Case Based Learning Series

Presenting Complaint

67 / M driver with PMH of HTN on unknown Rx referred from RRH by ambulance with

- Worsening dysphagia * 2 days
- Shortness of Breath * 1 week

Audience

- Any additional information?

Expert

What are your initial thoughts?

What is you preparation and approach to this patient?

Primary Survey

- **Airway**: Threatened by the reduced LOC
- **Breathing**: Reduced breath sounds, BL creps & rhonchi
- **Circulation**: PR= 150 bpm , JVD, S1/ S2, no murmurs
- **Disability**: Lethargic, GCS 11 (E3, V4, M4), neck soft, UE3/5, LE 4/5. RUL & RLL normal
- **Exposure**: Normothermic, NGT & urinary catheter insitu

Expert opinion?

Any additional thoughts at this point?

Any additional info you would want to get?

SAMPLE History

- **Signs & Symptom**: Worsening dysphagia & SOB preceded by palpitations
- **Allergies**: No known food or drug allergies
- **Medication**s: Unknown HTNsives. Started on Lasix, Spironolactone & Rivaroxaban. Developed dysphagia then noisy breathing
- **PMX**: Known HTNsive
- **Last Meal**: Was on NGT feeds
- **Events**: Reduced LOC

Family and social history

- No alcohol or smoking
- Family at bedside
- No family history of stroke or heart disease

Labs and imaging

CBC: WBC: 7.29, Neu: 6.4371, PLT: 246, HB: 12.4

RFT: **Cr: 357**, **BUN: 24.6** , Na: 148, K 3.89, eGFR: 18 (?**AKI**)

LFTs: **ALB2**: **30.5**, ALT: 11.0, AST: 13.0 BILD2: 3.2, **TP2: 61.8**

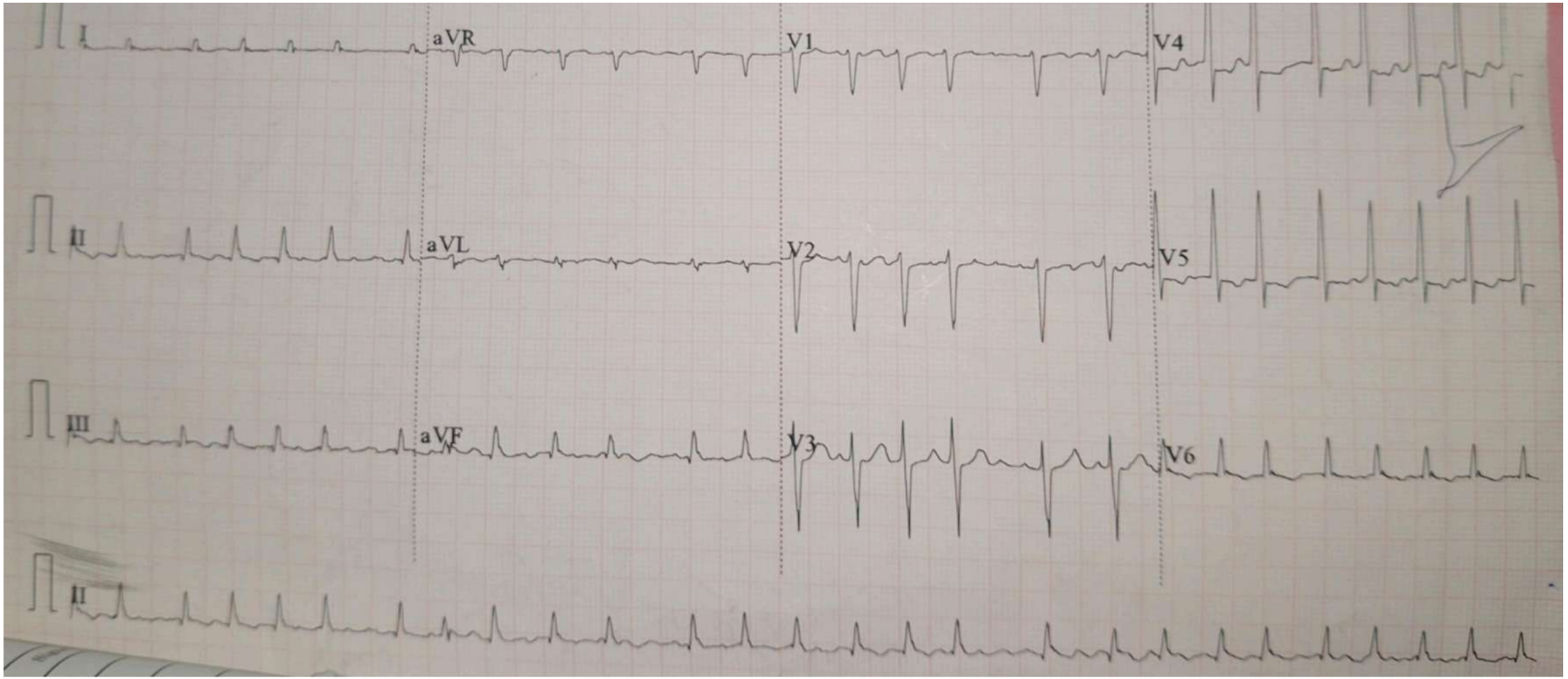
Coagulation Profile**: PT-5: 20.2, PT-INR: 1.63, D-dimer: 20.19**

