QUIZ

The ECG Quiz: "Similar but not the same!"

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History

An 80-year-old man presented to the ER with a history of palpitation for several hours. He had no other complaints. No more history was available.

His exam was unremarkable and his vital signs were stable.

His EKG on presentation is shown in Fig.1. His old EKG (baseline) is shown in Fig.2.

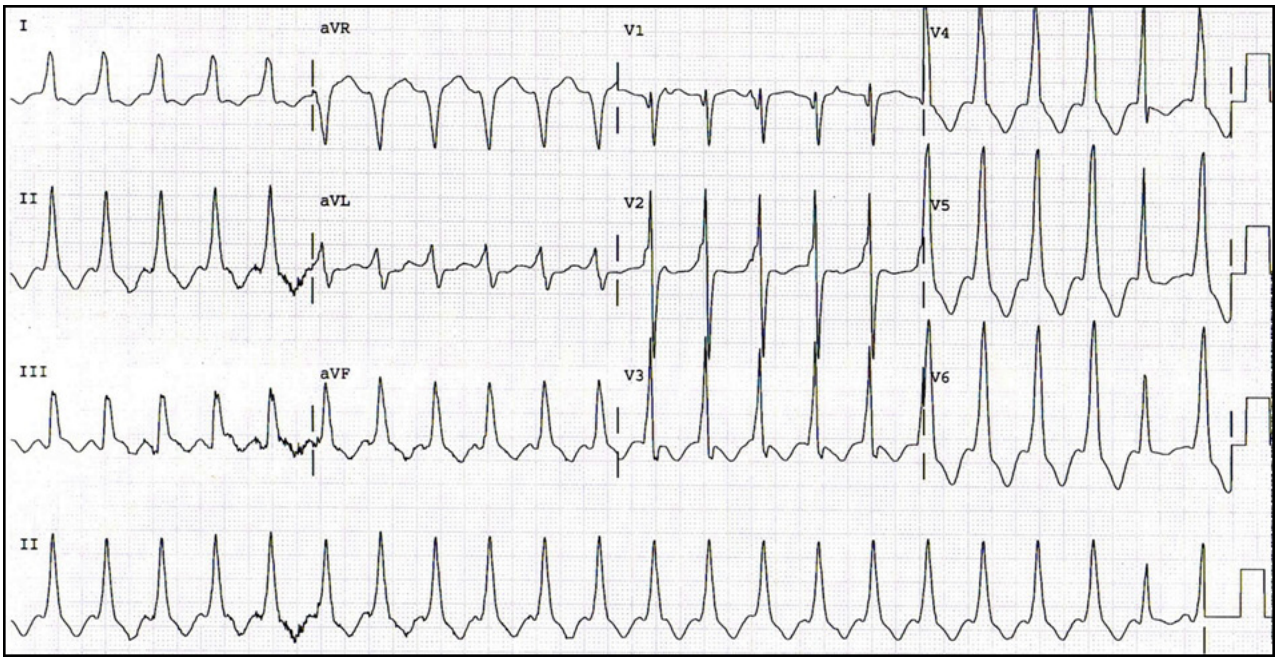

Figure 1: EKG on presentation to ER

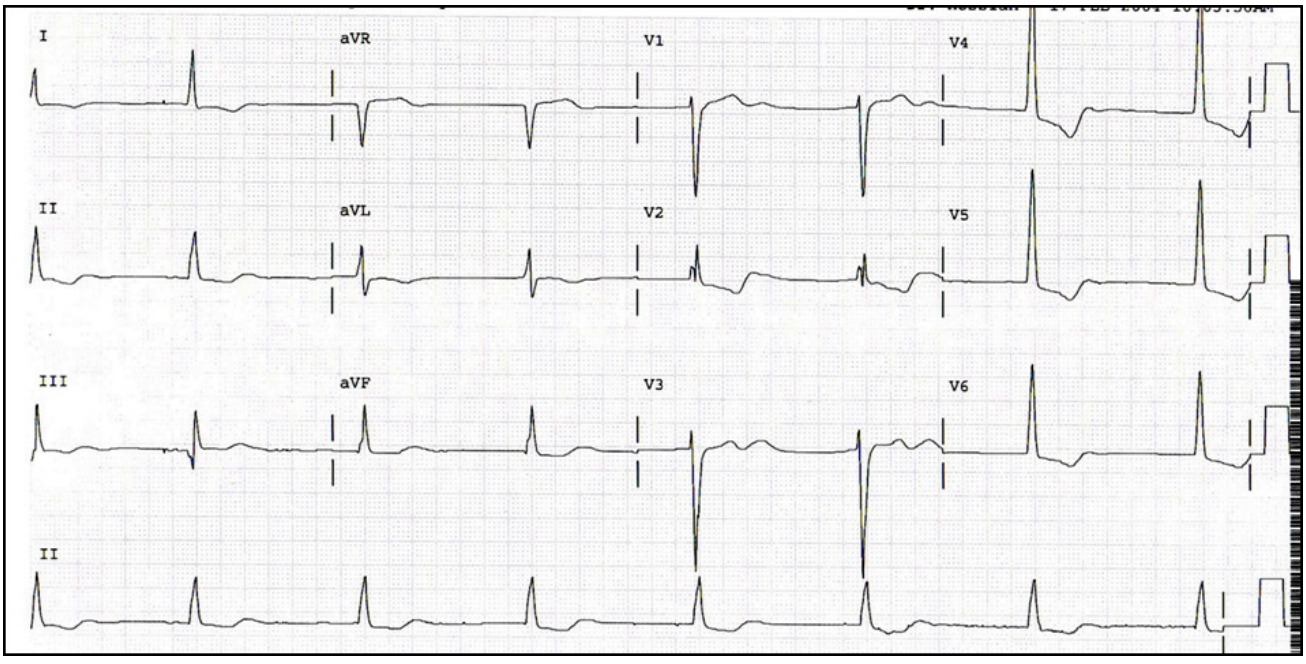

Figure 2: Baseline EKG.

Questions

1-What is the diagnosis of the rhythm shown on his EKG on presentation (Fig.1)?

a. Supraventricular tachycardia with underlying intraventricular conduction delay
b. Ventricular tachycardia (VT)
c. Supraventricular tachycardia with a pre-existing accessory pathway (WPW syndrome)
d. Atrial flutter with 2:1 AV conduction

2-What are the best short-term and long-term management strategies?

a. Break tachycardia with adenosine then consider a long-term suppressive therapy with either calcium channel blockers and/or beta-blockers for recurrent palpitations.
b. Cardioversion (chemical/electrical) then rule out structural heart disease and treat accordingly.
c. Break tachycardia (drugs/cardioversion) then consider ablation of an accessory pathway.
d. Heart rate control and/or cardioversion of atrial flutter then consider a long term Coumadin therapy if no contraindications.

Answers

1-b
2-b

Discussion

The presenting EKG shows a regular wide QRS tachycardia at a rate of about 130-140 bpm. The old EKG (assumingly his baseline EKG) shows a bradycardia with a heart rate of about 42 bmp. On that EKG, the atrial activity is not well-seen which raise the suspicion of a junctional rhythm, however, one may argue that the second beat was preceded by an atrial paced beat with a pacemaker spikes best seen in lead III and lead I, and possibly there was a P wave with a long PR interval preceding the third beat that is most visible in lead II at the bottom of the tracing. Regardless of what the atrial rhythm was, the QRS complexes are more relevant here and they appear to be slightly wide (about 120 ms) with increased voltages (especially chest leads) and marked repolarization changes. These QRS complexes have some similarity to the QRS complexes during tachycardia in most leads. This observation needs to be taken into consideration when interpreting his EKG on presentation.

