Atrial Flutter Mimicking Acute Myocardial Infarction*

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ABSTRACT

A case of ST-elevation myocardial infarction (STEMI) mimic produced by prominent atrial flutter waves is being presented and discussed.

A 37-year-old male came to the emergency room complaining of atypical chest pain of a few hours duration. Initial ECG displayed atrial flutter with 3:1 atrioventricular (AV) conduction with mostly pronounced inferior-wall but also diffuse ST-T changes suspect of acute ST elevation myocardial infarction (STEMI) (Figure, panel A) with already established inferior Q waves. However, due to non-convincing clinical history, further investigation was undertaken, which showed a normal bedside echocardiogram, while determination of cardiac enzymes confirmed normal values which remained normal at subsequent measurements.

At a repeat ECG (panel B), obtained a couple of hours later and displaying 4:1 AV conduction, one can discern more clearly that sized flutter waves distort the ST segment and produce the apparent ST elevation (even slightly more prominent now), while the QRS complexes do have small but discrete initial positive forces in the inferior leads, thus confirming the absence of q waves. Finally, the patient’s family fetched a

KEYWORDS: atrial flutter; acute myocardial infarction; STEMI; STEMI mimic; ECG

ABBREVIATIONS
ECG = electrocardiogram
STEMI = ST elevation myocardial infarction

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copy of a previous ECG (panel C) recorded 2 months earlier confirming that these ST-T wave changes were not new. Patient’s course was uneventful and he was discharged home the next day in stable clinical condition, with instructions focused on managing the arrhythmia.

Artifacts or Parkinson’s disease mimicking atrial flutter has been described. Rarely has atrial flutter been noted mimicking ischemic ST depression. However, atrial flutter masquerading ST elevation and acute myocardial infarction has not been previously reported. Indeed, atrial flutter has not been included in or considered as a STEMI mimic or confounder.

Of course during rapid heart rates, any tachycardia can produce ischemic or non-ischemic ST depression due to subendocardial ischemia, or occasionally ST elevation due to epicardial ischemia caused by the rapid rate in those with underlying ischemic heart disease. However, in the present case, the cardiac (ventricular) rate was not that rapid and the
big flutter waves were those interfering with the ST segment that produced the apparent but false ST-T changes masquerading as STEMI.

Atrial flutter is a macroreentrant, usually right atrial, tachycardia, whereby in its most common (~80%) and typical form, as in this case, the electrical impulse travels around the right atrium in a counterclockwise rotation within the arrhythmia circuit, ascending the interatrial septum and descending via the crista terminalis before it passes through the cavitricuspid isthmus producing the saw-toothed appearance of the flutter (F) waves in the inferior ECG leads (II, III, and aVF). When the same right atrial circuit is reversed (clockwise atrial flutter), predominantly positive F waves are seen in the inferior leads. In both cases this arrhythmia is amenable to catheter ablation of the cavitricuspid isthmus with a high success rate (~90%), and this was the treatment recommended to this patient.

Flutter (F) waves may be partly hidden in the early ST segment, and are indeed best seen in the inferior leads and lead V1. However, the free wall of the right atrium, whose electrical depolarization is best reflected on the body surface by lead V1, coarse atrial fibrillation may be misdiagnosed as atrial flutter in this lead, and review of the inferior leads may help discern the organized atrial activity in atrial flutter from the disorganized atrial depolarization of atrial fibrillation. On the other hand, these leads may help identify the flutter or fibrillation waves in patients presenting with tachycardia without discernible atrial waves in the other leads. Furthermore, one may have to resort to the “Lewis” leads (using the right and the left arm electrodes of lead I to explore the precordium) in an attempt to amplify and identify the atrial depolarization in order to differentiate a tachycardia.  

Apparently the prominent flutter waves distorted the ST segment in this particular patient and misled the physicians to initially consider an inferior STEMI. However, the lack of reciprocal ST changes (ST depression) in the precordial leads might have been a clue. The second ECG helped to exclude q waves, but produced some additional confusion showing more prominent ST elevation in the precordial leads (V2-V4). The third ECG confirmed that these changes preexisted, although one may still wonder whether there is an additional component of early repolarization changes in the precordial leads contributing to the appearance of a STEMI mimic.

REFERENCES