

Case Series

Atrial flutter with 1:1 AV conduction - a case series

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Submitted: 27 May 2024

Accepted: 26 June 2024

Published: 29 June 2024

ISSN: 2373-9312

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ISSN: 2573-1297

OPEN ACCESS

Keywords

- Atrial flutter
- 1:1 AV conduction

Abstract

Atrial flutter (AFL) is a common arrhythmia, and its hemodynamic effect depends upon ventricular rate and the severity of underlying heart disease. In AFL, the atrial rate usually is 250-350 beats per minute and atrioventricular conduction is 2:1 or slower. Rarely, AFL can present with 1:1 AV conduction. In a series of 1037 consecutive patients referred for flutter ablation, 8% presented with 1:1 AV conduction.

INTRODUCTION

Atrial flutter (AFL) is a common arrhythmia, and its hemodynamic effect depends upon ventricular rate and the severity of underlying heart disease. In AFL, the atrial rate usually is 250-350 beats per minute and atrioventricular conduction is 2:1 or slower. Rarely, AFL can present with 1:1 AV conduction. In a series of 1037 consecutive patients referred for flutter ablation, 8% presented with 1:1 AV conduction [1]. In most cases these happen in patients on antiarrhythmic drugs, particularly class 1c drugs which slow the atrial flutter rate leading to 1:1 AV conduction. However very rarely, AFL can conduct to ventricle in 1:1 fashion despite the patient not being on any antiarrhythmic drug and this can lead to a very high ventricular rate and hemodynamic compromise. In last eighteen months in our hospital, we have seen five patients of AFL with 1:1 conduction. Interestingly, four of them were not on any class I or III antiarrhythmics. This case series gives an approximate frequency of the condition in cardiology practice in a district general hospital (DGH) and contrary to popular wisdom, spontaneous 1:1 AV conduction was more common than drug induced in our series. Four cases are described in details below.

CASES**Case 1**

A sixty-five-year-old woman called an ambulance for episodes of palpitation, chest pain and dizziness for 3-4 days. She was known to suffer from paroxysmal atrial fibrillation for a while and was on flecainide 100 mg twice daily and rivaroxaban. She thought that present palpitations were different from her usual occasional paroxysms of atrial fibrillation. Her echocardiogram in the past showed structurally normal heart with preserved left ventricular function. Her coronary angiogram five years ago showed normal coronary arteries. Ambulance crew recorded

two ECGs over a period of twenty minutes. The ECG 1 was done during palpitation and the ECG 2 when she felt better (Table 1).

Looking at the ECG 1, one can consider supraventricular tachycardia with aberrancy, ventricular tachycardia and antidromic atrioventricular re-entry tachycardia as differential diagnosis, but the rhythm is atrial flutter with 1:1 AV conduction with left bundle branch block (LBBB) pattern with very prolonged QRS (QRS 296 msec). This is evidenced by the fact that in the ECG 2 the arrhythmia spontaneously goes into 2:1 AV conduction (ventricular rate becomes exactly half of what it was in ECG 1) and QRS becomes narrow proving that the initial rhythm was atrial flutter with broad QRS and not VT. In terms of aberrancy, there is possibly LBBB, but we know that flecainide can also cause QRS prolongation by prolonging intramyocardial conduction and this effect happens more in high heart rate (use dependence) (Figure 1).

