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Atrial flutter with 1:1 conduction in a 70-year-old man with hyperthyroidism

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Abstract

Atrial flutter (AFL) is a rapid, regular atrial tachyarrhythmia that occurs most commonly in patients with underlying structural heart disease. AFL with 1:1 atrioventricular (AV) conduction is a rare occasion. We describe a 70-year-old male patient with hyperthyroidism in whom AFL was associated with 1:1 AV conduction. This case report emphasizes that AFL with 1:1 AV conduction should be kept in mind as a diagnostic alternative in patients with rapid supraventricular tachycardia and hyperthyroidism. (Cardiol J 2008; 15: 555–557)

Key words: atrial flutter, hyperthyroidism, electrocardiography

Introduction

Atrial flutter (AFL) with 1:1 atrioventricular (AV) conduction, although a rare occurrence, may be fatal [1]. It may be associated with class I antiarrhythmic drug use, accessory AV pathways, hyperthyroidism and exercise [2–5]. We describe a 70-year-old male patient with AFL with 1:1 AV conduction associated with hyperthyroidism.

Case report

A 70-year-old male patient was referred to our institution because of rapid heart rate. He had been diagnosed with hyperthyroidism, AFL and wide QRS tachycardia of unknown etiology 1 month ago at the referring centre (Fig. 1A). The patient complained of dyspnea and frequent rapid palpitations associated with shortness of breath for 2–3 years. He had a history of prior myocardial infarction, heart failure and chronic obstructive lung disease. His current medications included propylthiouracil 50 mg and propranolol 40 mg per day.

On admission, heart rate was 140 beats per minute and regular. Blood pressure was 120/80 mm Hg. There were no S3 or S4 gallops. A grade 1 systolic murmur was audible at the left lower sternal border and the apex. Breath sounds were decreased and expiratory phase was prolonged. A few crackles were heard at the lung bases.

The initial electrocardiogram showed AFL with 2:1 AV conduction and a ventricular rate of 142 beats per minute (Fig. 1B). Intravenous diltiazem slowed AV nodal conduction to 4:1 (Fig. 1C). He was put on diltiazem 180 mg per day and digoxin 0.25 mg per day for rate control. Lisinopril 2.5 mg per day, spironolactone 25 mg per day, enoxaparin 140 mg per day SC and atorvastatin 40 mg per day were added to the treatment regimen.

A transthoracic echocardiogram showed global hypokinesis with an ejection fraction of 0.31. A transesophageal echocardiogram showed no evidence of left atrial thrombus. The thyroid function tests revealed a euthyroid state.

On reviewing the previously recorded wide QRS tachycardia with right bundle branch block (RBBB) morphology (284 beats per minute), we decided that it was probably due to AFL with 1:1 AV conduction and right bundle aberrancy. But owing to the presence of low ejection fraction and a history of myocardial infarction, we decided to proceed with electrophysiological study (EPS) to rule out the

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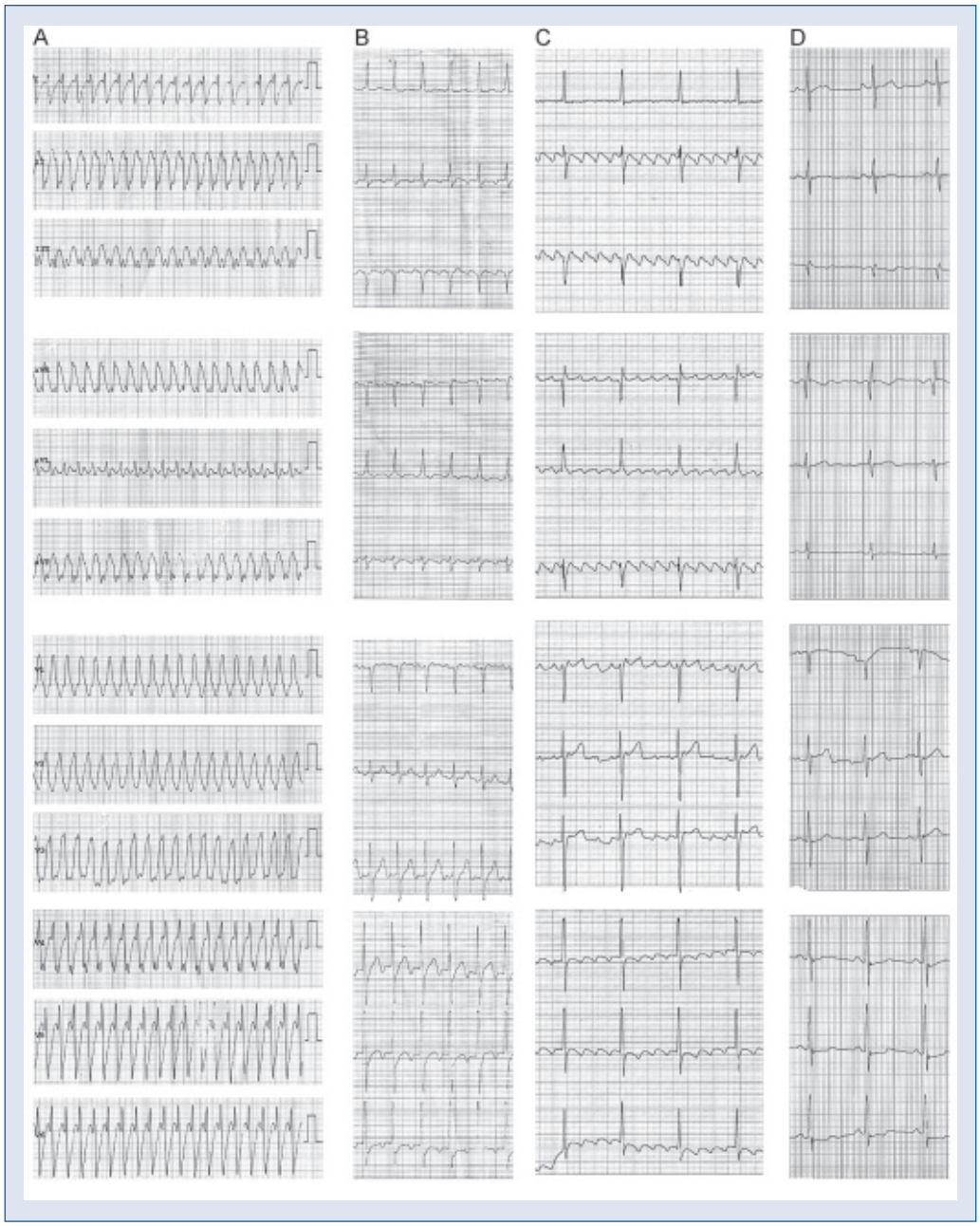

Figure 1. A. Wide QRS tachycardia recorded 1 month before admission; **B.** Admission ECG showing atrial flutter with 2:1 atrioventricular conduction; **C.** Follow-up ECG of patient after administration of IV diltiazem; **D.** ECG after synchronized cardioversion.

possibility of ventricular tachycardia. During the EPS, neither a ventricular tachycardia was induced nor an accessory pathway demonstrated. The AFL could not be terminated with overdrive atrial pacing, but a synchronized 50 J shock was successful in restoring sinus rhythm (Fig. 1D).

The patient was discharged on propylthiouracil, aspirin, diltiazem, lisinopril, digoxin, spironolactone and atorvastatin. At 1-year follow-up, the patient remained in sinus rhythm without the need for any antiarrhythmics. Moreover, ejection fraction was found to be improved from 0.31 to 0.45.

Discussion

Atrial flutter is a rapid, regular atrial tachyarrhythmia. Its incidence is 88/100,000 person-years in the general population, and is 2.5 times more

common in men [6]. The incidence increases with age. The risk of developing AFL increases 3.5 times in subjects with heart failure and 1.9 times in subjects with chronic obstructive pulmonary disease [6]. Hyperthyroidism is also a predisposing factor for AFL. Atrial flutter or fibrillation was reported to be found in 8.3% of patients within 30 days of the date of diagnosis of hyperthyroidism [7]. Male sex, increasing age, ischemic heart disease, congestive heart failure and valvular heart disease are associated with an increased risk of atrial fibrillation or flutter in patients with hyperthyroidism [7].

