Atrial Fibrillation and Other Supraventricular Tachycardias

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Case Presentation

A 49 year old male with a history of non-ischemic cardiomyopathy presented with worsening dyspnea on exertion, nausea, and three days of altered sensorium. A recent echocardiogram demonstrated severely reduced left ventricular ejection fraction (LVEF) of 15–20 %, moderate-to-severe functional mitral regurgitation, a dilated left ventricle, and a severely enlarged left atrium. At his prior office visits, electrocardiograms demonstrated sinus rhythm. He had been on a stable medical regimen of carvedilol 12.5 mg twice daily, lisinopril 40 mg daily, spironolactone 25 mg daily, furosemide 40 mg twice daily and he has not missed any of his medications. He denied dietary indiscretion or symptoms of infection. His heart rate was 106 beats per minute, blood pressure 88/60 mmHg, respiratory rate 26, temperature 98.8 F, and oxygen saturation 90 % on room air. On examination, he had cool extremities, elevated jugular venous pressure to the mandible while seated at 90°, bilateral rales half way up his lung fields, a prominent S3 gallop, a 3/6 holosysolic murmur best at the apex radiating to the axilla, and irregularly irregular, thready central pulses. He had 2+ pitting edema to his knees. He had no focal neurological deficits and his abdominal exam was unremarkable. His labs demonstrated a creatinine at 1.8 mg/dL, a sodium of 130 mmol/L, a potassium of 4.3 mmol/L, and a magnesium of 2.3 mg/dL. His lactate was 2.5. ECG is shown in Fig. 16.1.

Question What is this rhythm?

Answer Atrial fibrillation

This ECG demonstrates an irregularly irregular rhythm without discernable p-waves, consistent with atrial fibrillation (AF). The patient presented in cardiogenic shock. It was uncertain whether the AF was simply one manifestation of his decompensated heart failure or whether the onset of AF with a rapid ventricular rate was the primary reason for his decompensation, due to the rapid rate and loss of atrial contribution to ventricular filling. The duration of the arrhythmia was unknown, so the patient could not safely undergo elective cardioversion without anticoagulation and transesophageal echocardiography (TEE) to verify the absence of atrial thrombus. Because he was not hypotensive, immediate direct current cardioversion (DCCV) was not performed. Options for control of the ventricular rate were limited by his cardiogenic shock. He was admitted to the ICU where a heparin drip was initiated, and he was treated with intravenous furosemide. With diuresis alone, his shock state resolved, his

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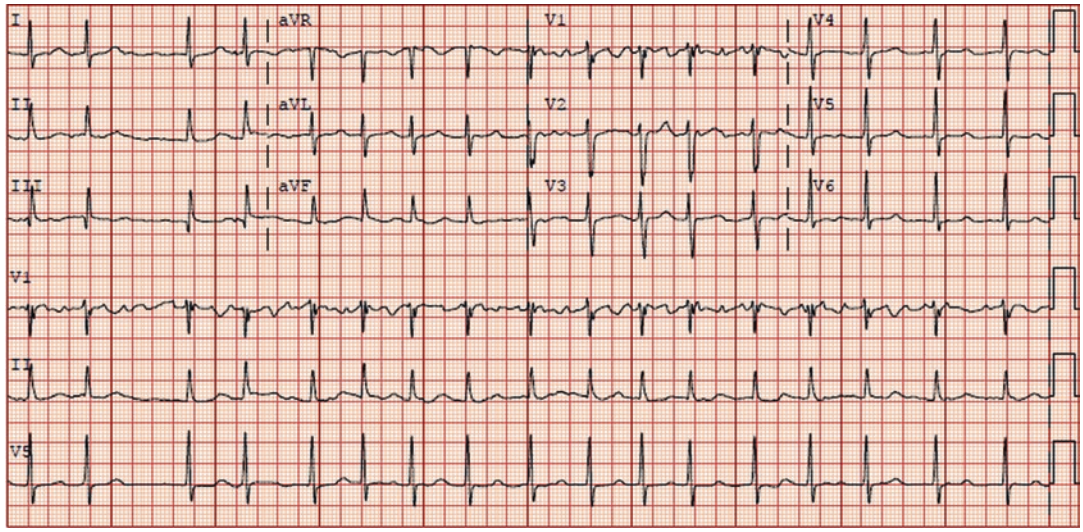

Fig. 16.1 12 Lead ECG with atrial fibrillation

creatinine, sodium, and lactate normalized, but his symptoms of dyspnea persisted. He underwent a TEE guided cardioversion, restoring sinus rhythm. He ultimately was discharged home with follow up with electrophysiology.

