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Electrocardiogram Quiz – Case 4

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A 46-year-old woman presented to the emergency department of our hospital with sustained palpitation and tachycardia for the last two hours. She had had palpitations for the last couple of months that were spontaneously resolving and hence she never sought for medical advice. The patient's personal and family history was free of any heart disease and she did not take any medication. At the emergency department she was hemodynamically stable with a blood pressure of 140/90 mmHg. An electrocardiogram showed a regular narrow complex supraventricular tachycardia with a frequency of 170 beats/min (fig. 1).

Carotid sinus massage had no effect. An intravenous bolus of 6 mg of adenosine was administered with subsequent termination of the tachycardia; however, a pause of 3.5 seconds ended by a ventricular extrasystole gave its place to an irregular bradycardia with a ventricular rate of 50 beats/min (fig. 2).

Questions

a. What is the basic rhythm demonstrated at figure 2?

b. How can this be explained in terms of electrophysiology?

Comment

The configuration is suggestive of atrial fibrillation with a slow ventricular response.

Rapid intravenous injection of adenosine seems to be associated with an increased propensity towards atrial fibrillation. The mechanism by which adenosine induces atrial fibrillation remains to be clarified; however it is believed that at least two mechanisms may be solely or partially responsible. First, the induction and maintenance of atrial fibrillation are believed to require a critical number of wavelets and a critical wavelength. The wavelength is the product of the atrial refractory period and the conduction velocity. Shorter wavelengths are thought to promote the induction and maintenance of atrial fibrillation. Therefore, factors, like adenosine, that shorten refractory periods or slow conduction velocity favor the induction and maintenance of atrial fibrillation. A second potential mechanism is that adenosine administration results in frequent atrial premature complexes and therefore a long-short atrial sequence that induces atrial fibrillation.

There are relatively few studies trying to assess the electrophysiologic effects of adenosine in patients with supraventricular tachycardias. Moreover, there are scarce reports describing the induction of atrial fibrillation after the use of adenosine in the treatment of supraventricular tachycardias, either with subsequent presence of