So, what is the rhythm on his EKG on presentation?

The full discussion on the differential diagnosis of "wide QRS tachycardia" is beyond the scope of this case vignette, however, the main question needs to be addressed here is whether the rhythm on presentation was a ventricular tachycardia (VT) or a supraventricular arrhythmia on a background of his relatively wide QRS at baseline.

When looking for clues, one may note that the QRS complex during tachycardia was not very wide and the rate was not very fast. In addition, the patient was hemodynamically stable during the tachycardia. So, when put together, these findings, as well as the fact that the QRS on presentation has a close resemblance to his baseline QRS, will favor the diagnosis of a supraventricular arrhythmia. However, a careful look at his presenting EKG again (Fig.3) would reveal a clear evidence of atrial activities in lead V1 (blue arrows) that seemed regular, slower than (about 80 bpm), and unrelated to the underlying QRS complexes (AV dissociation). Moreover, on the same tracing, the beat before last (beat # 21 - brown arrow) appears to be narrower, and

looks more similar to the baseline EKG than the rest of the beats. The timing of this beat is consistent with that of the tachycardia (i.e., it is not premature), so it likely represents a "fusion beat" which is a hybrid beat due to two separate sources of ventricular activation; one of them is an ectopic ventricular rhythm (likely VT) and the other source is a descending sinus/supraventricular beat that was conducted to the ventricle. These findings (AV dissociation, and fusion beats) are very solid criteria to support the diagnosis of VT despite of all the above mentioned features that may favor the alternative diagnosis. Other alternative diagnoses given in the question are incorrect.