Principles of Management

Diagnosis

Conditions which increase the risk for new onset AF:

Triggers for Atrial Fibrillation

Acute/chronic pulmonary: pneumonia, pulmonary embolism, COPD exacerbation, sleep apnea

Heart failure, myocardial infarction, mitral valve disease

Cardiac or thoracic surgery

Acute or chronic hyperthyroidism, alcohol use

The incidence of AF increases steadily with advancing age. AF is commonly classified into three categories: paroxysmal, persistent (sustained longer than 7 days), or permanent [1]. Physical exam demonstrates an irregularly irregular heart rate on auscultation of the heart and palpation of the pulse. ECG findings include a variable R-R interval, with no discernable P-wave preceding each QRS complex. R-R variability may be less apparent at elevated heart rates (Fig. 16.2).

Echocardiograms demonstrate absence of A waves on pulse- and continuous-wave Doppler of the mitral valve in the apical views, along with a single E wave on M-Mode of the mitral valve in the parasternal long axis view (Fig. 16.3).

AF may be asymptomatic or associated with a spectrum of symptoms ranging from palpitations to those of heart failure or cardiogenic shock, severe dyspnea, and lack of energy.

Physiologic Effects

The deleterious effects of AF come in two primary categories: hemodynamic embarrassment and cardioembolism. Hemodynamically, AF results in the lack of mechanical contraction of the left atrium, resulting in depressed preload of the left ventricle. In the setting of heart failure with reduced ejection fraction (HFrEF) or severe aortic stenosis, this acute loss of atrial "kick" can

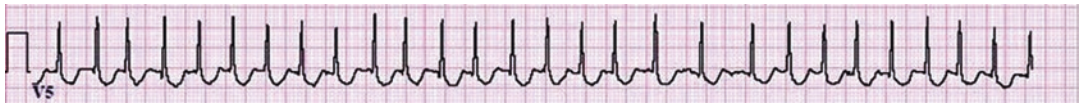

Fig. 16.2 R-R variability with very high ventricular rates

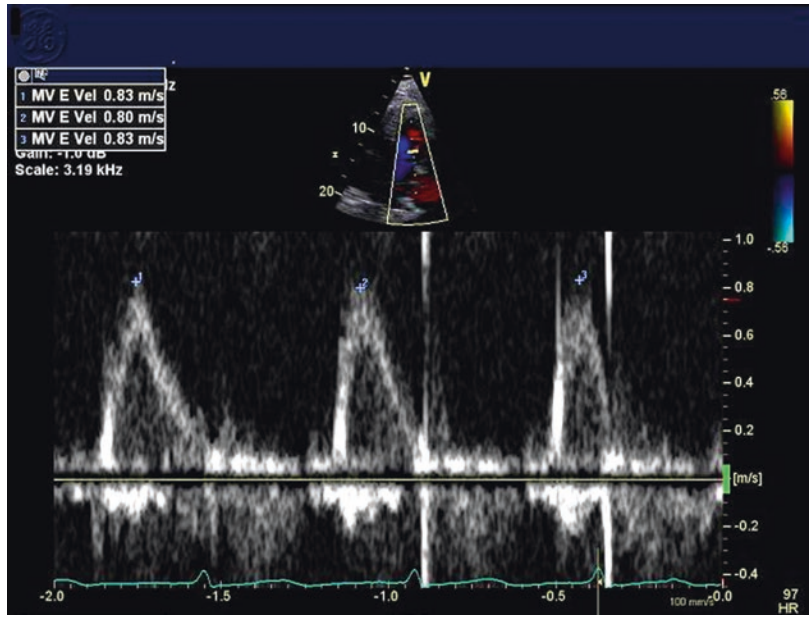

Fig. 16.3 Mitral inflow pattern in atrial fibrillation, with no atrial contraction, just passive filling (E wave only, no A wave)