HIV: Negative

ECG: Attached in the next slide. ECHO: Not done

Brain CT: Included in the next slide

12-lead EKG



CT scan

Findings.

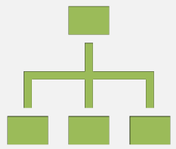
There are multiple intra-axial hypodense (CSF density) lesions in the frontal/parietal lobes (L>>R). There is associated loss of grey white matter differentiation and cerebral sulci. No calcifications. They measure 8.2x1.4 cm and 4.5x8.3 cm on the right and left respectively.

The rest of the brain parenchyma appears normal. The ventricles appear normal. No mid line shift. The ambient cisterns are widened. The other cerebral convexity sulci are patent. The PNS, mastoids and other skull bones are normal.
The cranio-cervical junction appears normal.

Conclusion: The findings are of bilateral multifocal frontal/parietal cortical/subcortical chronic ischemic infarcts with cystic gliosis as described above. Territories of MCA bilaterally. Note is made of grade global brain atrophy -grade 2.

Expert opinion

What are your differentials at this point



What is your management plan?

ED course

- Consults: Cardiology, Pulmonology
- NGT feeding started, IV Amoxiclav 2g BD, IV Hydrocortisone, Salbutamol nebulization, bisoprolol 5mg OD,
- O2 therapy 10L/min by NRM
- Request for ECHO, CTPA, RCT
- Transfer to 4A

Expert

- Additional thoughts
- Disposition plan and consults

Hospital course

- Consults: Cardio, pulm =>
- Patient progress:
- Any additional findings
- Final out come

Highlights/ learning points

1. Altered mental status
2. Shortness of breath
3. Irregular pulse (atrial fibrillation)
4. Cough and feeding difficulties

Day 1 Hospital Course

- Managed for suspected Bronchopneumonia, PTB, COPD, and Atrial fibrillation
- Rivaroxaban 15mg BD, Bisoprolol 5mg OD
- Examination: Mild edema, irregular pulse, bilateral crepitations, SPO2 98%
- GCS 13/15

Diagnosis

- 1. Atrial fibrillation of unknown cause
- 2. Right lower limb deep vein thrombosis (DVT)
- 3. Bronchopneumonia
- Cerebrovascular Accident (CVA)

Differential Diagnosis

- Pulmonary tuberculosis (PTB)
- COPD
- Pulmonary edema
- Left-sided heart failure

