EMERGENCY MEDICINE CASES
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Episode 20 – Atrial fibrillation

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- Most common dysrhythmia seen in ED, and incidence increasing with ageing population

Presentation

Common presentations: younger patients often feel palpitations and non-anginal chest discomfort, vs. older patients often present with vague symptoms such as fatigue, lightheadedness and dyspnea

Uncommon presentations: TIA, CHF or ACS

A.fib rarely causes syncope in itself – look for another cause: cardiomyopathy, Brugada syndrome, PE, WPW or vasovagal syncope

A.fib is rarely the sole cause of a patient's hemodynamic instability, so look for another cause – sepsis, hemorrhagic (GI, AAA), dehydration, etc

Etiology or precipitants of A.fib

PIRATES – ***P***E, ***I***schemia, ***R***espiratory disease, ***A***trial enlargement or myxoma, ***T***hyroid disease (check TSH and free T4 in first-time presenters), ***E***thanol ("Holiday heart" after binging), ***S***epsis or ***S***leep apnea

Others include myocarditis and chronic hypertension

Etiology of ***Slow*** A.fib: AV nodal blocking agents (digoxin, beta-blocker or calcium-channel blocker toxicity or at too high doses), sick sinus syndrome and severe hyperkalemia

1/3 are 'lone Afib' with no demonstrable cause

In most cases, when the underlying cause is addressed the A.fib resolves

ECG findings

Irregularly irregular narrow-complex tachycardia can be **A.fib**, **A.flutter with irregular conduction**, or **multifocal atrial tachycardia** (MAT) – rate of 100-150/min with 3 different P wave morphologies, variable PR length and poor response to typical medications; find and treat the underlying cause (often COPD)

ST depressions: common and often due to rate-related ischemia in atherosclerotic coronaries of older patients; should not be investigated further UNLESS patient has anginal chest pain or ACS is likely, or ST segments do not resolve once the patient is not in fast A.fib anymore (after rate control or cardioversion)

Rate control vs. rhythm control

AFFIRM trial showed no benefit in rhythm control vs. rate control in terms of morbidity and quality of life, but patients deemed not likely to tolerate being in A.fib were excluded from the study (thereby negating any possible benefit shown in these patients)

Consider rhythm control only if patient has had arrhythmia for <48hrs or has proper anticoagulation for 3wks (documented INR or compliant with dabigatran), especially in younger patients, those who are symptomatic from the arrhythmia (palpitations, or exercise intolerance), and the likelihood of recurrence is low (i.e. if a patient is back monthly with A.fib, a rate control strategy might be more appropriate)

The goal for rate control is HR<100 (according to Canadian guidelines) or HR<110 (according to US guidelines), except if patient is symptomatic at these values, in which case a goal of HR<80 is reasonable

Medications for rate control

Beta-blocker (works in 70% of patients): metoprolol 5mg IV q20min up to 15mg, followed by 25-50mg PO – consider using if patient has HTN, CAD, diabetes, prior MI or hyperthyroidism, but do NOT use in asthmatics or patients in acute heart failure

Calcium-channel blocker (works in 54% of patients): diltiazem 10-20mg IV, followed by PO

Digoxin: should NOT be used as ineffective for rate control, takes a long time to act, and is poor at controlling rate in exercise or anxiety (works through vagal stimulation), therefore does not prevent tachycardia-mediated cardiomyopathy

Amiodarone: consider using in A.fib patient with acute heart failure, esp. If BP is tenuous

In all cases, maximize first agent and avoid going to second agent – re-visit the diagnosis or consider consulting a consultant for admission in these cases

Cardioversion

40-70% of patients will spontaneously convert back to NSR at 24hrs, with shorter episodes more likely to predict conversion (66% if A.fib <24hrs duration, but only 17% once A.fib has lasted >24hrs); also less likely to convert if due to underlying cause or enlarged atrium (more likely permanent A.fib)

After discussion with patient, may elect to return next day for assessment; but most patients who present to ED want something done to improve their condition

Patients may be educated to wait 6-8hrs at home the next time they have an episode, EXCEPT if worrisome symptoms are present (chest pain, dyspnea, lighthheadedness)

Chemical cardioversion

Successful in 60% of cases, but carries higher risk of arrhythmias and hypotension, and longer length of stay in ED

CLASS I medications: flecainide (2mg/kg IV over 10min – most effective), ibutelide (1mg over 10min, repeat at 20min PRN; monitor QT prolongation and consider pre-treatment with MgSO4), and propafenone ("pill in pocket" used by patients when they feel onset of palpitations – must be used in conjunction with beta-blocker to prevent fast dysrhythmias)

Next line agents: procainamide (used in Aggressive Ottawa Protocol by Stiell et al.: 18-20mg/kg at 20-30mg/min and stop if convert to NSR, QT increase to 2 times duration, or hypotension), and amiodarone in cases of structural heart disease (monitor for bradycardia and hypotension; not very effective but consider using in pulmonary edema)