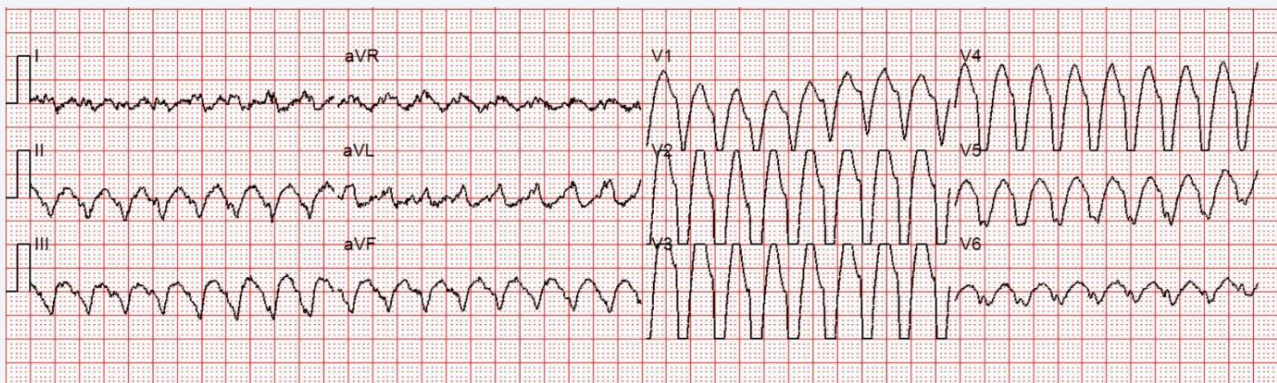
Case 2

A 47-year-old woman was admitted with palpitation and feeling very unwell. She was known to have congenital heart disease and underwent open pulmonary valvotomy, infundibular resection, atrial septal defect repair and patent ductus arteriosus closure at the age of four years. Her echocardiogram in the past showed mild right ventricular dilatation, moderate tricuspid regurgitation and moderate pulmonary regurgitation. Two ECGs at one hour interval showed atrial flutter with 1:1 and 2:1 AV conduction (ECG 3 and ECG 4), respectively. She was initially treated with rate control strategy, and while waiting for DC cardioversion as an outpatient she reverted to sinus rhythm. She was offered an electrophysiology (EP) study and ablation but chose to remain on medication. There was no further recurrence of arrhythmia in nine months on bisoprolol (Figure 2).

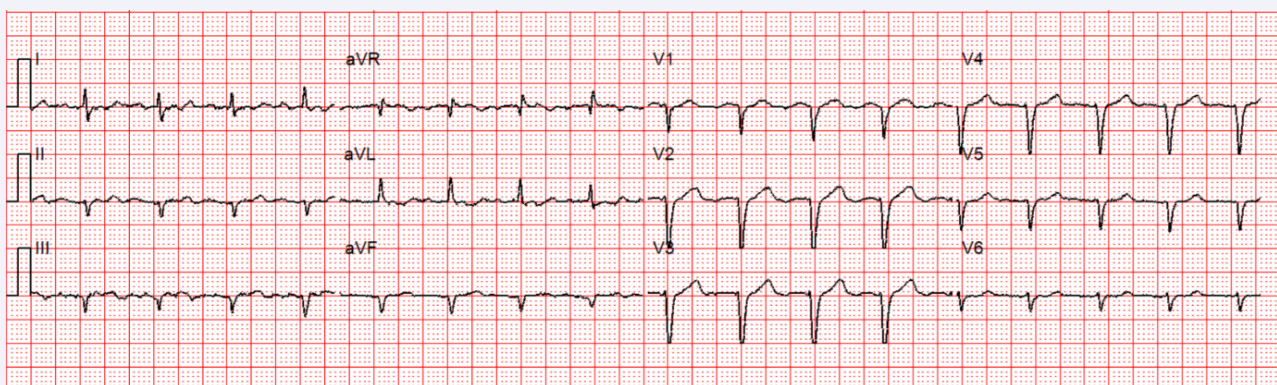
Table 1: Table shows details of the five cases found. One case (case No 1) has already been published (2). Three other cases are described below in more detail

Case No	Age and sex	Any possible contributory factor	H/o atrial fibrillation h/o other cardiac disease	Echo/CMR/Coronary angiogram	Co-morbidities
1	65 yrs, F	Was on flecainide	Known to have PAF	Structurally normal heart, normal coronaries	No co-morbidities
2	47 yrs, F	Was not on any antiarrhythmic	History of surgery for pulmonary valvotomy, infundibular resection, ASD repair and PDA closure in childhood	Echocardiogram showed mild RV dilatation, moderate TR and moderate PR	No co-morbidities
3	50 yrs, M	Was on Olanzapine 2.5 mg OD	No past cardiac history	Echo showed poor LV systolic function during tachycardia but after cardioversion, CMR in sinus rhythm showed normal LV function, no structural heart disease	Was diagnosed to have schizophrenia in the past
4	74 yrs, M	Was not on any antiarrhythmic	Known to have PAF before	Echo normal, coronary angiogram normal	Multi nodular goitre with normal thyroid function, Psoriasis
5	35 yrs, M	Was not on any antiarrhythmic	No past cardiac history	Poor LV function during flutter, Mild LV dilatation and mild LV dysfunction in sinus rhythm after cardioversion	Alcohol abuse

