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When narrow and wide complex tachycardia meet in one patient

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ABSTRACT

Evaluation of arrhythmias, especially in the acute setting can be challenging. One of the most crucial steps is to accurately differentiate whether a tachyarrhythmia is of supraventricular (with aberrant conduction) or ventricular origin. A 12-lead electrocardiogram may be useful in some cases where specific morphology or features can be sought.

1. Introduction

This is a case where it appears as if the patient had several different arrhythmias within a short span of time. It turned out to be atrial flutter–fibrillation, with a transient left bundle branch block pattern.

This can be explained via the mechanism of enhanced or accelerated atrio–ventricular nodal conduction, together with a refractory period occurring in the left bundle branch.

2. Case report

PSF is a 58 year old gentleman with a history of hypertension and hyperlipidaemia, on diet control. He has no history of ischaemic heart disease (IHD) or thyroid problems and is not a regular coffee drinker.

He presented for the first time with giddiness and palpitations whilst sitting at his desk at work.

The symptoms came on suddenly and there were associated cold sweats. He went to the general practitioner (GP) across the road from his office, who found he had tachycardia with a heart rate of 162, and blood pressure (BP), 122/75 mmHg.

He was afebrile at that time and had no symptoms of upper respiratory tract infections.

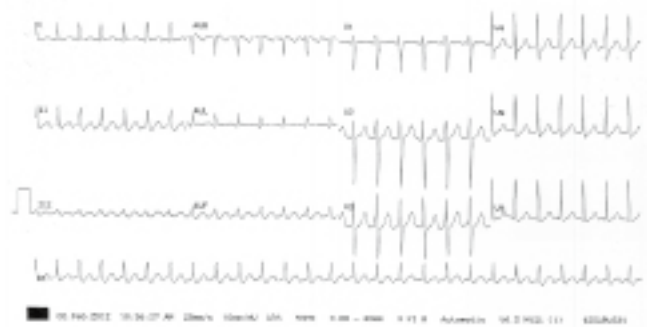


Figure 1. ECG at 1 056 h: atrial flutter with regular 2:1 block.

The GP documented and recorded several 12-lead electrocardiograms (ECGs), as seen in Figures 1–6. These showed:

- Atrial flutter with regular 2:1 block at 1 056 h

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(Figure 1)

- Broad complex looking tachycardia at 1 108 h

(Figure 2)

- Atrial fibrillation at 1 110, 1 113 and 1 116 h

(Figure 3–5) and finally

- Normal sinus rhythm at 1 118 h (Figure 6)

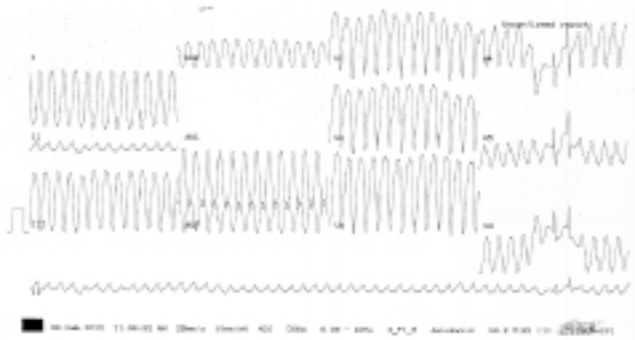


Figure 2. ECG at 1 108 h: appearance of broad complex tachycardia but it is atrial flutter with 1: 1 conduction and left bundle branch block.

The two narrow complex beats seen in Leads V₄₋₆ illustrates beats where the impulse could be transmitted down the bundle branch and conducted in the normal way.

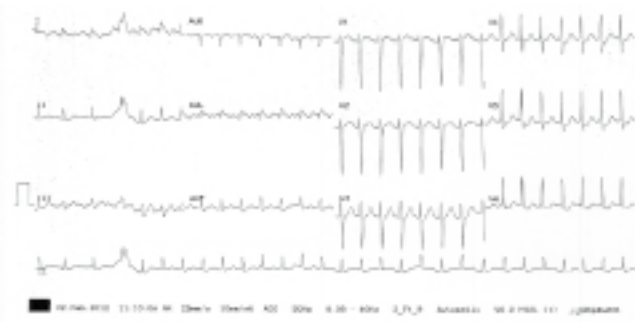


Figure 3. ECG at 1 110 h showing atrial fibrillation.

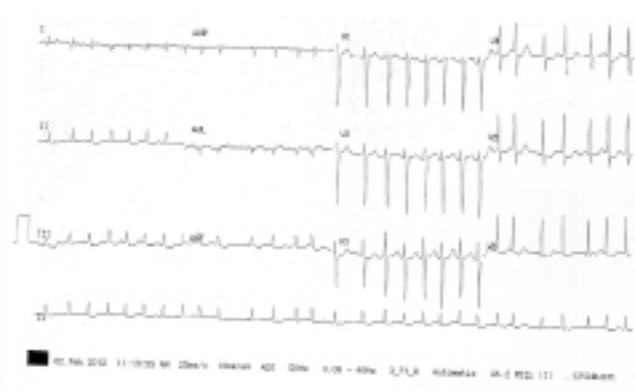


Figure 4. ECG at 1 113 h: atrial fibrillation.

