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Clinical review

Supraventricular tachcycardia practical guide for diagnosis and management

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Abstract Supraventricular tachycardia (SVT) is any atrial tachyarrhythmia with heart rate exceeding 100 bpm in rest. The simplest and clinically simple to apply is a division according to QRS duration (width), to SVT with narrow QRS (less then 120ms duration) and wide QRS tachycardias (over 120ms QRS duration). Diagnostic algorithm for SVT differentiation is started by a 12 lead ECG which is interpreted by its QRS duration as a fisrt step in SVT diagnostics. The second step is regularity assessment of the RR interval , while the third step is P wave identification. The last step is assessment of the P wave to QRS frequency ratio. ECG interpretation in these four steps enables SVT identification in most of the patients. In some patients a vagal maneuver or i.v application of Verapamil or Adenosin are needed in order to distinguish the SVT type. The therapy of SVT patients is divided into acute, which goal is to cease the tachycardia onset, and chronic, or ongoing whose goal is to maintain sinus rhythm. The acute therapy is administrated based on the QRS duration and hemodynamic state of the patient. Conclusions: everyday, clinically orientated approach to patients with supraventricular tachycardias is based on simple ECG criteria and not on complex mechanisms of tachycardia origin. The approach based on QRS duration, or QRS morphology divided into wide and narrow QRS complexes is simple and efficient approach for practical diagnostics and therapy of supraventricular tachyarrhythmias.

Kew words supraventricular tachycardia, QRS duration and width, tachycardia induced cardiomyopathy.

Introduction

Supraventricular arrhythmias are common and patients are often symptomatic, requiring management with drugs and electrophysiological procedures. In the general population, the Supraventricular Tachycardia (SVT) prevalence is 2.25/1000 persons and the incidence is 35/100 000 person-years. Women have a risk of developing SVT that is two times greater than that of men, and persons aged ≥65 years or have more than five times the risk of developing SVT than younger individuals[1]. In specialized centers, AV nodal reentry tachycardias (AVNRT) is the most frequently treated substrate after Atrial Fibrillation (AF), followed by atrial flutter and AV reentry tachycardias (AVRT), in patients referred for catheter ablation[2-4]. Women are more likely to be affected by AVNRT than men (ratio 70:30),[5-6] while the converse is true for AVRT (ratio 45:55)[7]. A relationship with the monthly cycle has been suggested,[8] and episodes are more frequent during pregnancy in women with pre-existing SVT[9].

How to recognize

Supraventricular tachycardia (SVT) is any atrial tachyarrhythmia with heart rate of more than 100 bpm at rest[1]. Supraventricular tachycardias can be divided according to their electrophysiological origin and QRS width.

Table 1. General electrophysiology division.

Atrial tachycardias
• Sinus tachycardia • Focal Atrial Tachycardia • Multifocal Atrial Tachycardia • Macro Reentry Atrial Tachycardia • Atrial Fibrillation
AV junctional tachycardias
• Atrioventricular nodal re-entrant tachycardia (AVNRT) • Non-re-entrant junctional tachycardia
Atrioventricular re-entrant tachycardia (AVRT)
• Orthodromic • Antidromic

The General electrophysiology division is based on pathophysiology/electrophysiology approach and mechanism of origin. In the remainder of this text we will base our focus on a clinically based approach to division of tachycardias. This approach is based on a simple wide vs narrow QRS morphology, or should we say "worry or not to worry morphology". As a border mark, a 120ms QRS width was taken. All further diagnostic and therapy approach in patients with supraventricular tachycardias featured in this article will be based on this value.