Electrical cardioversion

More effective than chemical cardioversion (90%), but patient may prefer not to use given fear or pain

Do NOT attempt in patients at high risk of thrombo-embolic events (valvular heart disease, severe mitral disease, cariomyopathy, prosthetic valve or prior TIA/CVA), or when procedural sedation is contra-indicated

Consider using initial **energy level of 150-200J** biphasic to increase the success rate and decrease the number of total shocks given

Anticoagulation

Risk of stroke is EQUAL for paroxysmal and persistent A.fib, so even if patient is in NSR at discharge from ED, he or she STILL needs anticoagulation based on risk stratification at least until follow up

Risk of CVA for non-valvular A.fib is 5% per year (1-2% if <60yo, but 25% if >80yo), and risk reduced to 1% with anticoagulation (risk reduction 70% with NNT 25); risk of CVA with cardioversion is dependent on risk factors and length of A.fib but ranges in 1-5%

Use **heparin** (UFH or LMWH) if patient has arrhythmia of >48hrs or unknown duration and hemodynamically unstable needing emergent cardioversion, or if <48hrs duration but high-risk (valvular A.fib, prior CVA/TIA)

Canadian Cardiovascular Society recommends using **$CHADS_2$ score** to predict risk of CVA

CHF, HTN, Age≥75, Diabetes, TIA/CVA (2 points) – score ≥1 = dabigatran or warfarin; consider ASA if score of 1 and patient reluctant to anticoagulate, but urge follow up with GP or cardiologist; also consider ASA if score is 0 and patient is not young

European guidelines recommend using **CHA_2DS_2VASc** score given that $CHADS_2$ does not include other (but still important) risk factors

CHF, HTN, Age ≥75 (2 points), Diabetes, TIA/CVA (2 points), Vascular diseases (CAD, MI, PVD), Age 65-74, Sex category (female 1 point, male 0 point)

No anticoagulation in younger patients with presumably no structural heart disease and $CHA_2DS_2VASc = 0$

Warfarin

NNT to prevent 1 stroke = 25; **NNH** to cause 1 intracerebral bleed = 400

HAS BLED mnemonic for bleeding risk: HTN, Abnormal renal or liver function, Stroke, Bleeding history, Labile INR, Elderly ≥65yo, Drugs that promote bleeding or excess alcohol use – Score ≥3 means higher (3.7%) risk of major bleeding

Dabigatran

Oral direct thrombin inhibitor with peak effect in 2hrs and no monitoring needed; however, can't check compliance, there is no reversal in major bleeding (attempt dialysis instead), and contraindicated in renal failure; it is also expensive and not covered

As effective as warfarin with slightly lower ICH but slightly higher GI bleeds, and may be associated with increased MI

Dosage: at 110mg PO od, equivalent than warfarin in preventing stroke with significantly lower bleeds; at 150mg PO od, superior than warfarin at preventing stroke with equivalent bleeds

Consider using it in patients with difficult to manage INR

Disposition

Most patients can be discharged home - prescribe medications to rate control if this strategy was chosen, ensure follow up (with TSH testing if not done in ED), and consider follow up with cardiologist to perform Holter, echocardiography or ablation, as well as in first-time presenters or patients with associated CAD or CHF

Indications for Admission: A.fib in association with hyperthyroidism, ischemia, pneumonia or CHF (increased mortality), rate difficult to control

Troponin

In an unpublished review of charts at the University Health Network, 86% of patients had troponins drawn, 14% were positive and 5% of patients were treated as ACS – most of these had hypotension, signs of heart failure, or ECG changes after conversion or rate control

Ischemia may be the result or the cause of A.fib, so consider doing troponins when there are clinical features of ACS present or risk factors for CAD

A.flutter

Look in leads II, III, aVF to see saw-tooth waves (or turn ECG upside down to see them better), especially if the narrow-complex tachycardia is around 150/min

Pharmacological cardioversion and rate control are both MUCH less effective than with A.fib

Unclear if it really takes less energy to cardiovert (50J), so consider using 150-200J initially

Wolff-Parkinson-White(WPW) syndrome

Differential of A.fib with wide QRS: **A.fib with aberrancy** (RBBB or LBBB – QRS usually has typical morphology), or **A.fib with pre-excitation** – eg, WPW: esp. when QRS morphology is bizarre, polymorphic and much faster than usual A.fib (sometimes approaching 300)

NEVER give AV nodal blocking agent (beta-blocker, calcium-channel blocker, adenosine, digoxin and even amiodarone) as the AV node will be blocked and impulses sent preferentially down the bypass tract – which doesn't have any slowing mechanism – and trigger VF

Treatment: electrical cardioversion, or procainamide is the safest medication

ECG of A.fib with WPW: