Oxford Medical Case Reports, 2025, **6**, 405–412
https://doi.org/10.1093/omcr/omae137
Educational Case Report

Educational case: a 46-year-old woman with palpitations and shortness of breath

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Abstract

In this educational case report, the journey of a woman with atrial fibrillation is discussed, to highlight contemporary management issues in atrial fibrillation. Issues discussed include decisions on anticoagulation, rate and rhythm control, the management of refractory high rates, and the genetic basis of atrial fibrillation.

Keywords: cardiology and cardiovascular systems

Part 1—Acute presentation of atrial fibrillation (AF) with rapid ventricular response

A 46-year-old woman presented to the Emergency Department with palpitations and shortness of breath which began 5 h ago. She had never experienced anything like this before, and denied any chest pain, light-headedness, or syncope. Her past medical history includes asthma for which she is on salbutamol, ipratropium and montelukast. She is not allergic to any medications. She is a non-smoker, drank 40 units of alcohol a week, and worked in an office job.

On examination, she was alert and oriented with warm peripheries. Her body mass index (BMI) is 23. Her chest was clear, and no murmurs were audible on auscultation. Her oxygen saturation was 96% on room air, blood pressure was 130/92 mmHg, and heart rate was 120 bpm and irregularly irregular on manual palpation.

An electrocardiogram (ECG) was performed, as shown in Fig. 1.

1a. What is the most likely diagnosis?

A. Atrial fibrillation
B. Atrial flutter
C. Multifocal atrial tachycardia
D. Atrio-ventricular nodal re-entrant tachycardia
E. Sinus tachycardia

1b. Given the history and examination findings, what is the most appropriate immediate treatment for this patient?

A. Electrical cardioversion without anticoagulation
B. Rate control with a beta-blocker e.g. bisoprolol
C. Rate control with a non-dihydropyridine calcium channel blocker e.g. verapamil
D. Rate control with a cardiac glycoside e.g. digoxin
E. Pharmacological cardioversion with amiodarone

Part 2—Anticoagulation

The CHA_2DS_2-VA score was used to help decide whether this patient would benefit from anticoagulation to reduce the risk of strokes and transient ischaemic attacks (TIA).

2a. What is this patient's CHA_2DS_2-VA score?

A. 0
B. 1
C. 2
D. 3
E. 4

2b. What approach would be appropriate regarding anticoagulation?

A. Commence aspirin
B. Commence direct oral anticoagulants (DOAC)
C. Commence prasugrel
D. Commence warfarin
E. Remain without anticoagulation

Part 3—Chronic management of symptomatic AF

The patient's AF spontaneously cardioverted to normal sinus rhythm and she is discharged. She underwent a transthoracic echocardiogram (TTE) as an outpatient which showed normal

Received: May 30, 2024. **Revised:** August 12, 2024

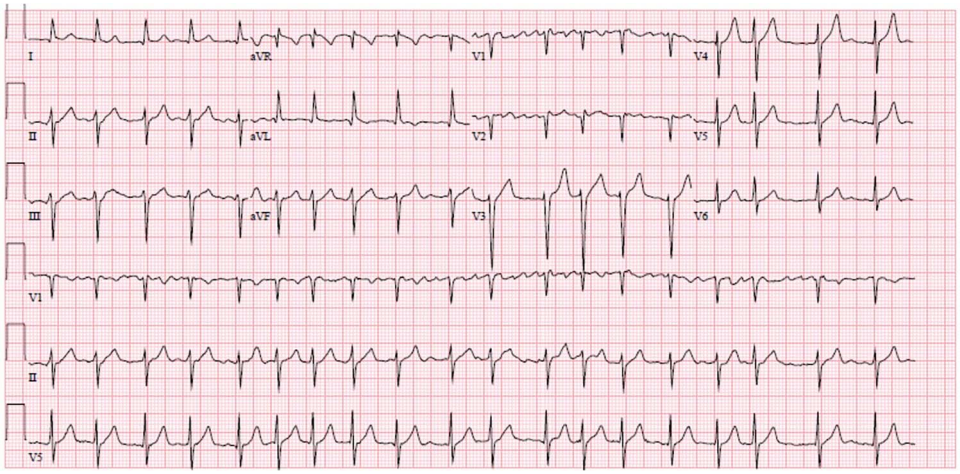

Figure 1. Emergency department ECG.

biventricular size and systolic function with no atrial dilatation or significant valvular heart disease. She was referred to a specialist AF clinic and at follow-up 3 months later has not had any further symptoms, with clinic ECG showing normal sinus rhythm.

3a. What is the appropriate first line pharmacological treatment for the long-term management of AF in this patient?

A. Regular bisoprolol
B. Regular digoxin
C. 'Pill-in the pocket' flecainide
D. 'Pill-in the pocket' sotalol
E. Regular verapamil

3b. What is the single most important lifestyle change that should be advised to this patient?

A. Alcohol reduction
B. Avoidance of caffeine
C. Avoidance of driving
D. Avoidance of strenuous activity
E. Ensuring adequate daily hydration

Part 4—Pacing and ablation

At follow-up 1 year later, the patient reported that she had been experiencing intermittent intrusive palpitations associated with breathlessness most weeks. She had significantly reduced her alcohol intake. Her 12-lead ECG showed a sinus rhythm, with a rate of 52 beats per minute.

4a. What management option should be considered for this patient?

A. Adding on amiodarone
B. Adding on sotalol
C. AF ablation following initiation of anticoagulation
D. Elective DC cardioversion
E. Insertion of a pacemaker

4b. What investigation is mandated prior to AF ablation?

A. Coronary angiography
B. Transoesophageal echocardiogram
C. Cardiac magnetic resonance imaging
D. CT Pulmonary Veins
E. None of the above

Part 5—Revisiting anticoagulation

The patient underwent a successful AF ablation and remained symptom free off antiarrhythmic medications for six years. She then developed palpitations again. In the interim, she had been diagnosed with hypertension, with a mean systolic blood pressure of 165 mmHg, and type 2 diabetes mellitus. At this time, she was fully abstinent from alcohol. Her BMI is 32 kg/m^2. Her ECG showed atrial fibrillation.

5a. What is the patient's CHA_2DS_2-VA score now?

A. 0
B. 1
C. 2
D. 3
E. 4

5b. What is the appropriate next step for this patient regarding anticoagulation?

A. Remain without anticoagulation
B. Commence aspirin
C. Commence warfarin
D. Commence direct oral anticoagulants (DOAC)
E. Commence prasugrel

Part 6—Refractory tachycardia

6. Over the years, she underwent multiple cardioversions on amiodarone and two subsequent re-do AF ablations with confirmation of successful pulmonary vein isolation. Despite this she was unable to maintain sinus rhythm and is diagnosed with permanent AF. At the age of 63, it was noted that her rate control remains suboptimal despite maximal tolerated beta-blocker and digoxin treatment. What management option could now be considered for this patient?

A. AV node ablation and pacemaker insertion
B. Commence vernakalant
C. Elective DC cardioversion
D. Re-do AF ablation
E. Switch DOAC to warfarin

Part 7—Role of Genetics in AF.

7. Upon taking a family history, the patient reported that her mother has also suffered with AF. What is the likelihood of the

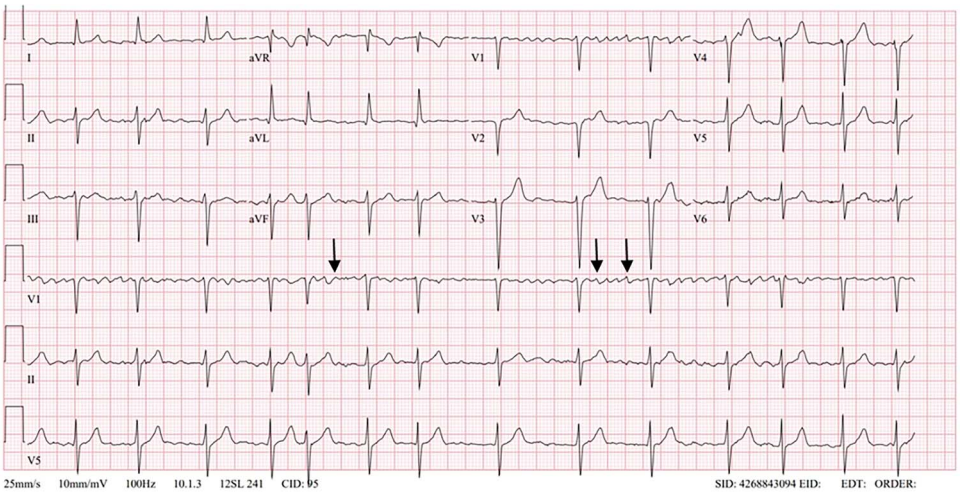

Figure 2. The ECG shows atrial fibrillation with an irregularly irregular rhythm, absence of P-waves, and coarse fibrillary waves in V1.

patient having inherited genetic variants that would predispose them to AF, assuming they are of European ancestry?