Diagnosis

In everyday clinical practice a 12 lead ECG is the simplest way ahead, but we also have additional simple tools like vagal maneuvers and some intravenous injection of medications. SVT diagnosis can be made based on three basic steps which represent the basis of diagnostic algorithm, using 12 lead ECG , vagal maneuvers and intravenous administration of Adenosine (or in our everyday clinical situation/s intravenous administration of Verapamil)

1. 12 leads ECG

In exam protocol all patients should have a 12 lead ECG done.
We divided a ECG interpretation into four steps in order

Table 2. QRS width division

Narrow tachycardias ≤120 ms QRS width
Regular rhythm narrow QRS tachycardias
• Sinus tachycardia • Focal Atrial Tachycardia • Atrial flutter with fixed AV conduction • Atrioventricular nodal re-entrant tachycardia (AVNRT) • Orthodromic AVRT • Idiopathic VT (especially high septal VT)
Irregular rhythm narrow QRS tachycardias
• Atrial Fibrillation • Focal Atrial Tachycardia or atrial flutter with varying AV block • Multifocal Atrial Tachycardia
Wide tachycardias >120 ms QRS width
Regular rhythm wide QRS tachycardias
• Ventricular Tachycardia or flutter • Ventricular paced rhythm • Antidromic AVRT • Supraventricular tachycardias with Bundle Branch Block (pre-existing or rate-dependent during tachycardia) • Atrial tachycardia with pre-excitation • Wide QRS Supraventricular tachycardias due to electrolyte disturbance or antiarrhythmic drugs
Irregular rhythm wide QRS tachycardias
• Atrial fibrillation or atrial flutter with varying block conducted with aberration • Antidromic AV re-entrant tachycardia with variable Ventricle-Atrial conduction • Pre-excited Atrial Fibrillation • Polymorphic Ventricular Tachycardia • Torsade de pointes • Ventricular fibrillation

to make a diagnosis of supraventricular tachycardia easier.
Step one: First comes first, it is necessary to measure the width of the QRS with the boundary being 120ms.
Step two: according to the RR intervals check the regularity/irregularity of the tachycardia
Step three: P wave presence/absence, the best way to see it is in the V1 lead.
Step four: See the relationship of P frequency and QRS frequency.
After step one, if we have **narrow QRS tachycardia**: we judge the regularity of the same. If the tachycardia is irregular, we probably are encountering atrial flutter, fibrillation or atrial tachycardia. If the tachycardia is regular, we search for P waves and their relationship with QRS complexes and frequency of the QRS.
Back to step one, **wide QRS tachycardia**: If we have wide QRS tachycardia, this brings out the old dilemma weather this tachycardia is supraventricular or ventricular in origin. On the other hand, this new division of narrow and wide QRS tachycardias gives a non-electrophysiologist a unique opportunity to be relatively safe and efficient in therapy regardless of the electrophysiological origin of the tachycardia.

2. Vagal maneuvers

There are several possibilities of reaction to these maneuvers (1):

- **Slowing of AV node conduction**: thus, unmasking atrial electrical activity and revealing P waves.

This happens in atrial fibrillation, flutter or focal Atrial tachycardia

- **Temporary decrease in the atrial rate:** sinus tachycardia and focal atrial tachycardia
- **Tachycardia termination:** AVRT and AVNRT
- **No effect**

3. Intravenous administration of Adenosine or Verapamil in regular narrow QRS tachycardia[1]:

- **Slowing of AV node conduction:** thus, unmasking atrial electrical activity and revealing P waves. This happens in atrial fibrillation, flutter or focal Atrial tachycardia
- **Temporary decrease in the atrial rate:** sinus tachycardia and focal atrial tachycardia
- **Tachycardia termination:** AVRT and AVNRT
- **No effect** if the dose or delivery is inadequate

Management

All therapeutic approaches in SVT are generally divided into acute and ongoing (chronic). Acute management is dependent on the QRS width and hemodynamic compromise at the presentation patients with supraventricular tachycardia. Acute management has as its objective to cease the tachycardia. Chronic or ongoing has as its goal prevention of tachycardia or at least making the probability of it happening much lower.

Acute management of supraventricular tachycardias

Narrow QRS tachycardias without established electrophysiological origin[10-15]

Hemodynamically stable patients

- **A 12 lead ECG** during tachycardia is mandatory
- **Vagal maneuvers**
- **Adenosine** (6-18 mg intravenous bolus) if vagal maneuvers fail
- **Verapamil** (intravenous) if vagal maneuvers and adenosine fail (or adenosine not available)
- **Metoprolol** intravenous if vagal maneuvers and adenosine fail (or verapamil was not applied)
- **Synchronized direct-current cardioversion** if everything above mentioned fails.