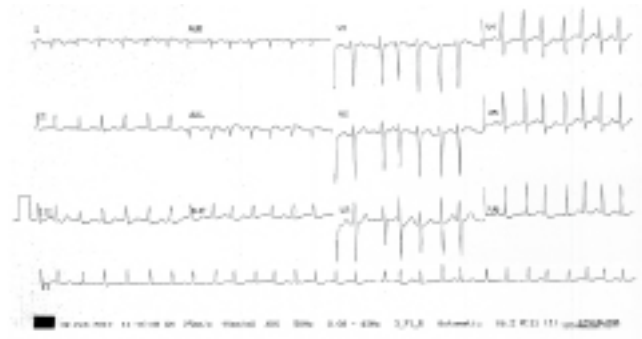


Figure 5. ECG at 1 116 h: atrial fibrillation.

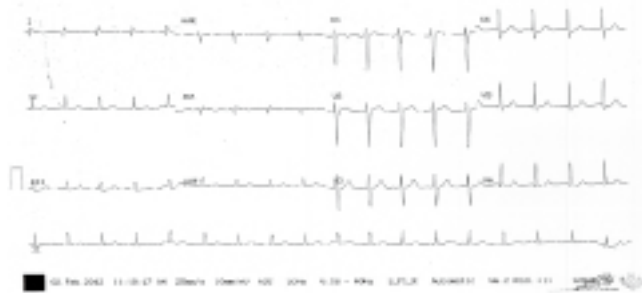


Figure 6. ECG showing normal sinus rhythm at 1 118 h.

The transition from one rhythm to the other, as in the timeline above was not associated with the administration of any drugs or intervention, by the GP. He was subsequently sent to the Emergency Department (ED) for management.

His BP remained stable. His full blood count, urea, electrolytes, calcium, magnesium, phosphate, thyroid function test and cardiac markers were all within normal limits. The chest X-Ray was also normal.

He remained in sinus rhythm and was admitted to a telemetry bed in the Cardiology ward. PSF underwent electrophysiological studies (EPS) the next day. He was noted to have no inducible atrial or ventricular tachycardia. No ablation was done.

He was discharged well with Clopidogrel and Bisoprolol. At one month follow up, he remained well and had not experienced any further attacks



Figure 7. Normal sinus rhythm at 1 200 h.

3. Discussion

PSF presented with palpitations and it appeared as if there were several arrhythmias detected from his electrocardiograms (ECG), at first. However, with closer observation and scrutiny of the ECGs, the underlying arrhythmia is essentially atrial flutter-fibrillation.

With atrial flutter-fibrillation what happens is:

1. The heart rate becomes tachycardic (can be irregular or regular; as in flutter with 2:1 block)
2. Bombardment of the atrio-ventricular node with very frequent/irregular electrical impulses generated from the atria which is transmitted to the ventricles.

The ECG, where it seems as if he was having an episode of broad complex tachycardia (Figure 2), was actually atrial flutter with left bundle branch block. The explanation for this may be due to the very rapid heart rate and enhanced/accelerated conduction at the AVN, it reaches a point when the rate becomes too rapid, and the impulses cannot be conducted down one of the bundle branches (in this case it was the left bundle branch)^[1-3].

It is likely that as the impulse is being transmitted it encroaches on the refractory period of the left bundle branch. Thus, there appeared to be a broadening of the QRS complex (as if it was a BCT) or what is termed an aberration. This aberration may also sometimes result from concealed retrograde penetration (e.g. from a premature ventricular complex (PVC) originating in the left ventricle) into the left bundle branch making it refractory to oncoming beats/ impulses^[3-5].

In PSF, this lasted transiently (about 2 min) and the rhythm then spontaneously converted to atrial fibrillation (Figure 3). This would mean that the left bundle has recovered and overcome its refractoriness and conduction thus proceeded as usual. This refractory period is often noted to be short as the cycle length is also shortened, when a PVC is generated^[4-5].

There was no ablation done by the cardiologist in this case. PSF was commenced on an anti-arrhythmic drug and an antiplatelet to reduce the risk of clot formation. There are occasions when radio-frequency ablation can be used to interrupt the flutter circuit in the atrium, in patients with very frequent attacks or those not responding to pharmacological therapy.

Arrhythmias represent a common form of acute presentation seen in the ED. The exact diagnosis can sometimes be challenging and misdiagnosis of broad complex tachycardia is common. EPS does have a role to play in correctly identifying these rhythms, selecting the most appropriate therapy and deciding on the long term follow up and prognosis.

Conflict of interest statement

We declare that we have no conflict of interest

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