ECG 1



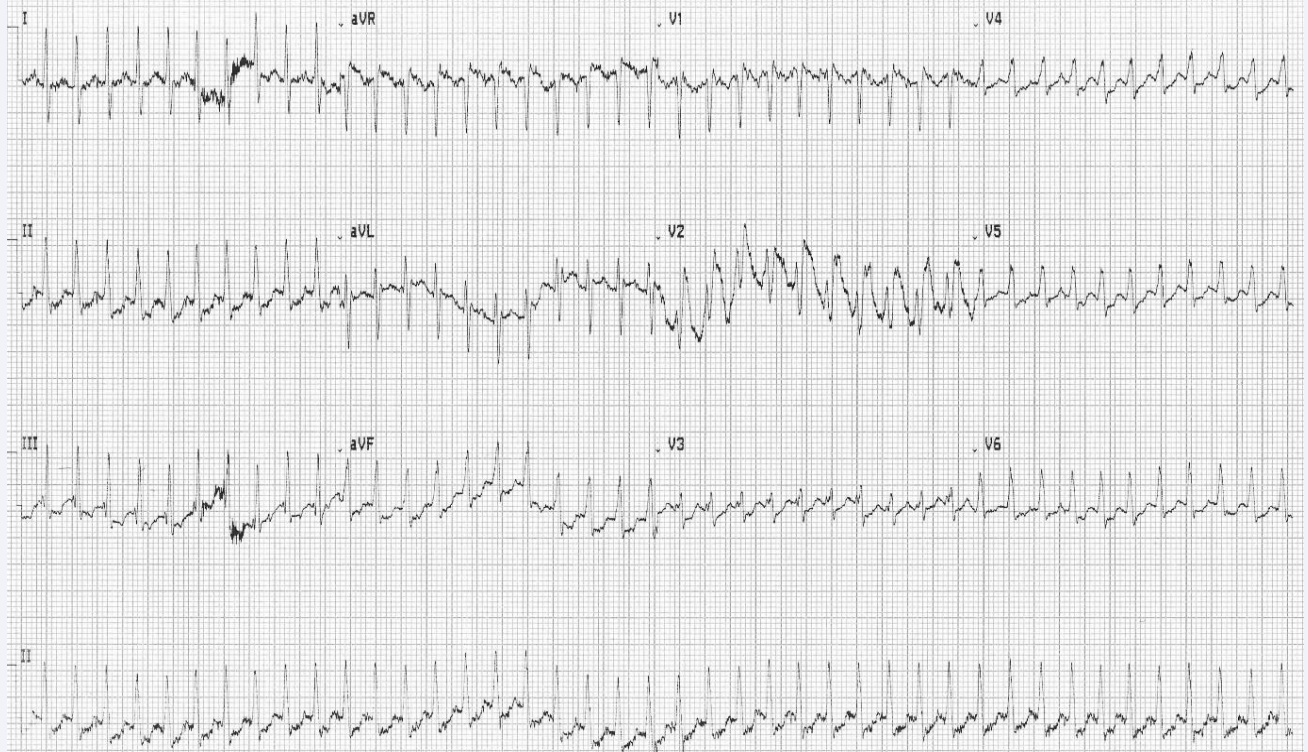
ECG 2

**Figure 1** ECG 1 and ECG 2**Case 3**

A 50-year-old man was admitted with shortness of breath on mild exertion, lethargy, and palpitation for six weeks. Clinically he was in heart failure and ECG showed atrial flutter with 1:1 and 2:1 AV conduction (ECG 5 and ECG 6). Echo showed poor LV systolic function (EF = 20%), bi-atrial dilatation. He had been treated for schizophrenia and discharged from mental

health team many years ago. He was on olanzapine 2.5 mg OD at the time of presentation. He was treated with rate controlling medication (bisoprolol and digoxin), ACE inhibitor, furosemide, and spironolactone. His condition improved and 4 weeks after discharge from the hospital, he had DC cardioversion as outpatient. After cardioversion, in sinus rhythm, cardiac MRI showed normal LV systolic function and there was no evidence of infarction/infiltration/scar. It is difficult to know whether