result in a meaningful decline in cardiac stroke volume and cardiac output [2]. Patients with heart failure with preserved ejection fraction (HFpEF) are exquisitely sensitive to preload so acutely lowering their preload conditions can have rapid deleterious effects on their cardiac function. The same principle applies to patients with hypertrophic obstructive cardiomyopathy (HOCM) and pulmonary hypertension [3]. In structurally normal hearts, some patients may be quite symptomatic from the loss of the atrial "kick", and others may be asymptomatic. In many asymptomatic patients, their first sign of the arrhythmia is an embolic event such as a stroke [4].

A patient's risk of embolic events such as cerebrovascular accidents (CVAs) or ischemic bowel can be calculated using a prognostic model such as the CHADS2-Vasc score that is available through a variety of online risk calculators (e.g. http://www.mdcalc.com/cha2ds2-vasc-score-for-atrial-fibrillation-stroke-risk/) (Table 16.1) [5]. This model has been validated and helps clinicians and patients understand the long term risk for embolic events [6].

Another model, the HAS-BLED score, uses similar inputs, but can help calculate the possibility of a severe bleeding event during anticoagulation (e.g. http://www.mdcalc.com/has-bled-score-for-major-bleeding-risk/) (Table 16.2) [7]. These models help predict risk of ischemic events over the course of years, so apply less to the acute inpatient management of patients with AF.

Treatment Strategies

Aligned with physiologic effects, treatment of AF has two main goals: hemodynamic and embolic risk mitigation. There are two potential strategies to mitigate the hemodynamic impact of AF rate control and rhythm control. Long-term outpatient management of AF was assessed in the AFFIRM trial and despite long-standing

Table 16.1 CHADS2-Vasc score [5]

Risk factor	Score
Congestive heart failure/LV dysfunction	1
Hypertension	1
Age ≥ 75 y	2
Diabetes mellitus	1
Stroke/TIA/TE	2
Vascular disease (prior myocardial infarction, peripheral artery disease, or aortic plaque)	1
Age 65–74 y	1
Sex **c**ategory (i.e. female gender)	1

Table 16.2 HAS-BLED score [7]

Letter	Clinical characteristic	Points awarded
H	Hypertension	1
A	Abnormal renal and liver function (1 point each)	1 or 2
S	Stroke	1
B	Bleeding	1
L	Labile INRs	1
E	Elderly	1
D	Drugs or alcohol (1 point each)	1 or 2

debate as to the applicability of the outcome, no significant mortality benefit to rhythm control was identified [8]. Inpatient management of AF is more guided by symptoms and clinical presentation.

Rate Control

Patients with AF frequently present with rapid ventricular rates which drive their symptoms. Heart rate control is achieved by two main classes of medications: beta-blockers and calcium channel blockers. Both classes of medications slow AV nodal conduction and exert negative inotropic effects. Care must be exercised with the use of these agents, particularly in patients with HFrEF, because of their negative inotropic effects. Diltiazem carries a greater risk of inducing cardiogenic shock and even death in patients with HFrEF, especially if they are in a decompensated state, versus metoprolol. Esmolol may be a reasonable option with very rapid offset that can be trialed in patients who may not tolerate rate controlling agents with negative inotropic effects. Digoxin has modest efficacy but is sometimes the best alternative when beta-blockers and calcium channel blockers are not tolerated. Extrapolation of data from the RACE 2 trial suggests that targeting a heart rate of less than 110 bpm is a safe management strategy, assuming hemodynamic stability (Table 16.3) [9].