Hemodynamically unstable patients

- **Synchronized DC cardioversion.**

Wide QRS tachycardias without established electrophysiological origin[16-18]

Hemodynamically stable patients

- **A 12 lead ECG** during tachycardia is mandatory
- **Vagal maneuvers**
- **Adenosine** (6-18 mg intravenous bolus) if vagal maneuvers fail, and with no preexcitation on resting ECG (if available) in this case verapamil is not recommended!
- **Procainamide (**intravenous) if vagal maneuvers and adenosine fail
- **Amiodarone** intravenous administration, if vagal maneuvers and adenosine fail
- **Synchronized direct-current cardioversion** if everything above mentioned fails.

Hemodynamically unstable patients

- **Synchronized DC cardioversion.**

The majority of failures in intravenous therapy come with erroneous application of the drug.
Adenosine: The mean dose required for termination is 6 mg. To achieve efficient rhythm correction, injection should be as a rapid bolus with immediate saline flush. Large, centrally located (e.g. antecubital) veins are likely to deliver more effective drug concentrations to the heart. Dosing should be incremental, starting at 6 mg in adults followed by 12 mg. An 18 mg dose should
be considered, also taking into account tolerability/side effects in the individual patient. Adenosine has a very short plasma half-life measured in seconds, with end-organ clinical effects complete within 20-30 s.
Repeat administration is safe within 1 min of the last dose.
Calcium channel blockers (verapamil intravenous) and beta blockers (metoprolol intravenous) are of value, particularly in patients with frequent atrial or ventricular premature beats.
Verapamil: 0.075 - 0.15 mg/kg intravenous (average 5 - 10 mg over 2 min), on the other hand is associated with a risk of hypotension. These drugs should be avoided in patients with hemodynamic instability, HF with reduced LV ejection fraction (<40%), a suspicion of VT, or pre-excited AF.

Metoprolol: 2.5-15 mg given intravenous in 2.5 mg boluses. Beta blockers are contraindicated in patients with decompensated heart failure. Caution is needed with concomitant use of intravenous calcium channel blockers and beta-blockers, because of possible potentiation of hypotensive and bradycardic effects.
Valsalva maneuver: Carotid sinus massage is performed with the patient's neck in an extended position, with the head turned away from the side to which pressure is applied. It should always be unilateral as there is a potential risk with bilateral pressure, and it should be limited to 5 s. The patient should be monitored. This technique should be avoided in patients with previous transient ischemic attack or stroke, and in patients with carotid bruits. Other maneuvers, such as facial immersion in cold water or forceful coughing, are rarely used[1].

Chronic management of supraventricular tachycardias
Two points should be made regarding the practical chronic management of supraventricular tachycardias[19-29].
First is the fact that the agent that stopped the acute onset does not necessarily is the agent of choice for the prolonged management.
Second, the ongoing management is quite often therapy orientated (medical or interventional) and then electrophysiological in nature.
In that light our points will be made according to agents and procedures used:
Beta blockers: are used for heart rate slowing and thus for SVT cessation. Today, Bisoprolol, Nebivolol, and Metoprolol are recommended for oral therapy.
Sinus tachycardia
- Atrial tachycardia/s
- AV nodal reentry tachycardias
- Manifest preexcitation

Calcium channel antagonist (Non dihydropyridine type): Verapamil or Diltiazem are recommended for oral therapy.
- Sinus tachycardia
- Atrial tachycardia/s
- AV nodal reentry tachycardias

I_f Channel blockers (Ivabradine): is used as a second line therapy when a full dose of beta blocker does not achieve sinus rhythm slowing. They are introduced along with beta blocker. On the other hand, they can be used as a single therapy agent in patients where beta blocker use is contraindicated. The are used only in patients with sinus rhythm, they are not of any use in patients with atrial fibrillation.
Sinus tachycardia
- Atrial tachycardia/s other than atrial flutter of fibrillation

Ic Vaughan-Williams group of antiarrhythmic drugs (Procainamide, Propafenone, Flecainide): These medication should not be used in patients with manifest coronary artery disease or in patients with heart failure.
- AV nodal reentry tachycardias
- Macro reentrant tachycardias-AVRT
- Manifest preexcitation