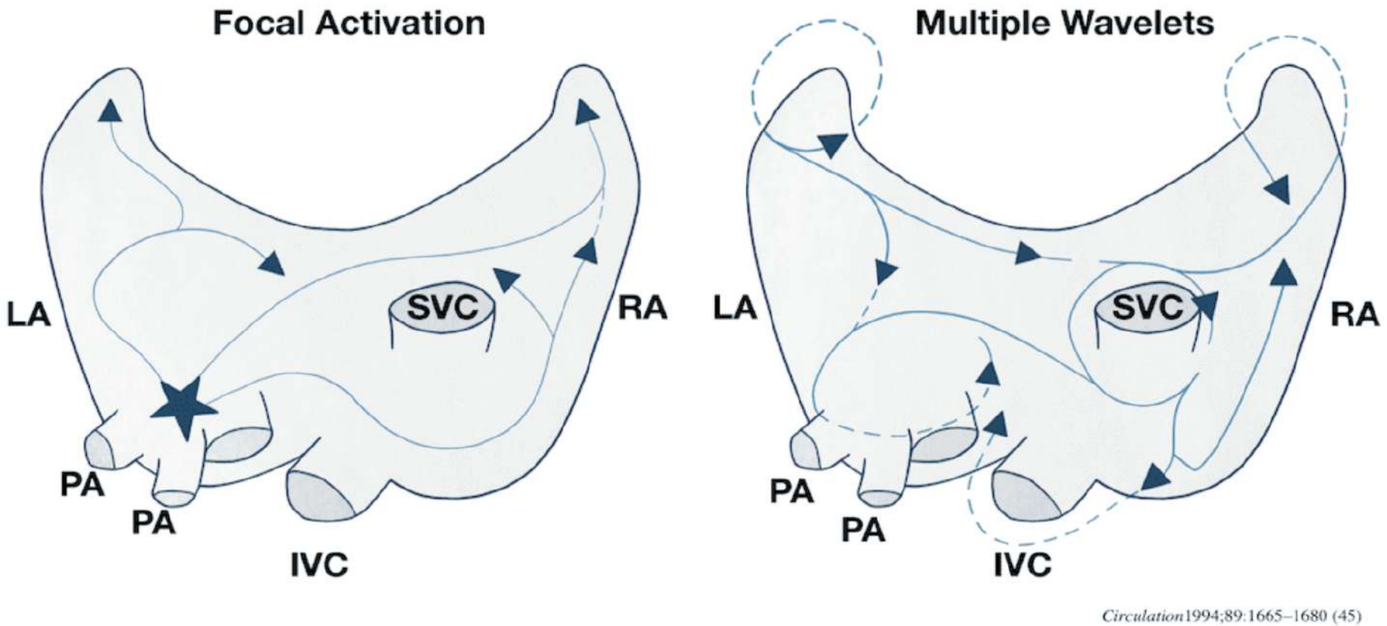
Atrial Fibrillation

- Irregularly irregular rhythm
- No P waves
- Absence of an isoelectric baseline
- Variable ventricular rate
- QRS complexes usually < 120ms, unless pre-existing bundle branch block, accessory pathway, or rate-related aberrant conduction
- Fibrillatory waves may be present and can be either fine (amplitude < 0.5mm) or coarse (amplitude > 0.5mm)
- Fibrillatory waves may mimic P waves leading to misdiagnosis

Classification of A.fib

- Classified depending on the presentation and duration
  - First episode – initial detection
  - Recurrent A. fib >2 episodes
  - Paroxysmal A. fib – self terminating episodes <7days
  - Persistent A. fib – Not self-terminating, duration >7days
  - Longstanding persistent A.fib >1year
  - Permanent A. fib - >1year (not on Rx or unsuccessful Rx)