Rhythm Control

This method is preferred for patients with acute hemodynamic collapse, acute severe symptomatic AF with controlled rates, and patients in whom AF worsens their symptoms from heart failure. The options for rhythm control include antiarrhythmic medications, direct current cardioversion (DCCV), and/or catheter ablation. DCCV should be considered in patients who are displaying evidence of cardiogenic shock, not solely defined as low blood pressures, but with evidence of end organ hypoperfusion possibly contributed to by loss of atrial contraction. This can be particularly important in preload dependant states such as severe aortic stenosis and hypertrophic obstructive cardiomyopathy. Antiarrhythmic medications are used in accordance with ACC/AHA guidelines and emphasize the importance of structural heart disease and coronary artery disease in the selection of the safest and most effective medication (Fig. 16.4; see ACCF/AHA guidelines http://content.onlinejacc.org/article.aspx?articleid=1854230) [1].

Stroke Prevention

Anticoagulation is critical in patients with AF. Commonly used medications include intravenous unfractionated heparin or subcutaneous low-molecular weight heparin. Prior to undergoing DCCV or chemical cardioversion, patients need to be therapeutic on anticoagulation and most should undergo a TEE, assessing for the presence of left atrial appendage thrombus, if a prolonged period of anticoagulation (≥4 weeks) is not possible prior to elective cardioversion [10]. If an atrial thrombus is present, it is recommended to maintain therapeutic anticoagulation for at least 1 month, after which a repeat TEE should be performed to assess for resolution of the thrombus [11]. With no thrombus present

Table 16.3 Common dosage of intravenous medications for rate control of AF

	Intravenous administration	Usual oral maintenance dose
Beta blockers		
Metoprolol tartrate	2.5–5.0 mg IV bolus over 2 min; up to 3 doses	25–100 mg BID
Esmolol	500 mcg/kg IV bolus over 1 min, then 50–300 mcg/kg/min IV	N/A
Nondihydropyridine calcium channel antagonists		
Verapamil	0.075 – 0.15 mg/kg IV bolus over 2 min; may give an additional 10.0 mg after 30 min if no response, then 0.005 mg/kg/min infusion	180–480 mg QD (ER)
Diltiazem	0.25 mg/kg IV bolus over 2 min, then 5–15 mg/h	120–360 mg QD (ER)
Others		
Digoxin	0.25 mg IV with repeat dosing to a maximum of 1.5 mg over 24 h	0.125–0.25 mg QD
Amiodarone[a]	300 mg IV over 1 h, then 10–50 mg/h over 24 h	100–200 mg QD

[a]Amiodarone should not be used when cardioversion is contraindicated such as in patients not previously anticoagulated