III rd group Antiarrhythmic drugs: Amiodarone, Dronedarone, Sotalol
- AV nodal reentry tachycardias
- Macro reentrant tachycardias-AVRT
- Manifest preexcitation

Catheter ablation: It is rarely used as a first choice therapy, almost by rule when other therapy options are exhausted.
- Sinus tachycardia (rarely)
- Atrial tachycardia/s
- AV nodal reentry tachycardias
- Macro reentrant tachycardias-AVRT
- Manifest preexcitation

Tachycardia induced cardiomyopathy like long term consequence of sustained untreated and uncontrolled supraventricular tachycardia/s

Definition

It is a reversible cause of impaired LV function due to persistent tachycardia or very frequent ventricular premature beats that can lead to HF and death[30-32].
However, it has not been fully established how the majority of patients with frequent premature ventricular contractions have a benign course, whereas ≤30% of them may develop cardiomyopathy.

Diagnosis

This sort of cardiomyopathy should be considered[33]:
1: In any patient with new onset of LV dysfunction. Typically, in LV ejection fraction is <30%, LV end-diastolic diameter is <65 mm, and LV end-systolic diameter is <50 mm.
2. In the presence of persistent or frequent tachycardia, or frequent premature ventricular contractions.
3. The diagnosis is made by excluding other more plausible causes of cardiomyopathy (NMR, MDCT, invasive angiography, biopsy etc.)
4. Recovery of LV function after cessation of the arrhythmia or control of the ventricular rate.
5. Serial assessment of N-terminal pro-B-type natriuretic peptide (NT pro BNP) and comparing it to the baseline level during follow-up can help differentiate it from irreversible idiopathic dilated cardiomyopathy.
6. ECG long term monitoring (24-72h Holter ECG, Telemonitoring, Cardiac Implanted Electronics Devices (CIED) in reaching diagnostic thresholds for arrhythmic events frequency).

Therapy

Medicament or interventional (ablation) intervention in stopping or reducing the frequency of the arrythmia events[34-37].

Conclusion

Clinical, everyday approach to supraventricular tachycardias and arrythmias should be based how much is possible on clinical and ECG criteria rather than complex arrythmia origin assessment. The approach based on narrow vs wide QRS morphology and duration is reliable

and efficient for practical diagnosis and treatment of supraventricular tachycardias.

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Sažetak

Supraventrikularna tahikardija (SVT) je bilo koja atrijalna tahiaritmija sa srčanom frevencom preko 100/min, u mirovanju. Najjednostavnija i klinički lako primenljiva je podela SVT prema širini QRS kompleksa, na SVT sa uskim QRS kompleskima (manje od 120ms) i na SVT sa širokim QRS kompleksima (preko 120ms). Dijagnostički algoritam za prepoznavanje SVT podrazumeva 12 ovdvodni EKG kod koga se interpretira širina QRS kompleksa kao prvi korak u dijagnostikovanju SVT. Drugi korak je određivanje regularnosti ili iregularnosti RR intervala, treći korak je identifikacija P talasa. Poslednji korak je odreživanje međusobnog odnosa frekvence P talasa I frekvence QRS. Interpretacijom EKG u ova četiri koraka kod najvećeg broja pacijenata omogućiće nam identifikaciju SVT. Kod nekih pacijenata biće potreno sprovesti Vagalni manevar ili intravenski primeniti Adenosin ili Verapamil. Terapija pacijenata sa SVT deli se na akutnu, sa ciljem da se prekine SVT i hronična, terapija održavanja sinusnog ritma. Akutna terapija primenjuje se u zavisnosti od širine QRS kompleksa i hemodinamskog statusa pacijenta. Zaključci: Svakodnevni, klinički pristup pacijentima sa Supraventrikularnim tahikardijama baziran je na jednostavnim EKG kriterijumima a ne na kompleksnim mehanizmima nastanka aritmija. Pristup baziran prema širini QRS kompleksa, morfologija širokih i uskih QRS kompleksa je jednostavan i efikasan pristup za prkatičnu dijagnostiku i terapiju supraventrikularnih tahiaritmija.

Ključne reči: *Supraventrukularna tahikardija, dužina trajanja QRS kompleksa, tahikardijom indukovana kardiomiopatija*