A. 12%
B. 22%
C. 32%
D. 42%
E. 52%

Explanations and answers

Part 1a

Correct answer: A. Atrial fibrillation.

This ECG shows atrial fibrillation (AF). There are 3 key differentials to consider for an irregularly irregular narrow complex tachycardia: (1) Atrial fibrillation. The ECG diagnosis of atrial fibrillation is based on an irregular rhythm and absence of P-waves (Fig. 2). Fibrillary waves, usually best seen in V1, may be present but are not necessary for diagnosis. Coarse fibrillatory waves might suggest a greater likelihood of successful rhythm control strategy than fine AF, as the latter may represent more complex atrial disease and therefore arrhythmogenic substrate [1]. (2) Typical atrial flutter with a variable block is characterised by the presence of sawtooth flutter waves at a rate of 300 beats per minute (Fig. 3). (3) Multifocal atrial tachycardia, which is commonly seen in the context of chronic respiratory disease. There are at least 3 different P-wave morphologies visible with an isoelectric baseline (Fig. 4). Multifocal atrial tachycardia is important to differentiate from AF or atrial flutter as it does not mandate anticoagulation. Neither atrio-ventricular nodal re-entrant tachycardia nor sinus tachycardia would present with an irregularly irregular rhythm, and additionally, P waves would be visible with the latter.

Part 1b

Correct answer: C. Rate control with a non-dihydropyridine calcium channel blocker e.g. verapamil.

This patient is clinically stable with no adverse features (hypotension, syncope, ischaemia, pulmonary oedema). Immediate electrical cardioversion is therefore not mandated. DC cardioversion would, however, be a consideration in this patient according to the European Society of Cardiology (ESC) AF guidelines [2]. The patient is young, has only one significant comorbidity, and has no history of thromboembolism, valvular, or structural heart disease. Moreover, onset of symptoms is less than 12 h prior to presentation, and this is the patient's first, or lone, episode of AF. Thus, cardioversion as an immediate management could be appropriate. However, this needs to be done following the immediate initiation of anticoagulation and answer (A) is therefore not correct as DC cardioversion would mandate at least 4 weeks of anticoagulation. There is a theoretical risk of previous short or asymptomatic runs of atrial fibrillation and some therefore advocate a transoesophageal echocardiography (TOE) guided cardioversion to rule out left atrial appendage thrombus where available or a delayed cardioversion approach [3].

Given the patient is haemodynamically stable and the rate of spontaneous cardioversion is reasonably high, we can trial rate control with a non-dihydropyridine calcium channel blocker such as verapamil [2]. Rate control with a beta-blocker would be contraindicated in this patient who has difficult to treat asthma due to the risk of bronchospasm. Digoxin is not a first-line agent for acute rate control in atrial fibrillation and its use should be reserved for patients with a contraindication to first-line therapies or as an adjunct where satisfactory rate control has not been achieved. It is typically more successful for patients with little to no physical activity [4].

Pharmacological rhythm control could also be considered in this patient. Amiodarone, although effective, would not be first line due to its associated longer term side effects in a young patient. A class 1a antiarrhythmic, such as flecainide, would be a more appropriate choice. Flecainide is contraindicated in patients with ischaemic or structural heart disease which has not yet been ruled out using imaging, although the likelihood of this is small in the present scenario. IV vernakalant, where available, can be used in up to moderate left ventricular (LV) systolic impairment with New York Heart Association (NYHA) I or II symptoms and in chronic ischaemic heart disease. Its use should be avoided in outflow tract obstruction conditions such was aortic stenosis and in severe LV systolic impairment or NYHA III or IV symptoms.

Part 2a and b

Correct answer 2a: A. 0.

Correct answer 2b: E. Remain without anticoagulation.