Mechanisms



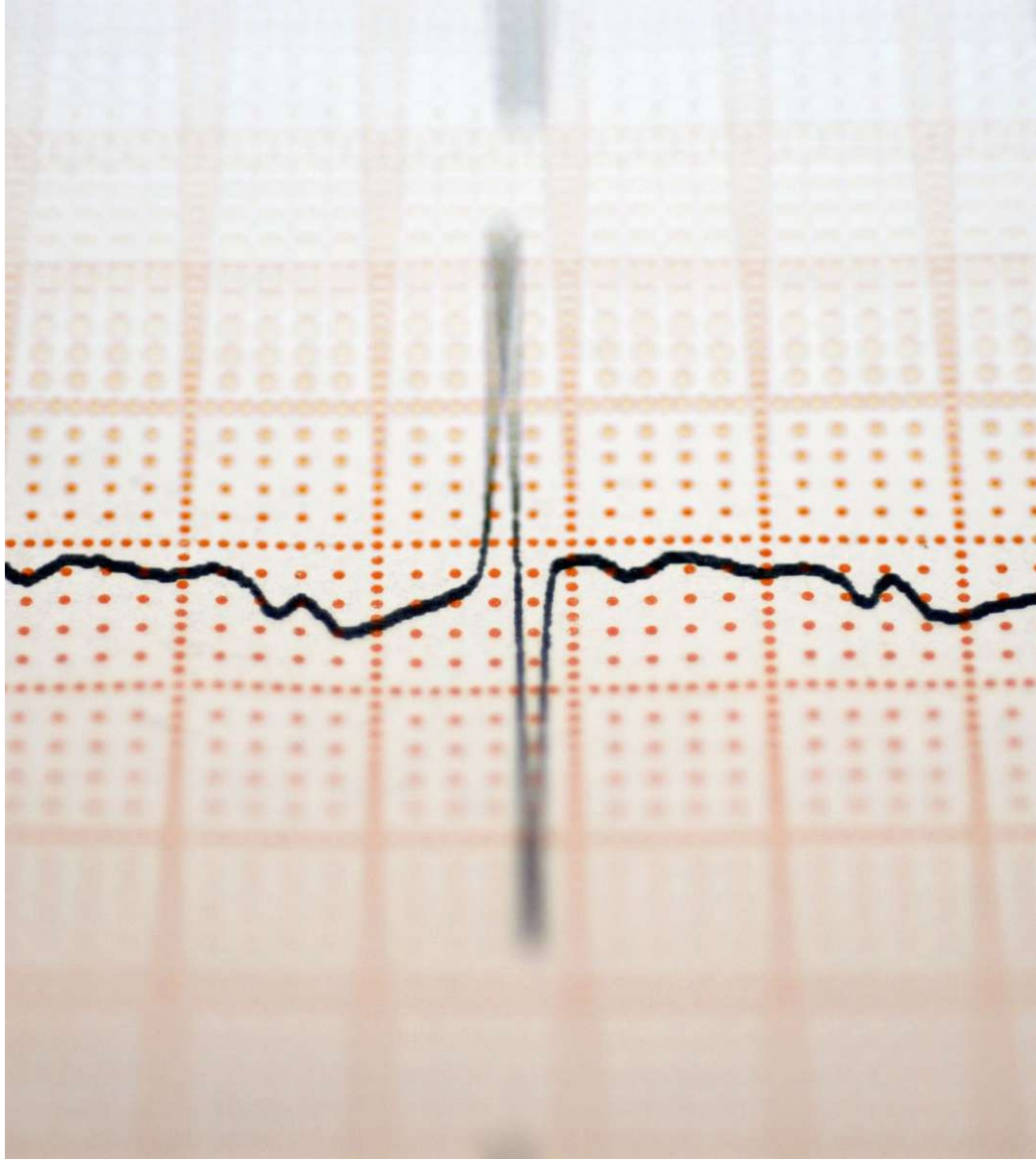
Circulation 1994;89:1665–1680 (45)