ECG 3



ECG4

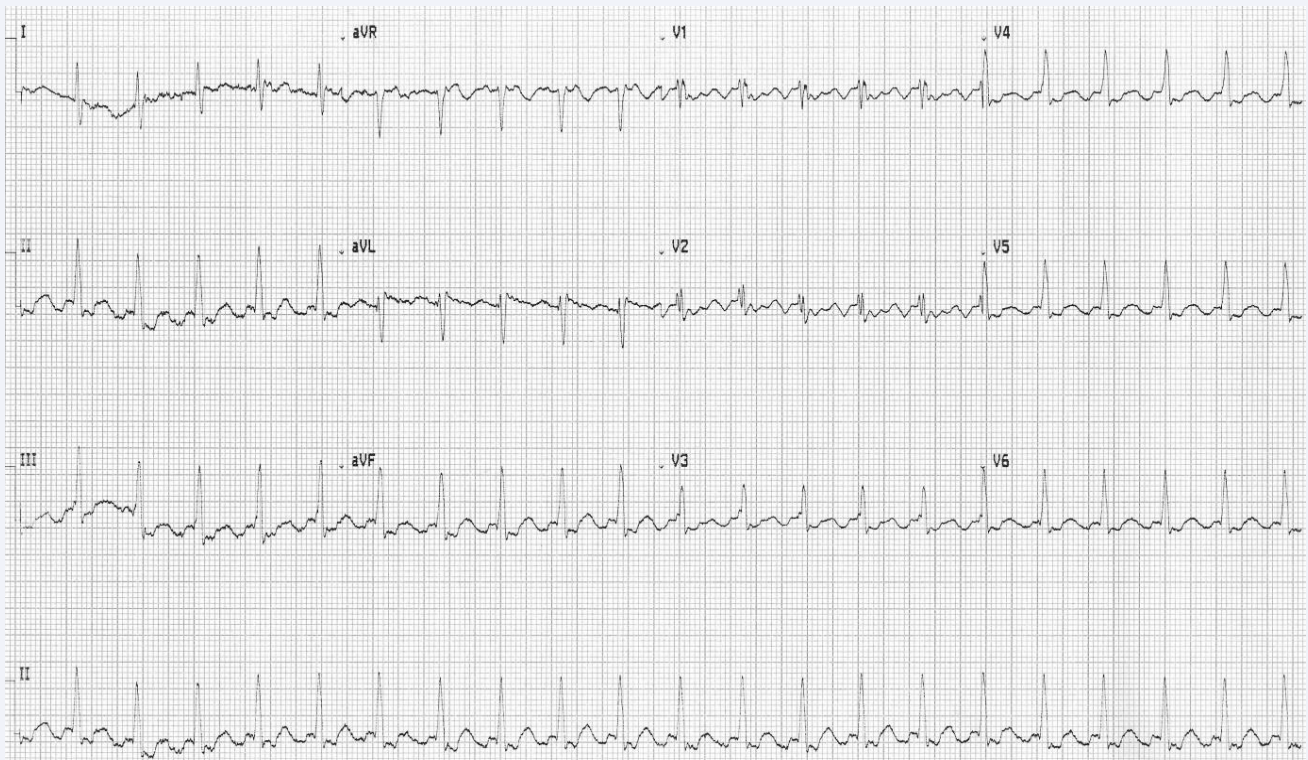
