Atrial Fibrillation – The Basics

Maggie Curry, CRNP

Cardiology Consult Service

National Institutes of Health

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Background

- Most common arrhythmia
- EKG findings - No distinct P waves, R-R intervals follow no repetitive pattern;
- HTN and CAD most common underlying disorders
- Other common predisposing factors include: valvular heart disease, heart failure, hyperthyroid, ETOH, OSA

Types – 3 "P"s

- Paroxysmal – AF that terminates spontaneously or with intervention within 7 days of onset. Episodes may recur with variable frequency

- Persistent – AF that fails to self-terminate within 7 days. Often require pharmacologic or electrical cardioversion to restore sinus rhythm.

- Permanent - term used to identify individuals with persistent atrial fibrillation where a joint decision by the patient and clinician has been made to no longer pursue a rhythm control strategy

Evaluation

- EKG
- Labs – CBC, electrolytes, thyroid studies, cardiac enzymes
- Transthoracic Echo – eval for structural causes of AF

Rate vs. Rhythm Control

- Determine onset
- Unless unstable, try to rate control first – often slowing rate will lead to resolution of symptoms
- If unstable or know specific time of onset can attempt rhythm control first – either pharmacologically or electrically

Rate Control

- Drugs of choice – beta blockers or calcium channel blockers (non-dihydropyridines – diltiazem, verapamil)
- choice among a beta blocker and CCB is frequently based upon physician and patient preference
- Some considerations - beta blockers are particularly useful when the ventricular response increases to inappropriately high rates during exercise, after an acute myocardial infarction, and when exercise-induced angina pectoris is also present. Intravenous beta blockade is more effective than intravenous calcium channel blockade for rate control, but also for conversion to sinus rhythm, especially after cardiac surgery. On the other hand, a calcium channel blocker is preferred in patients with chronic obstructive pulmonary disease and asthma.
- Digoxin considered ONLY in patients with HF or if fail other treatment first

Rhythm Control

- most patients with symptomatic new onset AF and most patients with apparently asymptomatic AF should have at least one attempt at cardioversion (either electrical or chemical) to sinus rhythm

- the duration of continuous AF is a strong predictor of the ability to restore and maintain sinus rhythm

Rhythm Control Cont'd

- low risk of systemic embolization if the duration of the arrhythmia is 24 to 48 hours or less and there are no cardiac abnormalities. No AC needed prior to CV

- If new onset AF of longer than 48 hours duration - cardioversion should be postponed until three weeks of anticoagulation has been achieved or a transesophageal has been performed and shows no left atrial appendage clots

Electrical vs. Pharmacologic CV

- Electrical - For first episodes, electrical cardioversion is preferred but depends on patient stability and comfort level of provider

- Pharm - paroxysmal episodes of AF, drug therapy is preferred if they will have sinus rhythm maintained with long-term antiarrhythmic drug therapy, and as patients with paroxysmal AF will likely convert anyway with or without electrical cardioversion

Pharmocological CV

- Ibutilide (Corvert) – 1 mg IV bolus x 1, can repeat x 1 if no conversion with 1st dose, works in both paroxysmal and persistent AF
  - Pros: one time use, can be used with structural heart disease, 28-51% success rate, fast conversion time (~ 30 min after start of infusion)
  - Cons: can prolong QT interval so higher risk of Torsades de Pointes (pre-load with Mg, monitor for 4 hours after infusion)

Pharm CV Cont'd

- Propafenone – 2 mg/kg IV over 10-20 min, or 450-600 mg PO, better for paroxysmal vs. persistent AF
  - Pros: high success rate (23-54% for IV, 53-86% for PO)
  - Cons: not good for structural heart disease, HF or CAD; slower conversion time (2-8 hours)

Pharm CV Cont'd

- Amiodarone - 150 mg IV over 10 min loading dose, then 0.5 mg/min x 18 hrs
  - Pros: may be helpful in converting/maintaing NSR if given before CV, significant rate slowing effect, preferred in pts with structural heart dz
  - Cons: long conversion time (8+ hrs to days), commit pt to long-term therapy, not FDA approved for a-fib, risk of liver and/or thyroid toxicity

