A CASE OF REFRACTORY ATRIAL FIBRILLATION TREATED WITH AMIODARONE

MD ZAHID ALAM[1], PRANOB KARMAKER[2],ROWSAN ARA[3],MONZOOR QUADER[4], MM ZAHURUL ALAM KHAN[5], SHABNAM JAHAN HOQUE[6]

Abstract

We report a case of atrial fibrillation (AF) of unknown duration with fast ventricular rate which was refractory to intravenous (i.v.) digoxin, verapamil and DC (direct current) shock regarding rate control. Amiodarone was given in bolus & then in maintenance dose which ultimately controlled the rate and finally converted into sinus rhythm.

Key words: *Atrial fibrillation, refractory, amiodarone.*

Introduction

AF is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function. It is the most common arrhythmia in clinical practice, accounting for approximately one-third of hospitalizations for cardiac rhythm disturbances. It is classified, according to duration, as paroxysmal (intermittent, maximum duration of <7 days), persistent (< 1 year), and permanent AF (lasts for > 1 year and cardioversion either has not been attempted or has failed). Typically, AF occurs in patients with underlying heart disease, such as hypertensive heart disease[1]. But non-cardiac causes are not uncommon, eg, chest infection or hyperthyroidism[2]. AF is associated with significant morbidity, such as thromboembolism, heart failure (precipitate or aggravate), angina (precipitate or aggravate), and increase in all-cause mortality. Management of AF is largely individualized i.e. according to presentation of the patient and underlying cause. Rate and rhythm control are basic two principle of management and these are achieved with electrical (DC shock) or chemical (antiarrhythmic drugs) cardioversion. Sometimes it is difficult to control AF with both of these methods, and operative procedures (eg. AV nodal ablation with permanent pacing, pulmonary vein ablation, etc) are then effective[1,2]. Here, we present a case that was refractory to conventional drugs and even DC shock, later it responded to amiodarone.

Case Report

A 45-year-old lady, known case of diabetes mellitus (DM) type 2 and hypertension (both were controlled with oral agents) with no history of tobacco or alcohol use, was hospitalized with high grade fever and productive cough for 10 days, and drowsiness with breathlessness for 2 days. Admission electrocardiography (ECG) showed AF with fast ventricular rate [heart rate (HR) was 216/min]. She was transferred to coronary care unit (CCU) for better management. No history (given by her relatives) was suggestive of any structural heart disease or hyperthyroidism. A previous ECG (routinely done 7 months ago as a routine check up) was normal. She responded to vocal command, oral temperature was 102°F, blood pressure 90/60 mmHg, respiratory rate 40/min, no visible goiter, apex beat couldn't be localized, no parasternal heave or palpable P2 and no murmur or added sound was present. Lung findings were compatible with consolidation (diminished movement of left side of the chest with impaired percussion note over left lung field from apex to 6th intercostals space with crepitation over same area). Chest radiograph revealed left upper zone consolidation, complete blood count showed neutrophilic leucocytosis with ESR 40 mm in 1st hour and arterial blood gas analysis showed respiratory alkalosis.

She was attempted for rate control with intravenous digoxin (1 mg) for symptomatic AF with hypotension. HR became 250/min after total 2 mg of i.v. digoxin.

1. Junior Consultant, Cardiology, BIRDEM Hospital, Shahbag, Dhaka.
2. Assistant Registrar, NICVD, Dhaka
3. MD Rheumatology Final part student, BSMMU, Shahbag, Dhaka.
4. Medical Officer, Medicine, BSMMU, Shahbag, Dhaka.
5. Associate Professor, Cardiology, BIRDEM Hospital, Shahbag, Dhaka.
6. Registrar, Cardiology, BIRDEM Hospital, Shahbag, Dhaka.

Then i.v. verapamil (5 mg) was given but the HR reached 300/min after giving 30 minutes of verapamil infusion. At this point, the lady was gasping for breath. As the AF was proved to be resistant to digoxin and verapamil, DC shock was tried with prior i.v. bolus heparin. But after giving total 3 shocks (each 100 joules, synchronized with biphasic defibrillator) the HR became 320/min. This time we started i.v. bolus amiodarone (150 mg over 10 minutes). HR came down to 120/min after bolus infusion. Maintenance dose (1 mg/kg over 6 hours, then 0.5 mg/kg over next18 hours) was continued followed by oral dose (200 mg tds). Blood pressure rose into 100/60 mmHg after 30 min and sinus rhythm was achieved after 10 hours of amiodarone infusion. The lady became fully conscious after 12 hours. She developed sinus bradycardia (40/min) on 6th day of her admission, so oral amiodarone was stopped. Meanwhile, she was also treated with i.v. antibiotics for pneumonia, insulin infusion for DM and aspirin as an antiplatelet. Her ECG showed normal sinus rhythm on 10th days.

Thyroid function test, serum electrolytes and troponin-I (2 samples, each 12 hours apart) were normal. Blood and urine culture showed no growth. After achieving sinus rhythm, an echocardiogram was done which showed diastolic dysfunction (grade 1) with good LV systolic function.

Discussion

As stated above, AF has some non-cardiac etiology. Our patient developed AF most likely due to pneumonia as no cardiac disease that can cause AF could be identified during her hospital stay.