Due to the physiological properties of the AV node, normally not all of the impulses can reach the His-Purkinje system, so that ventricular rate is lower than the atrial rate during atrial flutter and fibrillation [8]. The most common AV conduction ratio in patients with untreated AFL is 2:1, which produces the typical ventricular rate of 150 beats per minute. Although very rare, life threatening 1:1 AV conduction during AFL may also be seen [1]. Class I antiarrhythmic drugs may slow the atrial rate, thus increasing the likelihood of 1:1 A:V conduction during AFL especially in patients with rapid AV nodal conduction [2]. In patients with accessory AV pathways, impulses may be conducted from the atrium to the ventricle without delay in the AV node [3]. Hyperthyroidism may enhance AV conduction, so it may result in 1:1 AV conduction during AFL [4]. Even in the absence of the aforementioned factors, exercise per se may cause 1:1 AV conduction due to both increased flutter cycle length and enhanced AV conduction [5].

In our case, to elucidate the mechanism of wide QRS tachycardia, EPS was performed. However, during the study neither a wide QRS tachycardia could be induced nor an accessory pathway demonstrated. Also in the hospitalization period no wide QRS tachycardia could be observed. Nevertheless, since the wide QRS tachycardia rate was exactly the same with the admission AFL rate, definite diagnosis was thought to be AFL with 1:1 AV conduction and RBBB aberrancy.

The patient was observed for 1 year. There were no further recurrences of AFL or wide QRS tachycardia during this period. The left ventricular ejection fraction improved from 0.31 to 0.45 implying that AFL with rapid ventricular response might have played a role in the initial low ejection fraction. Luchsinger and Steinberg [9] showed that restoration of normal sinus rhythm by ablation in patients with chronic AFL and cardiomyopathy substantially improves left ventricular function.

In summary, when wide QRS tachycardia with a rate of about 300 beats per minute is encountered in a patient, the clinician should be alert to the possibility of AFL with 1:1 AV conduction and hyperthyroidism.

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References

1. Lee KW, Yang Y, Scheinman MM. Atrial flutter: A review of its history, mechanisms, clinical features, and current therapy. Curr Probl Cardiol, 2005; 30: 121–167.
2. Brembilla-Perrot B, Houriez P, Beurrier D, Claudon O, Terrier de la CA, Louis P. Predictors of atrial flutter with 1:1 conduction in patients treated with class I antiarrhythmic drugs for atrial tachyarrhythmias. Int J Cardiol, 2001; 80: 7–15.
3. Moleiro F, Mendoza IJ, Medina-Ravell V, Castellanos A, Myerburg RJ. One to one atrioventricular conduction during atrial pacing at rates of 300/minute in absence of Wolff-Parkinson--White Syndrome. Am J Cardiol, 1981; 48: 789–796.
4. Suarez WA, Van Hare GF, Wexler ID, Arnold JE. Atrial flutter: An uncommon pediatric manifestation of hyperthyroidism. Pediatrics 1997; 100: E11.
5. van den Berg MP, Crijns HJ, Szabo BM, Brouwer J, Lie KI. Effect of exercise on cycle length in atrial flutter. Br Heart J, 1995; 73: 263–264.
6. Granada J, Uribe W, Chyou PH et al. Incidence and predictors of atrial flutter in the general population. J Am Coll Cardiol, 2000; 36: 2242–2246.
7. Frost L, Vestergaard P, Mosekilde L. Hyperthyroidism and risk of atrial fibrillation or flutter: A population-based study. Arch Intern Med, 2004; 164: 1675–1678.
8. Lau SH, Damato AN, Berkowitz WD, Patton RD. A study of atrioventricular conduction in atrial fibrillation and flutter in man using His bundle recordings. Circulation, 1969; 40: 71–78.
9. Luchsinger JA, Steinberg JS. Resolution of cardiomyopathy after ablation of atrial flutter. J Am Coll Cardiol, 1998; 32: 205–210.