Current Trends in Atrial Fibrillation

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BHII Primary Care Symposium
February 27, 2015
Financial Disclosures

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Atrial Fibrillation

- Mechanism
- Risk Factors
- Anticoagulation
- Rate Control
- Rhythm Control
- Future directions
Structurally normal heart or mild HTN disease, AF is triggered by PACs from the PV ostia.

The heritability of AF is complex, most likely related to polygenic phenomenon.

Chronic, heavy alcohol use does increases risk of AF in men, ? Women.

Incidence and prevalence of AF increases with age and weight.

Hypertensive heart disease and coronary heart disease are the most common disease process associated with AF. ETOH, CHF, Valve heart disease and hyperthyroid.

Rheumatic heart disease are associated with AF.

High incidence of CABG and cardiac valve surgery.
# Anticoagulation

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Score</th>
<th>CHA2DS2-VASc score</th>
<th>Stroke Risk per Year</th>
</tr>
</thead>
<tbody>
<tr>
<td>Congestive Heart Failure/LV dysfunction</td>
<td>1</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1</td>
<td>1</td>
<td>1.3%</td>
</tr>
<tr>
<td>Age ≥ 75 years</td>
<td>2</td>
<td>2</td>
<td>2.2%</td>
</tr>
<tr>
<td>Diabetes Mellitus</td>
<td>1</td>
<td>4</td>
<td>4.0%</td>
</tr>
<tr>
<td>Stroke/TIA/Thromboembolism</td>
<td>2</td>
<td>5</td>
<td>6.7%</td>
</tr>
<tr>
<td>Vascular Disease</td>
<td>1</td>
<td>6</td>
<td>9.8%</td>
</tr>
<tr>
<td>Age 65 – 74</td>
<td>1</td>
<td>7</td>
<td>9.6%</td>
</tr>
<tr>
<td>Female</td>
<td>1</td>
<td>8</td>
<td>6.7%</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>9</strong></td>
<td><strong>9</strong></td>
<td><strong>15.2%</strong></td>
</tr>
</tbody>
</table>
# Rate Control

<table>
<thead>
<tr>
<th>Recommendations</th>
<th>COR</th>
<th>LOE</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control ventricular rate using a beta blocker or nondihydropyridine calcium channel antagonist for paroxysmal, persistent, or permanent AF</td>
<td>I</td>
<td>B</td>
<td>93–95</td>
</tr>
<tr>
<td>IV beta blocker or nondihydropyridine calcium channel blocker is recommended to slow ventricular heart rate in the acute setting in patients without pre-excitation. In hemodynamically unstable patients, electrical cardioversion is indicated</td>
<td>I</td>
<td>B</td>
<td>96–99</td>
</tr>
<tr>
<td>For AF, assess heart rate control during exertion, adjusting pharmacological treatment as necessary</td>
<td>I</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>A heart rate control (resting heart rate &lt;80 bpm) strategy is reasonable for symptomatic management of AF</td>
<td>IIa</td>
<td>B</td>
<td>95,100</td>
</tr>
<tr>
<td>IV amiodarone can be useful for rate control in critically ill patients without pre-excitation</td>
<td>IIa</td>
<td>B</td>
<td>101–103</td>
</tr>
<tr>
<td>AV nodal ablation with permanent ventricular pacing is reasonable when pharmacological therapy is inadequate and rhythm control is not achievable</td>
<td>IIa</td>
<td>B</td>
<td>104–106</td>
</tr>
<tr>
<td>A lenient rate-control strategy (resting heart rate &lt;110 bpm) may be reasonable when patients remain asymptomatic and LV systolic function is preserved</td>
<td>IIb</td>
<td>B</td>
<td>100</td>
</tr>
<tr>
<td>Oral amiodarone may be useful for ventricular rate control when other measures are unsuccessful or contraindicated</td>
<td>IIb</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>AV nodal ablation should not be performed without prior attempts to achieve rate control with medications</td>
<td>III: Harm</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>Nondihydropyridine calcium channel antagonists should not be used in decompensated HF</td>
<td>III: Harm</td>
<td>C</td>
<td>N/A</td>
</tr>
<tr>
<td>With pre-excitation and AF, digoxin, nondihydropyridine calcium channel antagonists, or amiodarone should not be administered</td>
<td>III: Harm</td>
<td>B</td>
<td>107</td>
</tr>
<tr>
<td>Dronedarone should not be used to control ventricular rate with permanent AF</td>
<td>III: Harm</td>
<td>B</td>
<td>108,109</td>
</tr>
</tbody>
</table>

