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Case Report

# Atrial Flutter Masquerading As Ventricular Tachycardia, A Case Report

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## Abstract

We describe a challenging case of atrial flutter presenting as wide-complex tachycardia suspicious for ventricular tachycardia and intractable acute congestive heart failure with features of cardiogenic shock; electrophysiology study proved to be helpful in reaching the diagnosis, ablation therapy was key in reversing the course of the disease.

## Introduction

### History of presentation

A 71-year-old male with past medical history of hypertension, dyslipidemia, diabetes, peripheral vascular disease, stroke, chronic kidney disease, coronary artery disease, and coronary artery bypass grafts surgery in April 2017. Patient was admitted to the hospital with chief complaints of nausea, vomiting, shortness of breath, and fatigue; these symptoms were intermittent but worsened over the preceding few days. Physical exam was remarkable for borderline blood pressure with systolic pressure in the 90s mmHg, he had distended jugular veins, bilateral lungs rales, ascites, and significant bilateral legs edema up to mid thighs levels with cold skin.

### Investigations

Chest-x-ray was consistent with pulmonary edema, laboratory work-up was significant for mild elevation of the liver enzymes and worsening kidneys functions. Baseline 12-leads EKG showed normal sinus rhythm with right bundle branch block and left anterior fascicular block (Figure 1). Echocardiography was significant for severe and global systolic left ventricular dysfunction with ejection fraction of 20%, which was a remarkably worse when compared with previous study that showed ejection fraction of 55% in Oct 2017.

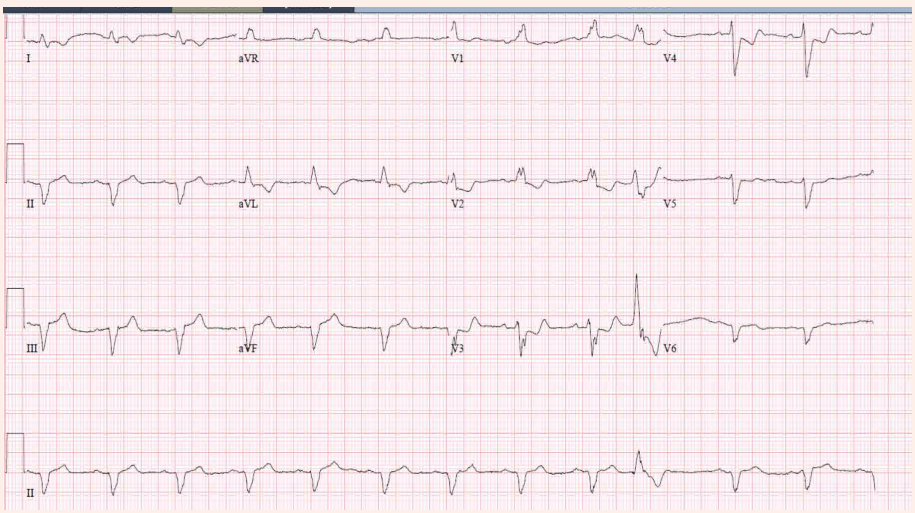


**Figure 1:** Baseline 12-leads EKG, right bundle branch block and left anterior fascicular block

### Differential diagnosis

Patient had evidence of biventricular congestive heart failure with significant decline in ejection fraction, his hospital course was remarkable for multiple episodes of wide-complex tachycardia (Figure 2); these required several cardioversions in view of hypotension with systolic blood pressure in the 80s and intractable heart failure signs and symptoms, diuretics were escalated and milrinone drip was added; patient also required BIPAP to support his oxygenation. Amiodarone drip was added, however patient continued to have episodes of wide-complex tachycardia. Subsequently, patient underwent emergent left heart catheterization which showed patent grafts and no evidence of new obstructive coronary artery lesions. The differential diagnosis of wide-complex tachycardia includes ventricular tachycardia or supraventricular tachycardia with preexisting or functional aberrancy or atrioventricular accessory pathways.

