Atrial tachycardia
Lizanne Laird, CNP
Heart and Vascular Institute

1. Differentiate atrial tachycardia from sinus tach and atrial ectopic rhythms
2. Discuss pathology
3. Discuss treatment options

75 y/o female
Hx of MI, PAD, HTN, dyslipidemia, DM, bifascicular block (RBBB/LAFB), sinus bradycardia

• Hospitalized for peripheral procedure
• Post procedure sinus rhythm in 85-95 bpm alternated with sinus brady 35-50 bpm
• Multiple pauses 2.0 up to 5.5 seconds
• Asymptomatic- blood pressure stable
• Labs: Na 133, K 4.6, Bun/Creat NL, CBC NL, TSH 2.04
• Echo: EF 49%, Normal RA and LA size, no valvular heart disease, apical hypokinesis.
Atrial Tachycardia (AT)

- SVT arising from localized atrial tissue different from SA node.
- Regular, organized atrial activity with discrete P waves.
- Change in P wave morphology, different from normal P wave.
- May be associated with prolonged PR interval
- Typically an isoelectric segment between P wave.
- May be paroxysmal or incessant.
Junction of an anastomosis with a venous structure or valve or septum

Atrial Tachycardia (AT)

• Usually Fast rhythm - 100 to 250 bpm
• Faster rate in younger patients
• Symptoms depend on frequency of episode, duration and whether occur with exercise or rest.
• Palpitations, chest pain, lightheadedness, DOE
• Rarely syncope
• One large review - AT increases with age up to 23% of SVT
Atrial Tachycardia

- At times irregularity is seen especially at onset (warm up) and termination (warm down)

Arrows point to the P wave, which is inscribed before the QRS complex. The focus of this atrial tachycardia was mapped during electrophysiological study to an area near the left inferior pulmonary vein.
AT-mediated cardiomyopathy

- Reported in 10% of patients with AT
- And as high as 37% in pt with incessant AT
- Presenting with HF

<table>
<thead>
<tr>
<th>Triggers</th>
<th>Frequent PVCs</th>
<th>Atrial Fibrillation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased HR</td>
<td>RV dysynchrony</td>
<td>AV dissociation</td>
</tr>
<tr>
<td></td>
<td>LV irregularity</td>
<td>HR irregularity</td>
</tr>
<tr>
<td></td>
<td>Pre-excitation</td>
<td>Atrial fibrillation</td>
</tr>
<tr>
<td></td>
<td>Pre-excitation</td>
<td>Atrial fibrillation</td>
</tr>
</tbody>
</table>

Effect

- Effects on myocardial function
- Effects on electrical and mechanical function
- Effects on arrhythmia activation

Arrhythmia Suppression

- Ablation
- Antiarrhythmic drugs

Recovery

- Normalized LVEF
- Ventricular relaxation
- Statistic dysynchrony
- Reactive hypotrophy
- Persistent Mancia

![Image of arrhythmia and cardiac function](image)

**FIGURE 11** Ongoing Management of Focal Atrial Tachycardia

- Focal atrial tachycardia
- Ablation candidate, pt prefers ablation
- Catheter ablation (Class I)
- Beta blockers, diltiazem, or verapamil (Class IIa)
- Flecaïnide or propafenone (in the absence of SHD) (Class IIa)
- Amiodarone or sotalol (Class IIb)

Drug therapy options

- If ineffective
Atrial Tachycardia

• Tachycardia should be first classified if regular or irregular
• Irregular: AF, MAT, atrial flutter with variable AV conduction
• Regular: SVT, AT, Junc tach, re-entrant tachycardia's (AVNRT, AVRT)
• Has P wave morphology or PR interval lengthened
• Upright and distinct P waves, short PR interval, narrow QRS- ST likely

Atrial Tach or Sinus Tachycardia?
Treatment

• Coreg stopped
• 30 day MCT (mobile cardiac telemetry)
• MCT showed multiple pauses, up to 8 seconds, occurred during sleep and awake
• Ectopic atrial rhythm = 80-95 bpm
• Pt denied any symptoms during the time wearing monitor.
• Dual chamber pacemaker was implanted.

What is the take home message?

• Common problem of differentiating ST from various atrial tachycardia’s and re entry tachyarrhythmia’s.

• When apparent ST is associated with prolonged PR interval one should suspect that it is NOT really ST.

• If change in P wave morphology and heart rate greater than 100 bpm for no apparent reason- suspect atrial tachycardia
Look for reasons for ST and if none may be an AT

References

Huzar et al., Arrhythmia-induced cardiomyopathy; JACC Vol 73, NO.18, 2019
2015 Acc/AHA/HRS SVT Guidelines
Curtis, et al., Arrhythmia in patients > 80 years or age; JACC /vol 71, No.18, 2018
80 year old female with symptomatic SVT

- Symptomatic - palpitations, shortness of breath.
- Unresponsive to Vagel maneuvers
- On diltiazem CD 240 mg with minimal improvement.
- Underwent AVNRT ablation.
Tachycardia-induced cardiomyopathy (T-CM):

1. T-CM refers to the presence of a reversible left ventricular (LV) dysfunction solely due to increase in ventricular rates, regardless of tachycardia origin.

2. Animal models of T-CM show that rapid atrial or ventricular pacing causes structural and electrical remodeling. Cessation of tachy-pacing results in significant recovery of LV ejection fraction (LVEF) or its normalization. Importantly, however, fibrosis appears to persist despite elimination of the tachycardia and normalization of LV function.

3. An ambulatory electrocardiogram (ECG) monitor for ≥2 weeks should be considered to confirm or exclude T-CM. The final diagnosis of T-CM can only be confirmed after recovery or improvement of LV systolic function within 1 to 6 months after elimination of the tachyarrhythmia.

4. In addition to treating tachycardia with antiarrhythmic drugs or radiofrequency ablation, the initial treatment of T-CM should include initiation and optimization of medical therapy for heart failure and LV systolic dysfunction (beta-blockers, angiotensin-converting enzyme inhibitors or angiotensin-receptor blockers, diuretic agents, and aldosterone blockers) to optimize reverse remodeling.