AF indicates atrial fibrillation; AV, atrioventricular; bpm, beats per minute; COR, Class of Recommendation; HF, heart failure; IV, intravenous; LOE, Level of Evidence; LV, left ventricular; and N/A, not applicable.
Location of AF triggers

Diagram showing various anatomical structures:
- RAA
- LAA
- SVC
- CT
- RAFW
- IAS
- CSO
- IVC
- PV
- 48
- 55
PAC induced AF
Ablation
LARIAT System

Percutaneous, epicardial placement of pre-tied suture loop
LAA ligation with the LARIAT Suture Delivery Device
Event Rate (% patient-years) vs. CHADS2 score

- National Registry of AF Stroke rate
- National Registry of AF Stroke rate with ASA

6.2% Expected ER

Observed ER: 1%

Reduction of 80%

CHADS2 score of the LARIAT Population
Patient Selection Criteria

1. Diagnosed non-valvular atrial fibrillation
2. Current CHADS$_2$ score $\geq 2$ or CHA$_2$DS$_2$VASc $>3$
3. Currently contraindicated to or intolerant of standard long-term oral anticoagulation (OAC) therapy (i.e., warfarin, dabigatran, apixaban, and rivaroxaban) due to a history of internal or external bleeding.
   Examples:
   a) Neurological: intracranial, subdural, subarachnoid, parenchymal
   b) Intraocular bleeding
   c) GI: diverticulosis, ulcerative colitis, Crohn’s disease, recurrent gastric or duodenal ulcer, erosive gastritis, esophageal tears, AV malformations, frequent nose bleeds
   d) Urological: hemorrhagic cystitis, urolithiasis with recurrent bleeding
   e) Pulmonary: hemoptysis, arterial-venous malformations

Contraindication to long-term OAC due to high risk for bleeding
Examples:
   a) Recurrent syncope and traumatic falls
   b) High-risk occupations (i.e. professional athletes, soldiers, fire fighters)
   c) Intracranial aneurysms
   d) Dual antiplatelet therapy with high risk of bleeding

5. Failure of OAC (e.g. embolic event despite OAC)
6. Intolerance or difficulty in using OAC
Effect of Endoepicardial Percutaneous Left Atrial Appendage Ligation on Arrhythmia Burden in Patients with Atrial Fibrillation

Change in AF burden (3 months post LARIAT)

- Pre Lariat AF burden: 78%
- Post Lariat AF burden: 44%

P < 0.001
LAA exclusion as adjunctive therapy to PVI for the treatment of AF

What is the rationale?

- Elimination of atrial arrhythmias arising from the LAA
- Left atrial debulking results in decreased left atrial dispersion
## Outcomes of LAALA-AF

<table>
<thead>
<tr>
<th>BASELINE CHARACTERISTICS</th>
<th>LARIAT + AF ABLATION (n=69)</th>
<th>AF ABLATION ONLY (n=69)</th>
<th>P-VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>AF recurrence at 12 months after 1(^{st}) ablation</td>
<td>35%</td>
<td>61%</td>
<td>0.002</td>
</tr>
<tr>
<td>Repeat Ablation needed</td>
<td>16%</td>
<td>31%</td>
<td>0.04</td>
</tr>
<tr>
<td>Stroke or TIA during f/u</td>
<td>0 (0%)</td>
<td>2 (3%)</td>
<td>0.78</td>
</tr>
</tbody>
</table>

Sub group of patients who had a leak (median 3 mm) recurrence rates are significantly higher 3/6 (50%) compared to those who did not have the LAA leak.

Complete mechanical isolation is probably important for electrical isolation.
Conclusion

- Atrial fibrillation has many risk factors but weight and age are two large contributors.
- Anticoagulation should be considered for anyone at high risk of stroke.
- Rate Control with beta blockers and Calcium channel blockers.
- Rhythm Control considered for symptomatic atrial fibrillation.
- Ablation consideration.
- Left atrial appendage removal/closure.
QUESTIONS