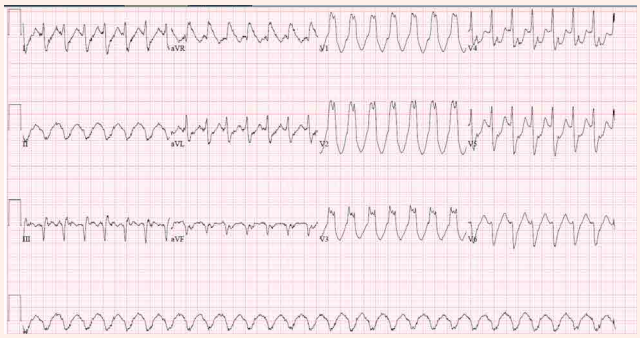

**Figure 2:** 12-leads EKG, wide-complex tachycardia

## Management

As patient failed to respond to medical treatment, decision was made to perform electrophysiology study to seek a definitive diagnosis and treatment. Pt was in the wide complex tachycardia at the time of the study, diagnostic catheters were positioned in the His area, the right ventricle and the coronary sinus, it was readily obvious that the patient was in supraventricular tachycardia with 2:1 block, and with proximal to distal coronary sinus activation (Figure 3), a multi-electrode diagnostic catheter (Pentaray) was used to perform activation mapping, map revealed typical counterclockwise cavotricuspid isthmus dependent flutter, surface EKG was identical to the clinical tachycardia, ablation line connecting the tricuspid valve and the eustachian valve terminated the tachycardia bidirectional block was verified using differential pacing, the tachycardia became noninducible. Patient made a steady and remarkable recovery, he remained in sinus rhythm with no recurrence of the wide complex tachycardia, he continued to diurese well, he was weaned off milrinone and BIPAP, his kidneys functions returned to baseline, a repeat echocardiography few days later showed normalized ejection fraction.

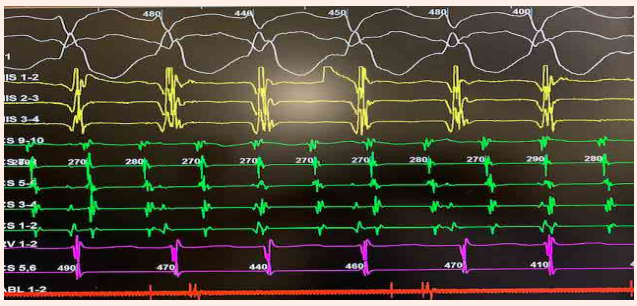

**Figure 3:** more atrial signals that are dissociated from the ventricular signals on coronary sinus channel in green

## Follow-up

Patient remained with no heart failure symptoms or signs, in normal sinus rhythm and with normal ejection fraction on his follow up visits in 3 and 6 months after his hospitalization.

## Discussion

The clinical wide-complex tachycardia which was documented several times by 12-leads EKG was very suggestive of ventricular tachycardia, in matter of fact, all "Wellens" criteria [1] were met; these include RBBB with QRS duration >140ms, "Rabbit ear" in V1 and RS<1 in V6; in addition to right superior axis and AV dissociation. The presence of more atrial signals that are dissociated from the ventricular signals (figure 3) was diagnostic for supraventricular tachycardia in our case. The QRS of the tachycardia has right bundle branch block morphology, similar but wider than baseline QRS, this is explained by progressive right bundle branch block which occur at faster rate [2]. This case is an example of the limitations of surface EKG driven criteria. When encountering wide-complex bradycardia, several causes must be considered; yet, the most critical task is to determine whether that tachycardia has a supraventricular or ventricular origin, as this will impact the immediate patient care decisions and the long-term management strategies [3] Electrophysiology study, while invasive, can be crucial in reaching the diagnosis.

While different types of arrythmias can be triggered by cardiomyopathy, persistent or high burden arrhythmias can lead to cardiomyopathy; it is difficult to determine whether the arrhythmia is the initiator or the consequence of the cardiomyopathy and frequently the arrythmia can be considered as the consequence of cardiomyopathy which can lead to loss of opportunity to deliver an effective therapy [4]. Having a high index of suspicion can be useful when dealing with these cases. QRS widening at faster heart rates is commonly related to phase-3 block in the His-Purkinje bundle, ventricular pacing, or ventricular tachycardia with isorhythmic dissociation. The variability in QRS morphology in these cases is usually explained by either progressive bundle branch block or fusion

## Conclusion

While different types of arrythmias including wide-complex tachycardias can be triggered by cardiomyopathy, persiste5nt or high burden arrhythmias can themselves lead to cardiomyopathy. Maintaining a high index of suspicion that the arrhythmia is the initiator culprit is very useful in dealing with these cases. Electrophysiology study can be crucial in reaching the diagnosis and delivering an effective therapy.

## Conflict of interest

The authors confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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