned to the NSR. Patient was admitted for one day to the internal department and discharged after vital signs and ECG was normal.

CONCLUSION: The purpose of this report was to present SVT patients in the rural non medical center context which in some situation there may be no sufficient time to refer the patient to the center. Eventually immediate necessary treatment is required to safe life first.

The infraclavicular approach subclavian catheterized – Adenosinization, direct to the heart is another choices in the failure of peripheral adenosinization.

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