AF is a relatively common arrhythmia that is more prevalent in men and with increasing age[3]. AF can have adverse consequences related to a reduction in cardiac output and to atrial and atrial appendage thrombus formation that can lead to systemic embolization[4-7]. The choice of therapy is influenced by whether the AF is recurrent paroxysmal, recurrent persistent, or permanent (chronic) as defined above[1]. The American Heart Association (AHA) guideline on first-detected AF reached the following major conclusions[1]:

- Measurement of the HR at rest and control of the rate using pharmacological agents (either a beta blocker or verapamil or diltiazem and ,in most cases) are recommended for patients with persistent or permanent AF
- In the absence of preexcitation, intravenous administration of beta blockers (esmolol, metoprolol, or propranolol) or nondihydropyridine calcium channel antagonists (verapamil, diltiazem) is recommended to slow the ventricular response to AF in the acute setting, exercising caution in patients with hypotension or HF
- Intravenous administration of digoxin or amiodarone is recommended to control the heart rate in patients with AF and HF who do not have an accessory pathway
- It is reasonable to use ablation of the AV node or accessory pathway to control heart rate when pharmacological therapy is insufficient or associated with side effects
- Intravenous amiodarone can be useful to control the heart rate in patients with AF when other measures are unsuccessful or contraindicated.
- When the ventricular rate cannot be adequately controlled both at rest and during exercise in patients with AF using a beta blocker, nondihydropyridine calcium channel antagonist, or digoxin, alone or in combination, oral amiodarone may be administered to control the heart rate
- When the rate cannot be controlled with pharmacological agents or tachycardia-mediated cardiomyopathy is suspected, catheter-directed ablation of the AV node may be considered in patients with AF to control the heart rate

Our case was a first-detected symptomatic AF with hypotension. So we proceeded first with i.v. digoxin. However, this case was refractory to digoxin, verapamil and even DC shock probably and possibly due to a potential cause of AF, pneumonia, was present that time. The precise mechanism through which amiodarone suppress atrial fibrillation remains unknown[8]. Amiodarone (with its active metabolite, desethylamiodarone) blocks sodium, potassium, and calcium channels. It is also a relatively potent noncompetitive alpha-blocker and beta-blocker but has no clinically significant negative inotropic effect[1,8]. At rapid heart rates, sodium channel blockade is increased[9]. Amiodarone therapy is initiated with a loading dose (for its delayed onset of action) of approximately 10 g in the first 1 to 2 weeks followed by 400 mg given orally each day for the next 2 weeks[10]. Approximately 30% of patients have a reversion to sinus rhythm during this loading phase, and the remainder can undergo electrical cardioversion, which has a high rate of success[11,12]. Our case was fortunate enough to respond early to amiodarone and did not require surgical procedure.

References

1. Fuster V, Rydén LR, Cannom DS, Crijns HJ, Curtis AB, Ellenbogen KA, et al. ACC/AHA/ESC 2006

Guidelines for the Management of Patients With Atrial Fibrillation. Circulation 2006;114;257-354.

2. Newby DE, Grubb NR, Bradbury A. Cardiovascular disease. In: Colledge NR, Walker BR, Ralston SH, editors. Davidson's Principles & Practice of Medicine.21st ed. Edinburgh:Churchill Livingstone; 2010. P. 561-6.
3. Go AS, Hylek EM, Phillips KA, Chang Y, Henault LE, Selby JV, et al. Prevalence of diagnosed atrial fibrillation in adults: National implications for rhythm management and stroke prevention: the AnTicoagulation and Risk Factors In Atrial Fibrillation (ATRIA) Study. JAMA 2001; 285(18):2370-5.
4. Pritchett EL. Management of atrial fibrillation. N Engl J Med 1992; 326:1264.
5. Atrial fibrillation: current understandings and research imperatives. The National Heart, Lung, and Blood Institute Working Group on Atrial Fibrillation. J Am Coll Cardiol 1993; 22:1830.
6. Lip GY, Metcalfe MJ, Rae AP. Management of paroxysmal atrial fibrillation. Q J Med 1993; 86:467.
7. Disch DL, Greenberg ML, Holzberger PT, Malenka DJ, Birkmeyer JD. Managing chronic atrial fibrillation: A Markov decision analysis comparing warfarin, quinidine and low-dose amiodarone. Ann Intern Med 1994;120:449.
8. Roden DM. Antiarrhythmic drugs: from mechanisms to clinical practice. Heart 2000;84:339-46.
9. Goldschlager N, Epstein AE, Naccarelli G, Olshansky B, Singh B. Practical guidelines for clinicians who treat patients with amiodarone. Arch Intern Med 2000;160:1741-8.
10. Zimetbaum P. Amiodarone for Atrial Fibrillation. N Engl J Med 2007;356:935-41.
11. Singh BN, Singh SN, Reda DJ, Tang XC, Lopez B, Harris CL, et al. Amiodarone versus sotalol for atrial fibrillation. N Engl J Med 2005;352:1861-72.
12. Kochiadakis GE, Igoumenidis NE, Solomou MC, Kaleboubas MD, Chlouverakis GI, Vardas PE. Efficacy of amiodarone for the termination persistent atrial fibrillation. Am J Cardiol 1999;83:58-61.