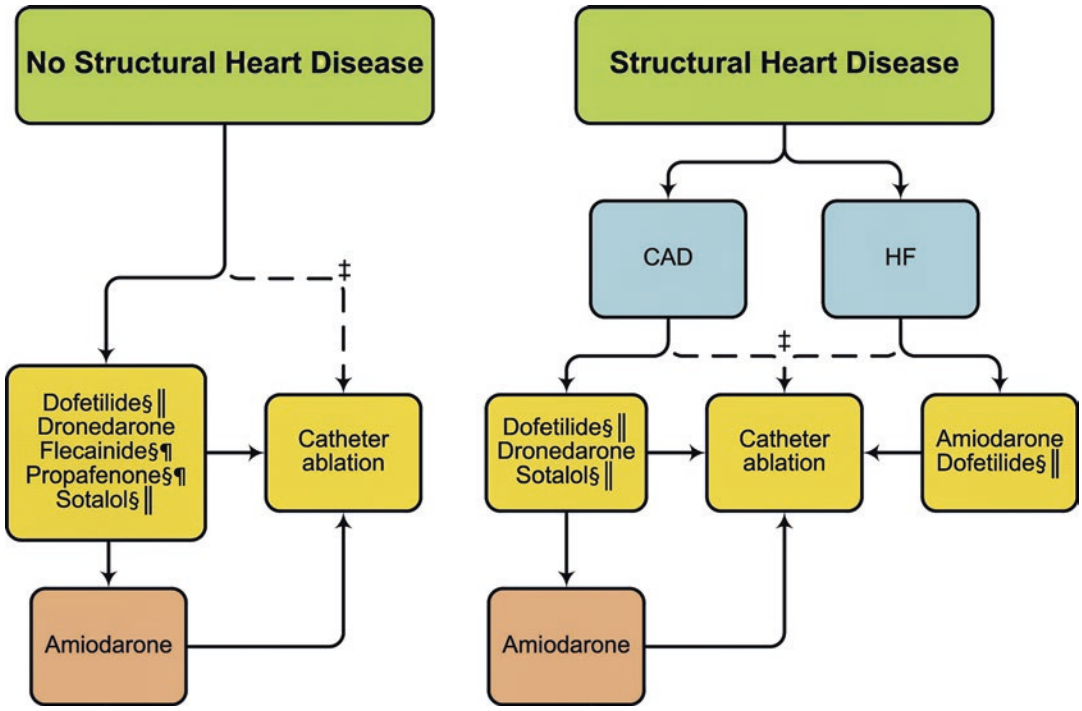

Fig. 16.4 ACCF/AHA guidelines for selection of antiarrhythmic drug therapy for AF

in the left atrial appendage, DCCV can be performed safely while on anticoagulation. The highest risk of embolic phenomena is present within the first month after DCCV, so diligence must be applied to anticoagulation during this period. For patients who acutely develop AF within the hospital or the exact time of onset is known via symptomatology, risk benefit ratio lies in favor of DCCV without TEE guidance if done within the first 48 hours [1].

Other Supraventricular Tachycardias (SVT)

Aside from AF, other non-sinus supraventricular tachycardias are common in critically ill patients. Like AF, these are narrow complex tachycardias unless there is aberrant conduction. These are typically divided according to the relationship between the R-wave and the P-wave, so called short- or long-RP tachycardia. See Table 16.4 and Fig. 16.5 for the differential of short- and long-RP tachycardia.

Hemodynamically, these SVTs can have similar features to AF. In the setting of hemodynamic instability and hypotension, DCCV is an appropriate first response. Diagnosis and acute management often fall under the same action, as many SVTs rely on the AV node for completing their circuit. Breaking that circuit, either with a vagal maneuver or administration of intravenous adenosine, can both reveal and abolish the re-entrant rhythm (Fig. 16.6).

To identify the rhythm, it is recommended to have a 12 lead rhythm strip recording while administering adenosine, so as to capture the termination of the rhythm and possibly to reveal underlying re-entrant rhythm by unveiling flutter waves (Table 16.5).

Certain rhythms, such as atrial flutter, can benefit from a standard catheter ablation, otherwise, treatment with beta-blockers is recommended for short and long term management. If these do not maintain sinus rhythm, catheter ablation may be attempted.

Table 16.4 Differential of short- and long-RP tachycardias

Short RP tachycardias	Long RP tachycardias
Typical AV nodal reentrant tachycardia	Atrial tachycardia
AV reentrant tachycardia using accessory pathway	Sinus tachycardia
Atrial tachycardia with first degree AV block	Atrial flutter
Junctional tachycardia	AV reentrant tachycardia

Evidence Contour

Despite the prevalence of AF (>9 % of Medicare patients in 2010) management still is quite challenging [12]. Certain populations of patients present challenges in dealing with this disease.

High Bleeding Risk in Post-operative Cardiac Surgery Patients

Some studies have demonstrated a near 25 % risk for the development of post-operative AF in patients undergoing cardiothoracic surgery [13]. The development of AF increases hospital length of stay and morbidity [14]. Strategies here often focus on rhythm control, most often achieved with the antiarrhythmic amiodarone [15]. Anticoagulation with heparin products may pose a prohibitive risk for bleeding, so discussion with the surgical team is recommended prior to initiation of anticoagulation in these patients.

Concurrent Inotropic Support

The onset of AF can complicate management of patients with cardiogenic shock on intravenous inotropic support. In these patients, rhythm control is not always possible as the adrenergic stimulus from the inotropic infusions is significantly arrhythmogenic. Rapid ventricular rates are frequently encountered, and rate control is both a challenge and there are no clinical studies to serve as a guide to therapy. Amiodarone and digoxin can be used in these cases, mainly for their rate control effects. Care must be exercised with digoxin in elderly patients, along with those impaired kidney function. Amiodarone should be used with caution in patients with underlying lung, liver, or thyroid disease.