Anticoagulation

- Should your patient be anticoagulated?
- CHA2DS2-VASc
    - CHF – 1 pt
    - HTN – 1 pt
    - Age – 2pt if > 75, 1 pt if 65-74, 0 pt if < 65
    - DM – 1 pt
    - Stroke/TIA/Thromboembolus hx – 2 pt
    - Female 1 pt (0 pt for male)
    - Vascular Dz – 1 pt
- Annual risk of ischemic stroke in untreated patients was 0.2, 0.6, and 2.2 for those with CHA_2DS_2-VASc scores of 0, 1, and 2
- Anticoagulation reduces stroke risk by 2/3 regardless of baseline risk

Anticoagulation

- CHA2DS-VASc = 0 → ASA 81 mg only or no AC
- CHA2DS-VASc = 1 → ASA 81 mg only or warfarin/NOAC
- CHA2DS-VASc = 2 → warfarin/NOAC

Selecting Anticoagulation

Table 5. Definition of the SAMe-TT$_2$R$_2$ Score, Used to Aid Initial Decision Making Between Vitamin K Antagonist (With Good Quality Anticoagulation Control) and a Non–Vitamin K Antagonist Oral Anticoagulant[a]
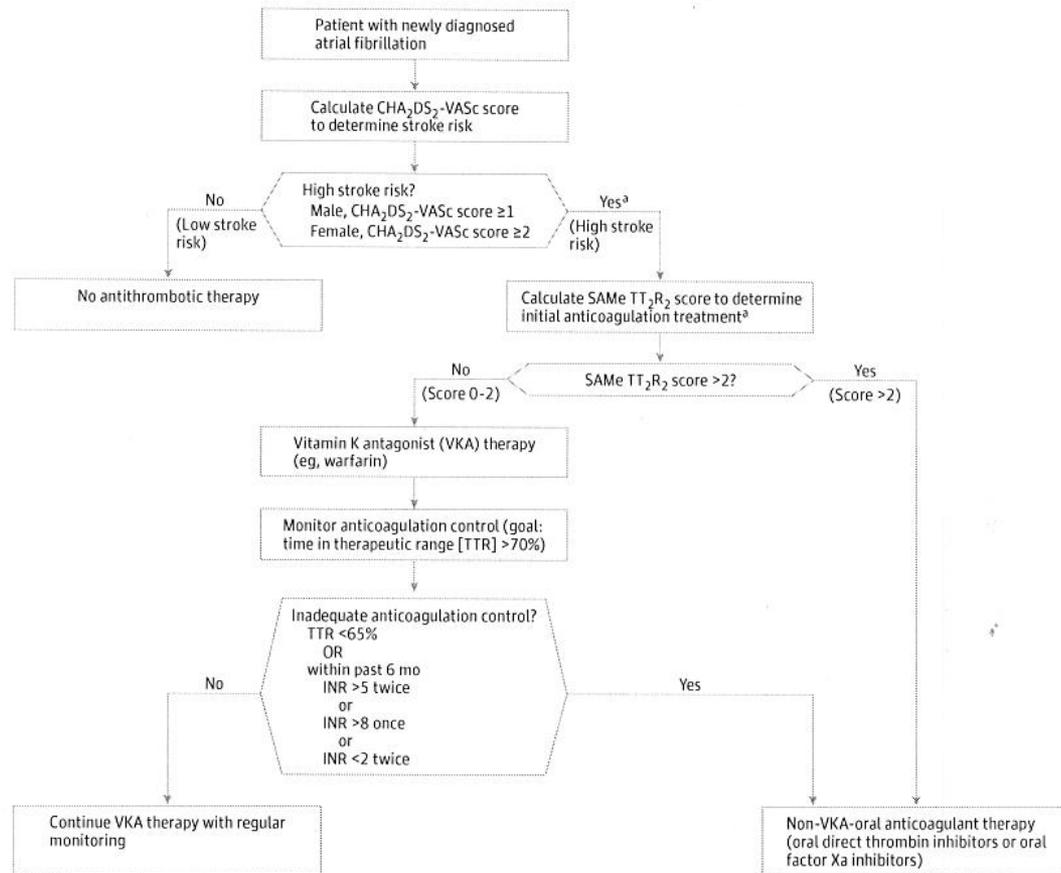
Definitions	Points
Sex (female)	1
Age (<60 y)	1
Medical history[b]	1
Treatment (interacting drugs, eg, amiodarone for rhythm control)	1
Tobacco use (within 2 y)	2
Race (not white)	2
Maximum points	8

