ECG of the Month

Atrial flutter is a common supraventricular arrhythmia. Electrocardiogram (ECG) is necessary for confirmation of diagnosis. It also helps to decide the management.

Keywords: Atrial flutter, Electrocardiogram, Supraventricular tachycardia

Mechanism

Atrial flutter is produced by a macro-reentry (MR) circuit usually involving the cavo-tricuspid isthmus in right atrium¹ (Fig. 1). It is initiated by a premature atrial contraction (PAC) (Fig. 2). Uncommon varieties include left atrial flutter, double wave reentry flutter, lower loop and upper loop reentry right atrial flutter.² Rarely rapid focal discharge may be responsible. It is uncommon in rheumatic heart disease. Hypertension, heart failure, ischaemic heart disease, MI cardiomyopathy and myocarditis are common causes (Fig. 3).

Atrial waves

Flutter (F) waves
Rate is around 250-350/minute.² It can be as slow as 200/min in patients treated with antiarrhythmic drugs. Rarely it can be as fast as 400/min (Fig. 4).

‘F’ waves have regular saw-tooth appearance without any isoelectric line (Fig. 5, lead II). These waves are best seen in leads II, III, aVF and V1.² Rarely ‘F’ waves may be seen clearly only in precordial leads V1 to V3.³ In precordial leads, ‘F’ waves may mimic discrete P waves with isoelectric line³ (Fig. 5). Rate and configuration in inferior leads helps

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**Fig. 1: Showing mechanism of atrial flutter-macro-reentry (MR)**

**Fig. 2: Showing initiation of atrial flutter by a premature atrial contraction (PAC) and spontaneous conversion to sinus rhythm**

**Fig. 3: Showing atrial flutter in a case of inferior infarction**

**Fig. 4: Showing fast atrial flutter (F)**

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Fig. 5: Atrial flutter (lead II) with discrete P waves and isoelectric line (arrow) in lead V1.

differentiation from atrial tachycardia. Such discrete ‘F’ waves are seen specially in atria that have extensive scar.\(^1\)

Depending on the configuration of ‘F’ wave in lead II, III and aVF, atrial flutter can be following:

**Type 1:** (Common, typical, counterclockwise) - flutter waves are negative (inverted) in leads II, III and aVF\(^2\) (Fig. 6).

**Type 2:** (Uncommon, reverse typical, clockwise) - flutter waves are positive in leads II, III, aVF\(^1\) (Fig. 7).

Atrial flutter not involving cavo-tricuspid isthmus may not have diagnostic contour but rate between 250-350/minute helps diagnosis. Flutter morphology may change during same episode of flutter\(^2\) (Fig. 8). It may spontaneously change to atrial fibrillation.\(^3\)

Digitalis promotes conversion of atrial flutter to fibrillation (Fig. 9 A,B) by shortening the refractory period of atrial myocardial cells.\(^2\) As atrial flutter changes to atrial fibrillation, ventricular rate may change from rapid and regular to relatively slow and irregular.\(^3\) Careful analysis of basal atrial rhythm helps in correct diagnosis. Control of ventricular rate by drugs is easier in atrial fibrillation than in atrial flutter. Atrial flutter may spontaneously revert to sinus rhythm (Fig. 2). If saw-tooth appearance shows some irregularity or distortion suggestive of atrial fibrillation, it is termed “atrial flutter-fibrillation” (Fig. 10). Most of these cases need to be managed as atrial fibrillation.

**Atroventricular conduction and ventricular rate**

In atrial flutter, atrial rate is around 300/minute. In normal persons, atrioventricular node can conduct up to 180 impulses per minute due to it’s long refractory period. Therefore, only alternate ‘F’ wave crosses the A-V node to reach the ventricle (2:1 A-V conduction). This results in classical ventricular rate of 150/minute in an untreated patient.\(^2\) In such an ECG, diagnosis of atrial flutter may be difficult because one of the ‘F’ waves is partly or completely masked by QRS and the other is masked by T wave.\(^3\) Such ECG is likely to be confused with atrial or AV nodal re-entrant tachycardia (Fig. 11). Procedures or drugs that slow AV conduction can

Fig. 10: Showing “atrial flutter-fibrillation”

Fig. 11: Atrial flutter (clear from initial beats) going into 2:1 conduction mimicking atrial or A-V nodal reentry tachycardia

Fig. 12 (A): Showing unmasking of atrial flutter (A) with 2:1 AV conduction and (B): by carotid massage
unmask the ‘F’ wave. These include carotid compression (Fig. 12 A, B), digoxin, verapamil, diltiazem and adenosin.