Rhythm Control in HFrEF

In a subset of the AFFIRM trial, rhythm control was preferred in patients with HFrEF [16]. In patients with HFrEF and cardiac resynchronization therapy devices, rhythm control has a lower mortality [17]. For these reasons, it is recommended to try a rhythm control strategy for patients with HFrEF who are still symptomatic with rate controlled AF [1].

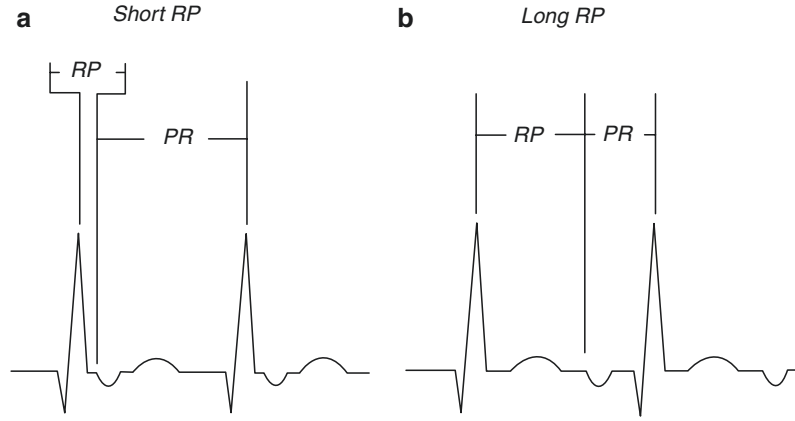

Fig. 16.5 (**a**) Short RP tachycardia, (**b**) Long RP tachycardia (Courtesy: Icyberrounds.com)

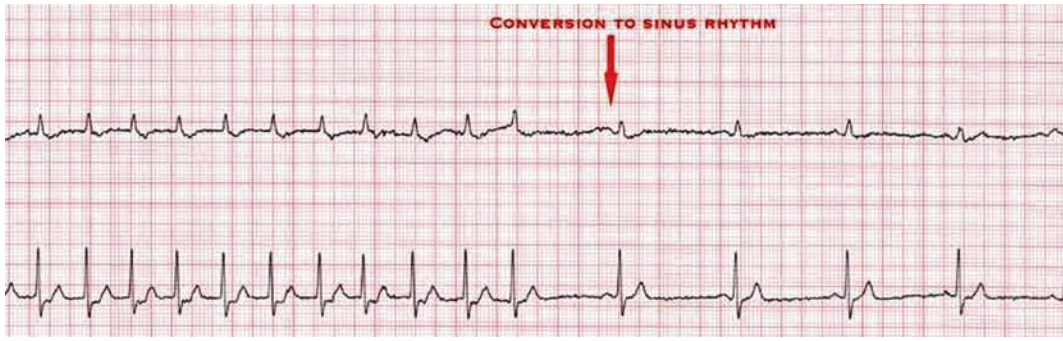

Fig. 16.6 Termination of SVT with adenosine (Courtesy: emedu.org)

Table 16.5 Effect of adenosine on short RP and long RP tachycardias

Short RP tachycardias	Effect of adenosine	Long RP tachycardias	Effect of adenosine
Typical AV nodal reentrant tachycardia	Terminates	Atrial tachycardia	Creates AV-block revealing underlying atrial tachycardia and slowing the ventricular rate
AV reentrant tachycardia using accessory pathway	Terminates	Sinus tachycardia	Creates AV-block revealing underlying atrial rhythm and slowing the ventricular rate
Atrial tachycardia with first degree AV block	Creates AV-block revealing underlying atrial tachycardia and slowing the ventricular rate	Atrial flutter	Creates AV-block revealing underlying atrial flutter and slowing the ventricular rate
Junctional tachycardia	Terminates	AV reentrant tachycardia	Terminates

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