[a] The SAMe-TT$_2$R$_2$ score is proposed as a means to help with decision making, to identify those newly diagnosed nonanticoagulated AF patients who have a probability of doing well while taking a vitamin K antagonist (VKA) (with SAMe-TT$_2$R$_2$ score, 0-2) and achieve a time in therapeutic range (TTR) of at least 65% or 70%. In contrast, a SAMe-TT$_2$R$_2$ score of more than 2 suggests that such patients are unlikely to achieve a good TTR while taking a VKA, and a non-VKA oral anticoagulant should be used upfront, without a "trial of warfarin" period.

[b] Two of the following: hypertension, diabetes mellitus, coronary artery disease or myocardial infarctions, peripheral artery disease, congestive heart failure, previous stroke, pulmonary disease, or hepatic or renal disease.

Selecting Anticoagulation

Figure. Algorithm for Risk Stratification and Selection of Anticoagulation Therapy for Stroke Prevention in Atrial Fibrillation

Patient with newly diagnosed atrial fibrillation

Calculate CHA_2DS_2-VASc score to determine stroke risk

High stroke risk?
Male, CHA_2DS_2-VASc score ≥1
Female, CHA_2DS_2-VASc score ≥2

No
(Low stroke risk)

No antithrombotic therapy

Yes[a]
(High stroke risk)

Calculate SAMe TT_2R_2 score to determine initial anticoagulation treatment[a]

No
(Score 0-2)

SAMe TT_2R_2 score >2?

Yes
(Score >2)

Vitamin K antagonist (VKA) therapy (eg, warfarin)

Monitor anticoagulation control (goal: time in therapeutic range [TTR] >70%)

Inadequate anticoagulation control?
TTR <65%
OR
within past 6 mo
INR >5 twice
or
INR >8 once
or
INR <2 twice

No

Yes

Continue VKA therapy with regular monitoring

Non-VKA-oral anticoagulant therapy (oral direct thrombin inhibitors or oral factor Xa inhibitors)

Anticoagulation

- Warfarin
  - Pros: it works!, easy to reverse, can be adjusted
  - Cons: needs to be closely monitored, can be hard to achieve therapeutic range, interacts with so many drugs/food
- NOACs – 2 types: direct thrombin inhibitors (dabigatran) and direct Factor Xa inhibitors (rivaroxaban, apixaban)
  - Pros: no monitoring needed, not inferior to warfarin, fixed dose, no bridging needed, lower incidence of ICH and major bleeding than with warfarin
  - Cons: no reversal agent, no long-term data, renal dosing, BID dosing for some (dabigatran, apixaban)

To bridge or not to bridge

- Don't need to bridge with NOACs
- Have to weigh risk of embolic event vs. risk of bleeding when starting oral anticoagulation
- For non-valvular A.fib – risk of embolic event in few days needed to become therapeutic on warfarin is very low – don't bridge
- For A.fib r/t valvular disease or in pts with previous CVA, known clotting d/o, etc. – risk of embolic event in few days needed to become therapeutic on warfarin is higher – bridge

Duration of Anticoagulation

- If in A.fib for > 48 hrs → 3 wks AC BEFORE cardioversion and 4 wks AC AFTER cardioversion
- If in A.fib for < 48 hrs → not necessary to AC before/after CV, although some will start heparin before CV only
- Pt will need to be reevaluated with Holter/EKG to determine if A.fib has resumed after CV to help further determine duration of AC

Questions?