Patients with chronic AF are, in general, six times more likely to develop a stroke [5]. However, on an individual level the risk is dependent on multiple factors. The CHA_2DS_2-VA score is a

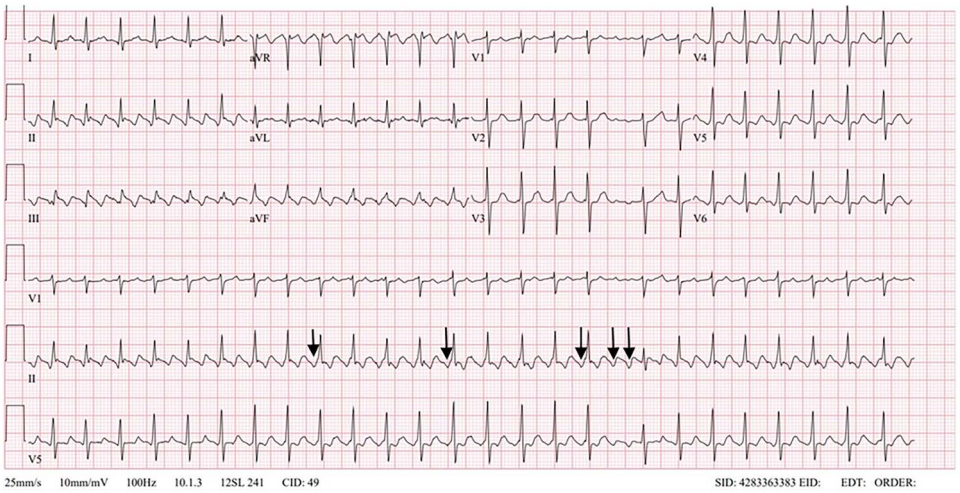

Figure 3. The ECG shows a generally regular rhythm, although there are periods of irregularity corresponding to variable atrioventricular conduction. The arrows mark out homogenous flutter waves, with a classical sawtooth morphology. This is suggestive of typical (cavotricuspid isthmus dependent) atrial flutter. The inverted flutter waves in the inferior leads implies there is anticlockwise conduction.

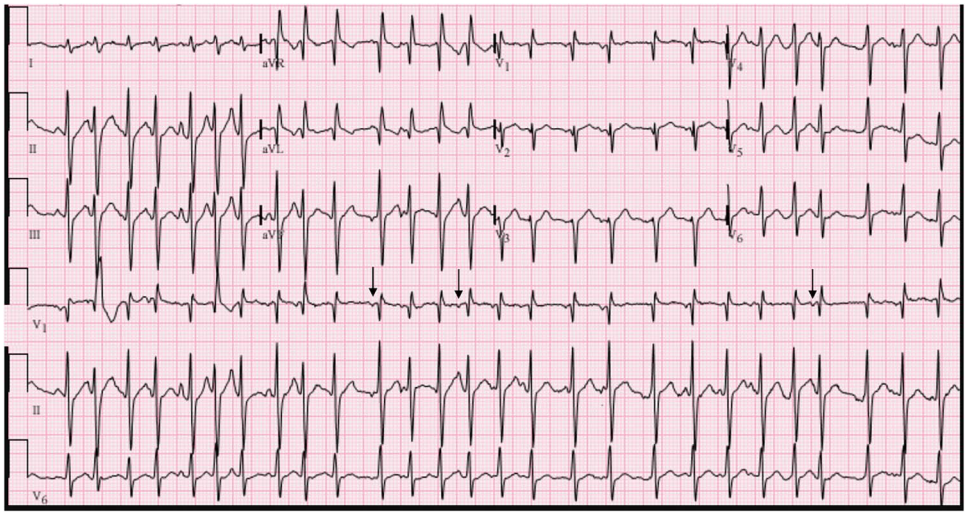

Figure 4. The ECG shows an irregularly irregular tachycardia, with at least 3 discrete P-wave morphologies—Marked by the arrows. This is diagnostic of a multifocal atrial tachycardia. Image adapted from 'Multifocal_atrial_tachycardia' by Jason E Roediger CC ASA 3.0.

well validated stratification tool, recommended by the ESC, for calculating the risk of stroke and other thromboembolic events in patients with non-valvular AF (Fig. 5). The higher the score, the greater the risk. In the recent ESC guidelines for the management of AF, sex (S) has been omitted from the CHA_2DS_2-VASc score, as it was felt to complicate decision making and was recognised not to be fully inclusive [6]. Moreover, given that the ESC had previously recommended different score levels for anticoagulation commencement for men and women, the argument was made that CHA_2DS_2-VA was already effectively in place. This patient scores 0, and therefore does not meet the criteria for anticoagulation. It should be noted, however that the CHA_2DS_2-VA score is not an exhaustive list of all stroke risk factors in AF and that special populations exist where anticoagulation would be mandatory despite a low score; for instance, in those with hypertrophic cardiomyopathy. In addition, anticoagulation may sometimes be initiated for pragmatic reasons, such as if cardioversion or ablation are being considered.