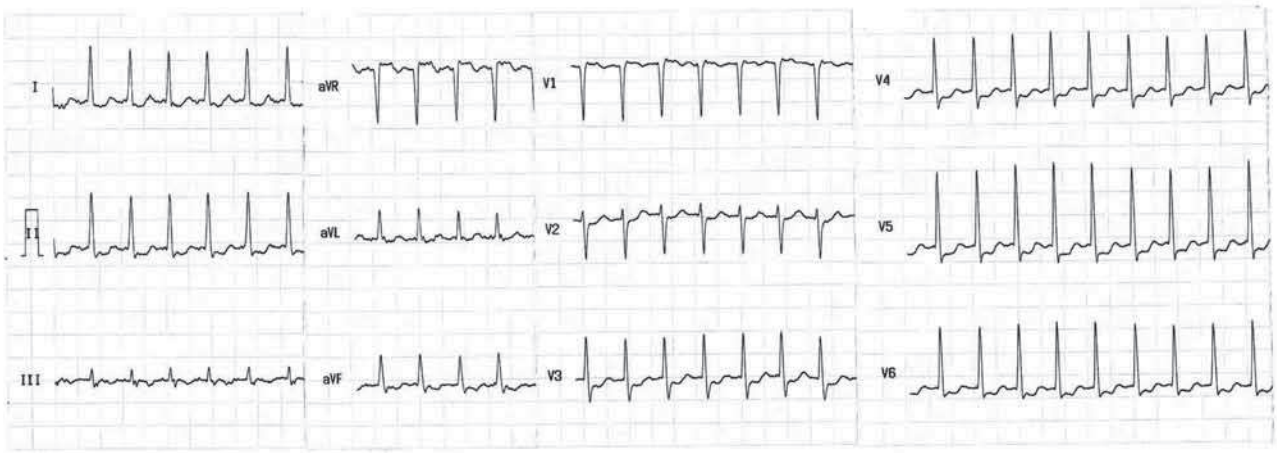

Figure 1

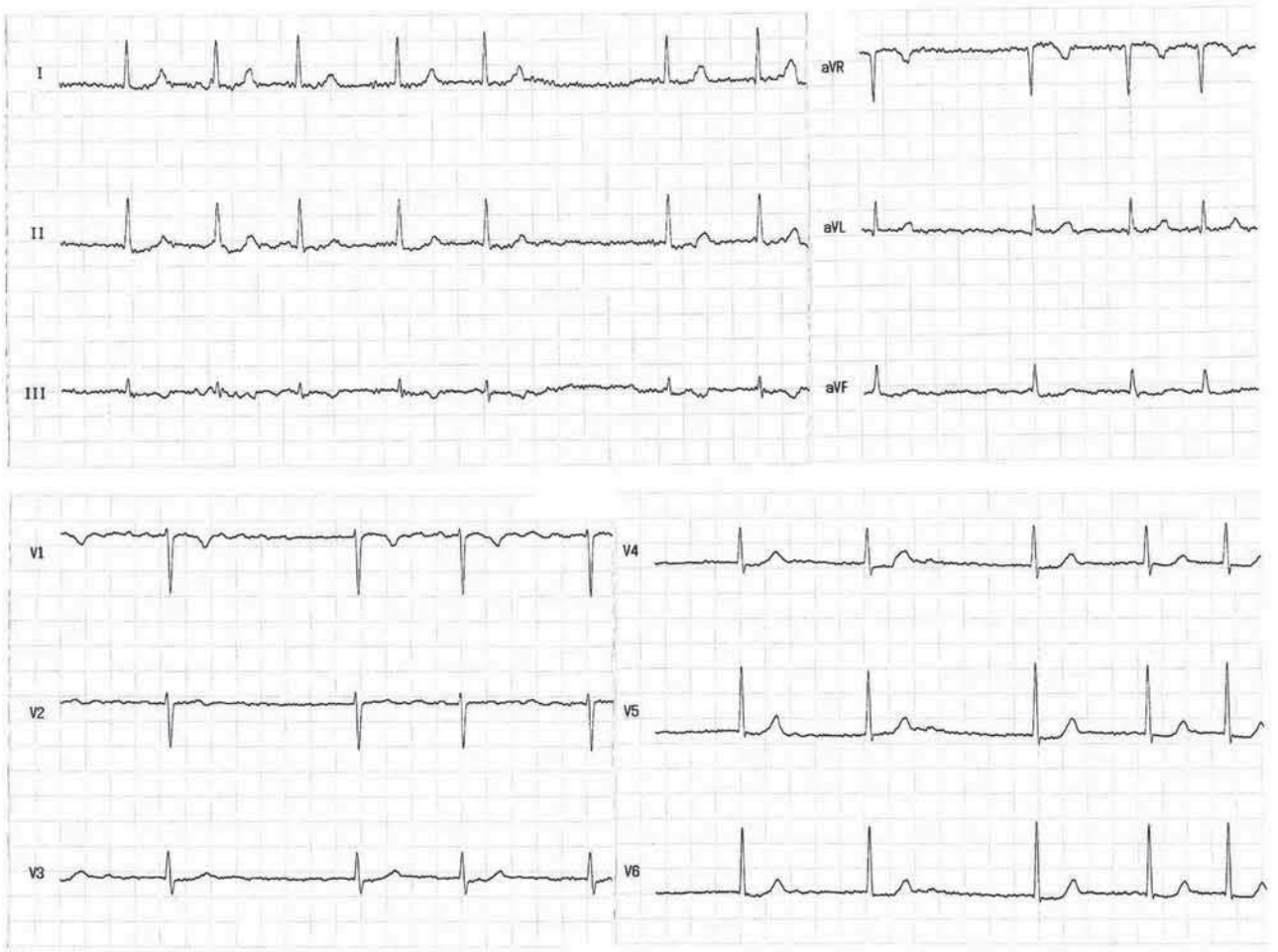

Figure 2

more severe arrhythmias, such as ventricular tachycardia, and hemodynamic deterioration or not. There are also reports of atrial flutter rate acceleration following adenosine administration. Furthermore, deaths of patients immediately after prehospital administration of adenosine for presumed supraventricular tachycardia have been reported. In other cases, the induction of atrial fibrillation unmasked accessory pathways and revealed pre-excitation syndromes.

In conclusion, the role of adenosine in the diagnosis and treatment of tachyarrhythmias is unparalleled; however additional caution should be exercised by every physician who is trying to terminate a tachyarrhythmia with adenosine, both in the hospital and the pre-hospital routine everyday practice.

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