Although most classically VT is very wide (> 140 ms), fast, and hemodynamically unstable; it still can present with a totally opposite set of features. The QRS complex may not be very wide if, for example, the VT circuit happens to be close to and involves part of the His-Purkinje system, and hence part of the ventricle gets activated antegradely in a fashion similar to that during sinus rhythm. The rate of VT can be affected by several factors, and hence it can be relatively slow. Finally, VT can be well-tolerated and hence hemodynamically very stable especially if the heart rate is not very fast and the patient's ventricular function is reasonably preserved. Therefore, the width of the QRS complex, the rate of tachycardia, as well as the degree of clinical stability, can be misleading in making a final decision about the diagnosis of a wide QRS tachycardia. On the other hands, features like AV dissociation, fusion beats, and capture beats (a premature sinus beat that captures the ventricle in the midst of tachycardia) are virtually pathognomonic for VT and should be sought first.

In the given scenario the most appropriate immediate course of action is to achieve sinus rhythm. In a hemodynamically stable patient, chemical cardioversion should be considered first, however, if that fails, then synchronized cardioversion should be performed after adequate sedation is given. Once sinus rhythm is achieved, the patient needs to be evaluated for the presence of underlying structural heart disease; specifically coronary artery disease and/or cardiomyopathy. In addition precipitating factors (e.g., electrolyte disturbances, ischemia...etc) should be corrected if present. Long-term treatment depends upon the findings of the initial evaluation.

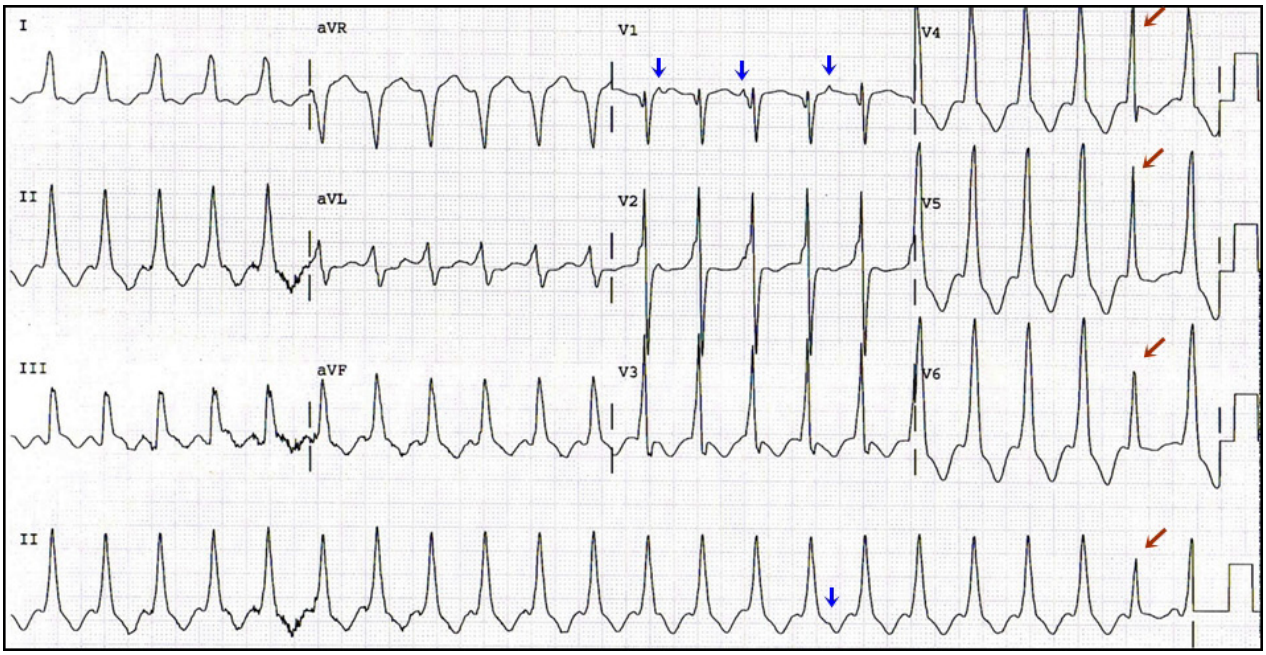

Figure 3: Evidence of AV dissociation. Note the atrial activities in lead V1 (blue arrows) and the fusion beat (brown arrow)

The take-home message: Look for more specific features first and try to resist the natural temptation of calling an arrhythmia "supraventricular" when the QRS complexes during the arrhythmia and baseline are similar; as similar is not necessarily the same.

Suggested Readings

1. Josephson ME. Clinical Cardiac Electrophysiology. 3rd ed. Philadelphia: Lippincott Williams & Wilkins; 2002.

2. Bogun F, Good E, Reich S, Elmouchi D, Igic P, Tschopp D, et al. Role of Purkinje Fibers in Post-Infarction Ventricular Tachycardia. Journal of the American College of Cardiology 2006;48(12):2500-7.
3. Guo H, Hecker S, Lévy S, and Olshansky B. Ventricular tachycardia with QRS configuration similar to that in sinus rhythm and a myocardial origin: differential diagnosis with bundle branch reentry. Europace 2001;3:115–23.