Concomitantly, when deciding on anticoagulation initiation, bleeding risk also needs to be considered. A frequently used score is HAS-BLED (Fig. 6), which calculates the patient's likelihood of a major haemorrhagic event, and our patient would score a 1. HAS-BLED can be used in conjunction CHA_2DS_2-VA to help quantify the risk and benefits of anticoagulation therapy, and

Letter	Risk Factor	Score
C	Congestive heart failure/LV dysfunction	1
H	Hypertension	1
A_2	Age ≥ 75	2
D	Diabetes mellitus	1
S_2	Stroke/TIA/thrombo-embolism	2
V	Vascular disease*	1
A	Age 65-74	1
	Maximum score	8

Congestive heart failure/LV dysfunction means LV ejection fraction ≤40%.
Hypertension includes the patients with current antihypertensive medication.
*Prior myocardial infarction, peripheral artery disease, aortic plaque.

LV: left ventricular; TIA: transient ischaemic attack

Figure 5. Table outlining the components of the CHA_2DS_2-VA scoring system [35].

Letter	Risk factor	Score
H	Hypertension	1
A	Abnormal renal and liver function (1 points each)	1 or 2
S	Stroke	1
B	Bleeding	1
L	Labile INRs	1
E	Elderly (e.g., age >65 years)	1
D	Drugs or alcohol (1 point each)	1 or 2

Figure 6. Table outlining the components of the HAS-BLED scoring system [35]. The ESC defines alcohol excess as > 14 units per week, or when the clinician believes the alcohol intake is significant enough to affect the patient's health.

should be combined with shared decision making when deciding on initiation. It is important to note that a high HAS-BLED score does not necessarily contraindicate anticoagulation, but should prompt further discussions with the patient and efforts to mitigate bleeding risk factors. In the unusual cases where anticoagulation is absolutely contraindicated (e.g. recurrent unaddressable bleeding) consideration can be given to left atrial occlusion devices.

Part 3a

Correct Answer: C. 'Pill-in the pocket' flecainide.

TTE is important to establish the presence structural heart disease, as an aetiology which might itself require treatment, and also to guide management strategies. Blood tests may also identify relevant comorbidities such as thyroid disease, anaemia, electrolyte disturbance, and renal impairment. As this is a young patient with paroxysmal AF and a low burden of symptoms, we could opt for the 'pill-in-the-pocket' strategy with flecainide as per NICE guidelines [4]. Flecainide is appropriate in the absence of structural heart disease; however, it has recently been shown to be relatively safe in LV hypertrophy [7] and in tachycardiomyopathy [8]. With this approach, patients do not take regular medication, but have an antiarrhythmic available to take as a stat dose when they experience an event, thus reducing medication side effects and also the cost of therapy. The ESC recommends class 1c antiarrhythmic drugs—either flecainide or propafenone—as drugs of choice for this strategy, not sotalol [2]. Given infrequent symptoms, there is no need to start regular rate control with verapamil, and beta-blockers would not be appropriate agents for this patient as discussed in Part 1b. Flecainide monotherapy should not be used in atrial flutter due to the potential for reduction in the macro re-entry cycle length and therefore 1:1 conduction in those with slick AV nodal conduction, as this can result in sudden death. For this reason, it is advisable for flecainide to be used in a monitored environment with confirmation of AF by healthcare professionals on its first administration. Alternatively, its administration could be combined calcium channel blockers or beta blockers (where not contraindicated) as these inhibit the AV node and reduce the risk of 1:1 conduction. This is particularly useful when administering flecainide on a regular basis.

Part 3b

Correct Answer: A. Alcohol reduction.