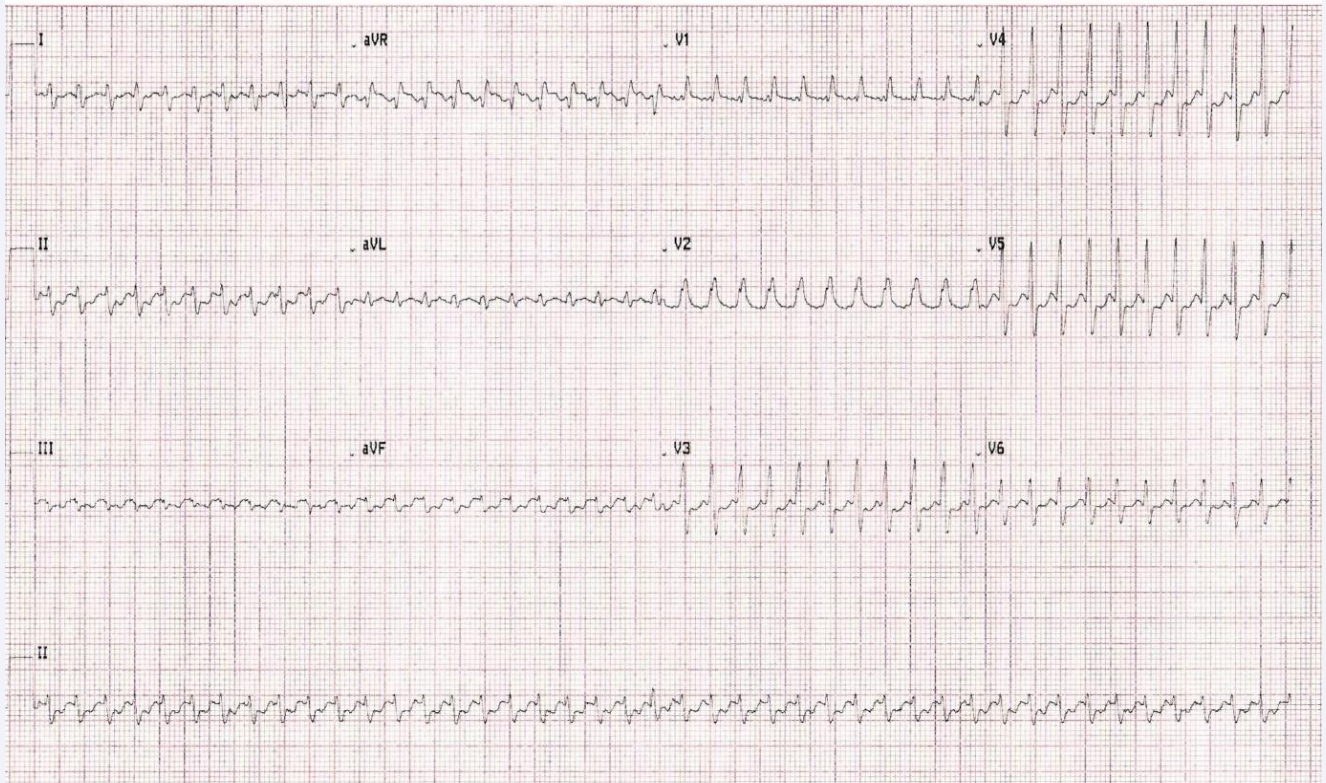