1:1 atrio-ventricular conduction can occur in patients with an accessory pathway with short refractory period. This can cause regular wide QRS complexes without clear atrial activity and may lead to an erroneous diagnosis of VT. Blocking of AV node does not help in diagnosis as antegrade conduction is over an accessory pathway. Electric cardioversion is effective in termination of such tachyarrhythmia. Presence of pre-excitation on previous or subsequent ECG in sinus rhythm can help diagnosis. Hyperthyroidism and inherently fast AV nodal conduction may also produce 1:1 conduction (Fig. 13A). Carotid compression helps in unmasking atrial flutter in such cases (Fig. 13B). Concomitant occurrence of VT with atrial flutter can also produce nearly similar ECG. Changing relation between ‘F’ wave and QRS (AV dissociation) can help diagnosis.

Atrial flutter rate is reduced by antiarrhythmic drugs that prolong refractory period of atrial muscle (quinidine, procaainamide, flecaainide) (Fig. 14). If atrial rate is reduced up to 200/min, each atrial impulse may reach AV node after recovery of its refractory period and may cross AV node (1:1 conduction). This results in increase in ventricular rate. Anticholinergic effect of these drugs also facilitates AV conduction. Therefore, in management of atrial flutter, class Ia antiarrhythmic agents should be used with drugs that block AV conduction (digoxin, beta-blockers, verapamil) to avoid increase in AV conduction and ventricular rate.

If there is slow AV conduction due to AV nodal disease or drugs (digoxin, verapamil, adenosine) atrio-ventricular ratio may increase from 2:1 to 3:1 or 4:1 with corresponding ventricular rate of 100/min or 75/min Atrio ventricular conduction is most often even numbered e.g. 2:1 or 4:1. Odd

numbered atrio-ventricular ratio e.g. 3:1 or 5:1 are uncommon (Fig.15).

Atrio-ventricular block can produce the following
- Regularly irregular ventricular response e.g., alternating 2:1 and 4:1 conduction (Fig. 16). This occurs because of Wenckebach block and produces group beating of QRS3 (Fig. 16)
- Irregularly irregular ventricular response e.g. switching between different AV conduction ratio (Fig. 17)
- Constant relation between ‘F’ and QRS suggests 2nd degree AV block (Fig. 18).
- Changing relation between ‘F’ and QRS suggests complete AV block (Fig. 19).

QRS configuration
- Normally the QRS is narrow due to normal intraventricular conduction.
- Electrical alternans may be present at fast rate (Fig. 20 A, B)

Fig. 15: Showing AV conduction changing between 2:1 and 3:1

Fig. 16: Showing group beating due to alternate 2:1 and 4:1 conduction

Fig. 17: Showing irregularly irregular ventricular rhythm due to changing AV conduction

Fig. 18: Showing atrial flutter with bradycardia and fixed ‘F’-R interval

Fig. 19: Showing atrial flutter with bradycardia and changing ‘F’-R interval (arrow)
Fig. 20(A): Lead V1 showing atrial flutter (F) with 2:1 A-V conduction (B): Lead V4 showing electrical alternans

Fig. 21: Atrial flutter with complete AV block showing intermittent change in QRS configuration (R) due to fusion of ‘F’ and QRS

Fig. 22(A): Lead II showing atrial flutter with alternating 2:1 and 4:1 conduction (B): Lead V1 showing aberrant conduction (R) in beats with short cycle length

- Atrial flutter with complete AV block can result in intermittent fusion of ‘F’ and QRS resulting in intermittent change in QRS configuration (Fig. 21)
- Regular broad QRS may be seen with
  + Pre-existing bundle branch block
  + 1:1 AV conduction with aberrancy
  + Antegrade conduction over accessory pathway
  + Concomitant VT
- Irregular broad QRS can occur with

Fig. 23: Showing false impression of ST elevation due to superimposition of flutter waves (arrow) in leads V1, V2 and V3

- Aberrant ventricular conduction: It is suggested by
  + rSR configuration in lead V1 (Fig. 22 a and b)
  + Broad QRS in short cycle following a long cycle
  + Initial QRS vector identical to narrow QRS
  + Change in QRS configuration with change in preceding cycle length

- Ventricular ectopics
  + They may have LBBB configuration. If QRS is upright in lead V1 it is either uniphasic or biphasic. Initial deflection is not identical to narrow QRS. QRS configuration does not change with length of preceding cycle length.

ST segment
Atrial flutter waves may give false impression of ST segment deviation (Fig. 23).

References