Left: Focal activation originating from pulmonary vasculature

Right: Multiple small wavelets, maintained by re-entry circuits

Causes: I HAVE A FIB

- | - Ischemia
- H – Hyperthyroidism
- A - Acute pericarditis
- V - Valvular heart disease (mitral stenosis)
- E - Embolus (PE)
- A - Atrial septal defect
- F - Failure (CHF)
- I - Infection
- B - Booze (orink alcohol, especially in large quentities)

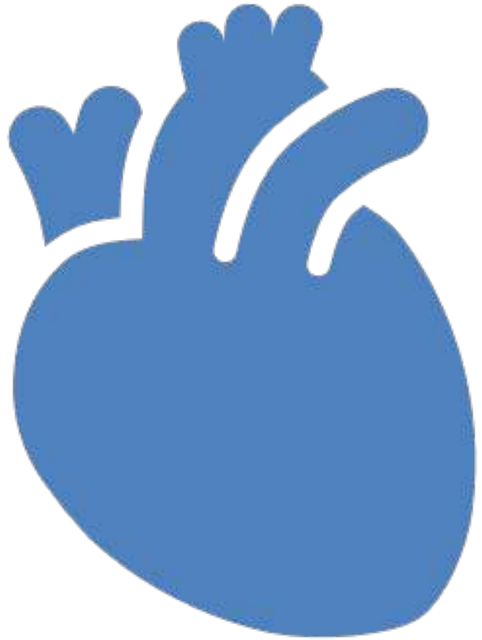


Management

- Stepwise
  - Diagnosis of A. fib
  - Assessment of duration
  - Assessment for anticoagulation
  - Rate or rhythm control
  - Treatment of underlying/associated diseases

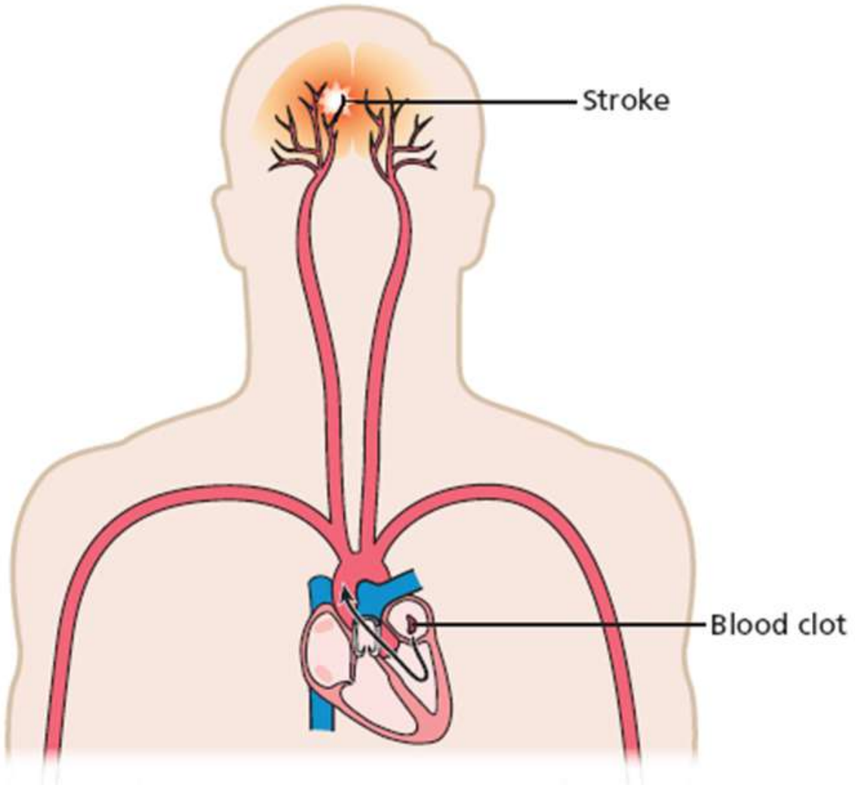
General approach

- Paroxysmal A. fib (Short episodes < 7 days) Lifestyle changes
- Persistent A. fib (Episodes > 7 days): cardioversion
- Long-standing persistent A. fib (> 1 year): poor response
- Permanent A. fib (refractory to RX): Symptomatic management