Modifiable risk factors for AF include obesity, hypertension, alcohol intake, obstructive sleep apnoea and dehydration. Caffeine is not a risk factor for AF [9] and some studies suggest that moderate caffeine intake may in fact be protective. Moderate or high alcohol consumption is a major risk factor for atrial fibrillation, particularly in men. Studies and meta-analyses demonstrate that abstinence from alcohol [10–12], or at least a reduction in alcohol intake to 2 units or less per week [13], lowers AF risk. Low level alcohol consumption is more controversial with some studies suggesting a linear dose response relationship [11] whilst others show a threshold effect [14] beginning at moderate consumption. Given that this patient has a significant alcohol history of 40 units a week, it is crucial to counsel her on this risk, including signposting to appropriate support services.

Part 4a

Correct Answer: C. AF ablation following initiation of anticoagulation.

AF catheter ablation involves the targeted destruction of cardiac tissue responsible for triggering or sustaining the arrhythmia. The cornerstone of this therapy, and to date the only strategy with outcomes data, is pulmonary vein isolation. The pulmonary veins may give rise to triggered ectopic activity or micro-entrant circuits which drive AF, and can be isolated typically using radiofrequency (RF) ablation (resistive heating) or cryoablation (coolant typically down to −50 to −60°C), although pulsed-field ablation (electroporation) is emerging as a novel technique. Cryoablation has the advantage of being rapid and can be easily performed without general anaesthetic. RF ablation typically takes long but provides greater flexibility in dealing with anatomical variation as well as the ability to modify other non-pulmonary vein targets. Both techniques have similar overall efficacy [15]. Modification of other substrate targets have not been found to be effective in randomised controlled trials, although success can be achieved in a tailored approach to individual patients. A large, randomised control trial, CABANA, showed no overall benefit from catheter ablation compared with antiarrhythmic drug therapy in terms of the primary end point of death, stroke, major bleeding or cardiac arrest. There was, however, a reduction in secondary outcomes of cardiovascular hospitalisations, AF recurrence and AF burden [16]. Some studies have suggested a mortality benefit of AF ablation in patients with heart failure [17, 18]. ESC guidelines allow for catheter ablation as either first line therapy or following failed antiarrhythmic drug therapy. Early rhythm control strategy has recently been suggested to have superior cardiovascular outcomes [19] which is likely due to a reduction in atrial remodelling caused by longer durations of AF. The ESC recommends that even after successful ablation, therapeutic anticoagulation to be continued for a minimum of 4 weeks, after which its continuation should be decided upon by the patient's stroke risk factors [2].

The patient has a sinus bradycardia without evidence of conduction disease. Pacing is not indicated given that her symptoms are likely attributable to pAF. Moreover, successful pulmonary vein isolation ablation has been shown, on average, to increase heart rate due to modification of the autonomic nervous system, and in some cases this may be sufficient to obviate the need for pacing where the patient was symptomatic from bradycardia [20].

Part 4b

Correct Answer: D. None of the above.

All patients undergoing AF ablation should have assessment of cardiac function, which is most commonly performed using transthoracic echocardiography (TTE). CT, TOE and CMR would all be alternative reasonable methods for assessing the ejection fraction but are not mandatory. If the patient had not been anticoagulated for at least 3 weeks, CT or TOE imaging are preferred for excluding a left atrial appendage thrombus [21]. Given the importance of pulmonary vein isolation some studies have looked at the role of pre-procedure imaging, particularly with CT, for mapping the anatomy of the pulmonary veins and the left atrium, as well as identifying causes for possible post-procedure complications [22]. However, CT guided AF ablation does not improve outcomes or

procedure duration but does increase cumulative radiation exposure [23]. Pre-procedure CT can be conducted where pulmonary venous anatomy is challenging (operator preference) or as part of a research study. Transoesophageal echocardiography would only be required where TTE suggested an underlying structural heart disease that needed clarification, although it can be useful during the procedure to guide transeptal puncture. Ischaemic heart disease is a risk factor for AF but routine coronary angiography would only be required if the patient had suggestive symptoms of angina, and even its role in this setting has more recently come into question when compared with good medical therapy. CMR can also be helpful to delineate difficult anatomy. Atrial fibrosis burden on CMR is prognostic [24], but targeting it for ablation does not improve outcomes [25]. Therefore, none of these modalities are mandated.

Part 5a, b

Correct Answer 5a: C. 2.

Correct Answer 5b: Commence direct oral anticoagulants (DOAC).