Figure 2 ECG 3 and ECG 4

ECG 5



ECG6

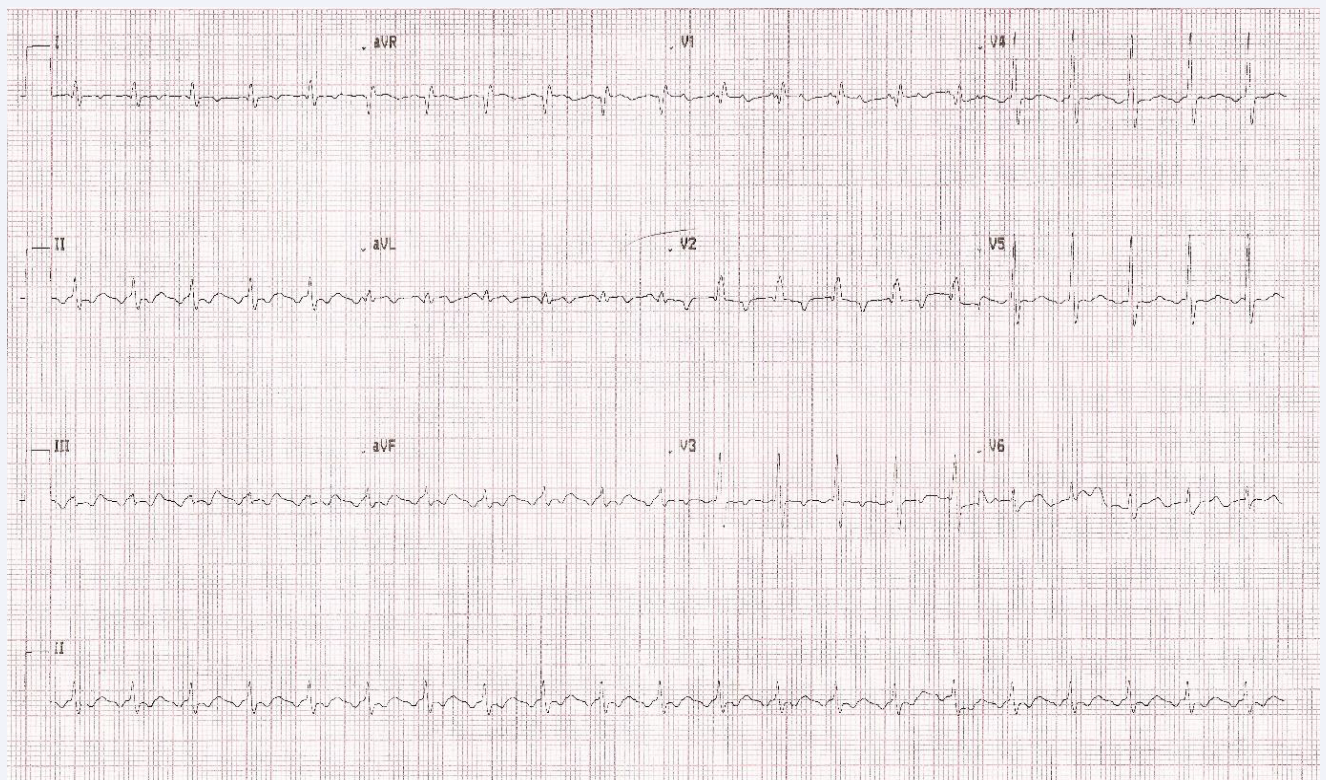


Figure 3 ECG 5 and ECG 6

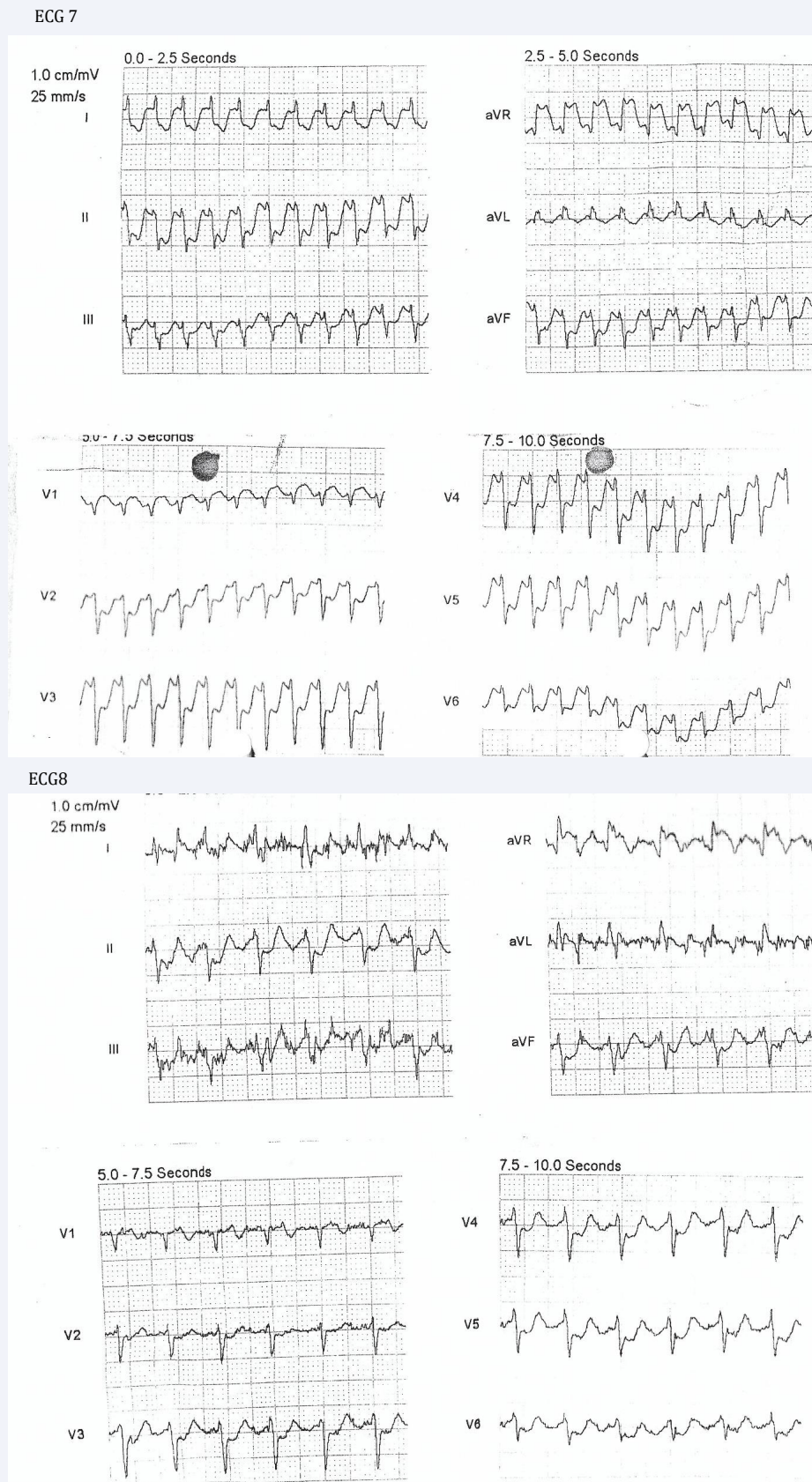


Figure 4 ECG 7 and ECG 8

olanzapine (a muscarinic receptor antagonist) had contributed to increased AV conduction during atrial flutter. He was offered cavotricuspid isthmus (CTI) ablation but chose to remain on medication. He has not had any recurrence in 12 months (Figure 3).

Case 4

A 73-year-old gentleman was admitted with palpitation and near faint. His ECG showed tachycardia at a rate of 280 beats per minute (ECG 7). Within few minutes of first ECG the 2nd ECG (ECG 8) showed tachycardia at a much slower rate, almost half of its original rate. He was not on any antiarrhythmic drug. He had documented paroxysmal atrial fibrillation in the past. Initially, the rhythm was thought to be ventricular tachycardia, but further review of the ECG clearly showed AFL with 1:1 and 2:1 AV conduction.

Coronary angiogram showed unobstructed coronaries and echocardiogram showed structurally normal heart with preserved LV systolic function. EP study failed to induce any arrhythmia and there was no evidence of accessory pathway and there was no AV node duality. He was also known to have multinodular goitre with normal thyroid function and psoriasis.

He had CTI ablation and pulmonary vein isolation procedure and has not had any recurrence of arrhythmia in 15 months (Figure 4).

DISCUSSION

Atrial flutter with 1:1 AV conduction is a rare but a serious