Atrial fibrillation – thrombotic risk

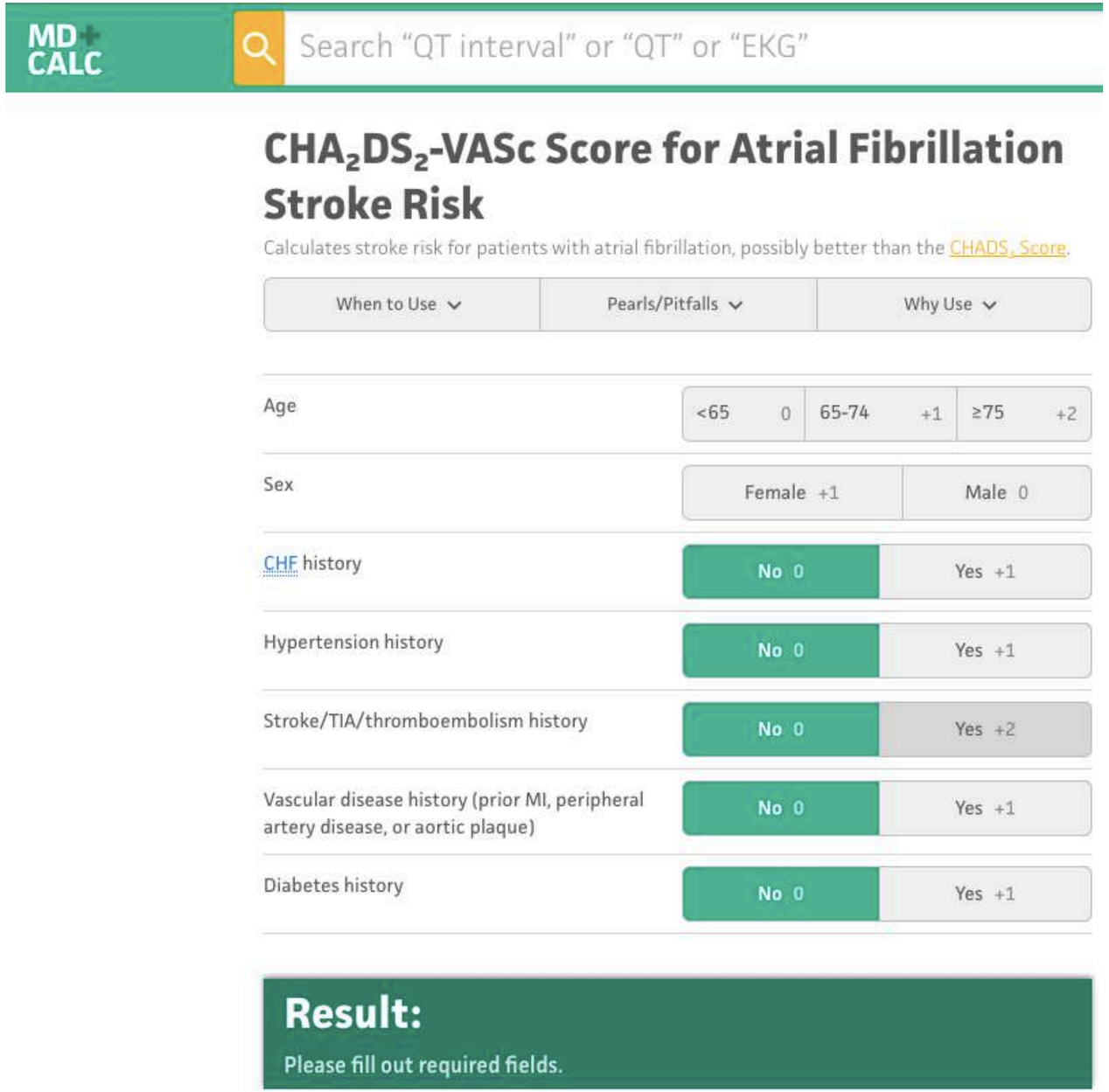
- Disorganized atrial contraction leads to stasis and thrombosis may develop in left atrium