Due to the development of further comorbidities—T2DM and hypertension—this patient now scores a 2 on CHA_2DS_2-VA. Thus, she meets the criteria for long-term anticoagulation. She now abstains from alcohol so her HAS-BLED score remains 1 (hypertension). It is worth nothing that, the hypertension definitions are slightly different in the two scores—for CHA_2DS_2-VA any history of hypertension qualifies as a risk factor, whereas for the HAS-BLED score hypertension is considered relevant if it is 'uncontrolled' defined as a systolic blood pressure over 160 mmHg. There are two widely available families of oral anticoagulants to choose from: Vitamin K antagonists (warfarin) or Direct Oral Anticoagulants (DOACs). Both NICE and ESC recommend DOACs as first choice, as these have been shown to have similar efficacy to warfarin in the reduction of embolic events but have slightly lower risk of intracranial bleeding. In addition, they are more convenient in terms of monitoring and interactions. Patients should be counselled on the risks and benefits of both options. Trials have shown that aspirin monotherapy alone is less effective in preventing thromboembolic events [26] with similar rates of bleeding complications. Similarly, dual antiplatelet therapy (aspirin and clopidogrel) provides a degree of increased thromboembolic prevention compared to aspirin alone, but this is less effective when compared to warfarin despite having similar profiles for major bleeding risk [27]. When antiplatelets are used in conjunction with oral anticoagulants, there is no additional thromboembolic prevention, only an increase in bleeding risk [26]. Thus, antiplatelets have no role in prevention of AF-associated thromboembolic events where anticoagulation is appropriate.

Part 6

Correct Answer: A. AV node ablation and pacemaker insertion.

Following failed rhythm control therapy, a rate control strategy would be first line in this relatively young patient, where long term pacing would not be desirable due to risks of repeated generator changes and lead complications. However, should this be unsuccessful or poorly tolerated, a pace and ablate strategy would represent definitive management. This is performed by application of RF energy to the AV node, thus electrically disconnecting the atria from the ventricles and subsequently preventing rapid conduction of the fibrillatory waves. The procedure is permanent and therefore renders the patient pacemaker dependent. In older or more comorbid patients, particularly where conduction disease is suspected, this strategy could be considered earlier or instead of ablation [2].

Choice of device is patient dependent with simple single or dual chamber devices being effective at improving quality of life [28]. Long term right ventricular (RV) pacing can however be detrimental to cardiac function by promotion of dysynchrony. This can be mitigated by use of biventricular (Cardiac Resynchronisation Therapy, CRT) [29] or conduction system pacing. A recent study suggested that AV nodal ablation with CRT in patients with an underlying narrow QRS complex was superior to medical therapy regardless of ejection fraction in patients hospitalised for heart failure symptoms with AF [30].

Given the younger age of this patient, the authors would therefore advocate for CRT or conduction system pacing to mitigate the risks of chronic RV pacing.

Part 7

Correct Answer: B. 22%.

The aetiology of AF is multifactorial and is composed of both environmental and genetic factors. Rare monogenetic mutations, for example in voltage-gated potassium and sodium channels, gap junctions, transcription factors and cytostructural proteins, have been described as direct causes. Recent studies have demonstrated that loss of function titin mutations maybe causative in approximately 2% of patients with AF in the under 65 age group [31]. AF can occur in genetic channelopathies (e.g. Brugada syndrome) and cardiomyopathies (e.g. hypertrophic cardiomyopathy and dilated cardiomyopathy). Mostly AF is likely to be polygenetic. Rates of AF were twice as high in monozygotic twins than dizygotic twins, suggesting a heritability of AF could be up to 62% [32]. In unrelated individuals of European origin AF heritability is estimated to be around 22% [33]. GWAS studies have identified numerous loci of interest in AF often corresponding to non-coding regions of the genome and therefore presumably altering gene expression.

At present, genetic testing is not recommended in routine clinical practice. A family history of AF, especially in young patients, can be helpful in holistic management of the patient and identification of other underlying pathologies. Identification of such patients may allow them to benefit from future advances in genotyping and bioinformatic technologies, potentially guiding targeted therapies [34].

Conflict of interest

No conflicts of interest.

Funding

RKC is funded by a Wellcome Trust PhD clinical fellowship. No funding was given for this educational case report.

Guarantor

RKC is the guarantor for the article, and accepts full responsibility for the work and the decision to publish.

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