arrhythmia. One needs to be aware of the possibility as an important cause of narrow and wide complex tachycardia at heart rate range of 200-275 beats. It can cause hemodynamic compromise. Intermittent slow conduction (2:1 or slower AV conduction) may give a clue to the diagnosis. If there is no haemodynamic instability, administration of adenosine will increase AV block and reveal the flutter waves in ECG.

Patients with spontaneous 1:1 AV conduction are more likely to have higher flutter rate and less likely to have concomitant AF [2,3].

According to ESC guidelines catheter ablation should be considered after the first episode of symptomatic typical atrial flutter and is recommended after recurrent episodes [4]. There are no specific guidelines for atrial flutter with 1:1 AV conduction but definitive ablative procedure should be considered as first line treatment considering the hemodynamic consequences it can produce.

REFERENCES

1. Béatrice Brembilla-Perrot, Franck Laporte, Jean Marc Sellal, Jerome Schwartz, Arnaud Olivier, PierreYves Zinzius, et al. 1:1 Atrial flutter-prevalence and clinical characteristics. *Int J Cardiol.* 2013; 168: 3287-3290. Debjit Chatterjee. A broad complex tachycardia in a patient on flecainide. *Br J Cardiol.* 2021; 28: 119-120
2. Nathwani VN, Wong KW, Jones MJ, Hayes CH. Case series of atrial flutter with 1:1 conduction: a comparative analysis among groups of patients suffering spontaneous versus drug induced 1:1 conducting atrial flutter. *BCS abstracts.* 2013
3. 2019 ESC Guidelines for the management of patients with supraventricular tachycardia. *Eur Heart J.* 2020; 41: 655-720.