Afib guidelines

- CHA2DS2-VASc score (stroke risk) for all Afib patients.
- Anticoagulation if score is 2 or more
- AF for > 48 hrs or unknown => anticoagulation (3weeks b4 & 4wks after cardioversion)
- Recommend weight loss in obese AF patients
- Consider catheter ablation for low ejection fraction
- Advanced options for patients w/ contraindications to anticoagulation

Risk stratification



Condition	$CHADS_2$ score	Points	CHA_2DS_2-VASc score	Points
Congestive heart failure (or Left ventricular systolic dysfunction)	C	1	C	1
Hypertension: blood pressure consistently above 140/90 mmHg (or treated hypertension on medication)	H	1	H	1
Age ≥75 years	A	1	A_2	2
Diabetes Mellitus	D	1	D	1
Stroke or TIA or thromboembolism in history	S_2	2	S_2	2
Vascular disease (e.g. peripheral artery disease, myocardial infarction, aortic plaque)			V	1
Age 65–74 years			A	1
Sex category (i.e. female gender)			Sc	1

Score of 0: no anticoagulation
Score of 1 (unless point is for female sex): **anticoagulation**
Score of 2: full anticoagulation

Anticoagulation

Direct Oral Anti Coagulants (DOACs) preferred to warfarin except in patients with mechanical heart valves

Do LFTs and RFTs before DOACS.

Low CHA2DS2-VASc => no aspirin

DOACs: apixaban, dabigatran, edoxaban, and rivaroxaban

*DOAC Antidotes Idarucizumab & andexanet alfa are very expensive

Other Features

- Ashman Phenomenon – aberrant ventricular conducted beats
- Rapid ventricular response (RVR) if VR is > 100 bpm
- Slow AF if VR < 60 bpm (hypothermia, digoxin toxicity medications)
- SSS/SND causes and promote development of A. fib

ORIGINAL ARTICLE

A Comparison of Rate Control and Rhythm Control in Patients with Atrial Fibrillation

Author: The Atrial Fibrillation Follow-up Investigation of Rhythm Management (AFFIRM) Investigators* Author Info & Affiliations

Published December 5, 2002 | N Engl J Med 2002;347:1825-1833 | DOI: 10.1056/NEJMoa021328

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Rate vs Rhythm control

- "Mgt of A. fib with the rhythm-control strategy offers no survival advantage over the rate-control, and there are potential advantages, such as a lower risk of adverse drug effects, with the rate-control strategy. Continue anticoagulation in high-risk patients"

FAVOURING RATE CONTROL	FAVOURING RHYTHM CONTROL
Persistent AF	Paroxysmal AF or newly detected AF
Less symptomatic	More symptomatic
Age ≥65 y	Age <65 y
Hypertension	No hypertension
No history of HF	HF clearly exacerbated by AF
Previous failure of antiarrhythmic drug	No previous failure of antiarrhythmic drug
Patient preference	Patient preference

AF—atrial fibrillation, HF—heart failure.
Data from Gillis et al.[1]

Frankel, Grace & Kamrul, Rejina & Kosar, Lynette & Jensen, Brent. (2013). Rate versus rhythm control in atrial fibrillation. Canadian family physician Médecin de famille canadien. 59. 161-8.

Electrical Cardioversion

AFib: Wait-and-See or Early Cardioversion to Obtain Normal Sinus Rhythm?

REBELEM

Take home messages

- Wide range of differentials in the ED
- It’s never black and white in the ED
- Several categories with varying approach
- Preferred management option (rate Vs. rhythm control)
- CHA_2 DS_2 -VASc Score >2, no risk of bleeding, anticoagulated
- Keep rate < 100 at rest

Thank you