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LETTER TO THE EDITOR
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Prolonged asystole following adenosine. Was it necessary?

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A 77-year-old woman presented with interchangeable episodes of both fast and slow heart rate. Past history was notable for paroxysmal atrial fibrillation treated with eliquis and tambocor and atrial septal defect closure 35 years ago. Except for mild tachycardia, vitals and physical examination were unremarkable. The 12-lead electrocardiogram (Figure 1) showed narrow complex tachycardia at 106 beats/min without obvious P-waves. Verapamil (5 mg) was given by the emergency medical services prior to her admission. Subsequently, two rounds of intravenous metoprolol (5 mg each) were given in our facility, failing to slow her heart rate. There was no documented attempt to prefrom vagal maneuvers. Within 30 min, the emergency ward physician injected a low-dose (6 mg) intravenous adenosine. Some seconds later, prolonged complete atrioventricular block (AVB) occurred (Figure 2). The patient loss consciousness and cardiopulmonary resuscitation was commenced with chest compression and bag mask ventilation. The were no ventricular escape beats. The effect of adenosine lasted 40 s until atrioventricular conduction gradually recovered, and she became alert. The drug unmasked (atrial flutter, AFL), identified by its characteristic sawtooth waveform with atrial rate of 220 beats/min. The positive deflections in inferior leads II, III and aVF suggest the atypical form, the cavotricuspid isthmus-independent AFL. The cardiac rhythm post-adenosine was AFL with variable conduction at 90 beats/min. She was admitted to the cardiology unit, where continuous monitoring revealed tachycardia-bradycardia episodes, thus permanent dual-chamber pacemaker was implanted. As she spontaneously converted to sinus rhythm, we opted for follow-up of the atypical AFL. She was discharged with cardiloc 2.5 mg twice a day and increased tambocor 100 mg twice a day. During 12-months of follow-up including pacemaker interrogation, except several atrial high rate episodes, she remained asymptomatic.

The use of adenosine injection is well-established in the management of narrow complex regular tachycardia, along with vagal maneuvers, it serves as a diagnostic adjunct in of uncertain origin. Adenosine may abruptly terminate the tachycardia, result in gradual slowing followed by reacceleration, or lead to transient high-grade AVB, thus unmasking the underlying atrial activity and facilitating the diagnosis. The effect is achieved by exerting a dose-related prolongation of the atrioventricular conduction.[1] Indeed, AVB following adenosine is common but normally brief. Among its various adverse effects, adenosine is known to suppress His-Purkinje, and may produce prolonged ventricular standstill.[2] Adenosine is commonly perceived as an otherwise benign drug with mild and short-lived adverse effects, including transient chest discomfort, flushing and dyspnea. Interestingly, up to 12% of patients develop atrial fibrillation following adenosine.[3] A small subset develop adenosine-induced short-lasting premature ventricular complexes and non-sustained ventricular tachycardia. In 2007, Harvey and colleagues reported on a patient with AFL treated with combination metoprolol and diltiazem, who developed prolonged complete AVB requiring intubation and temporary pacing following adenosine administration.[4] Adenosine is frequently applied as a diagnostic tool to confirm a diagnosis of AFL by unmasking flutter waves, however it is not without risk.[2] According to the latest European Society of Cardiology guidelines, adenosine administration is supported in cases of AFL only if deemed necessary for diagnosis.[3] In our case, we believe the syncope episode could have been avoided. First, referring to the anamnesis, she was taking tambocor, a class IC antiarrhythmic agent used to control atrial arrhythmias. Potential adverse effect is IC-induced AFL with 1:1 atrioventricular conduction, either typical or atypical morphology, particularly if given without concomitant atri-

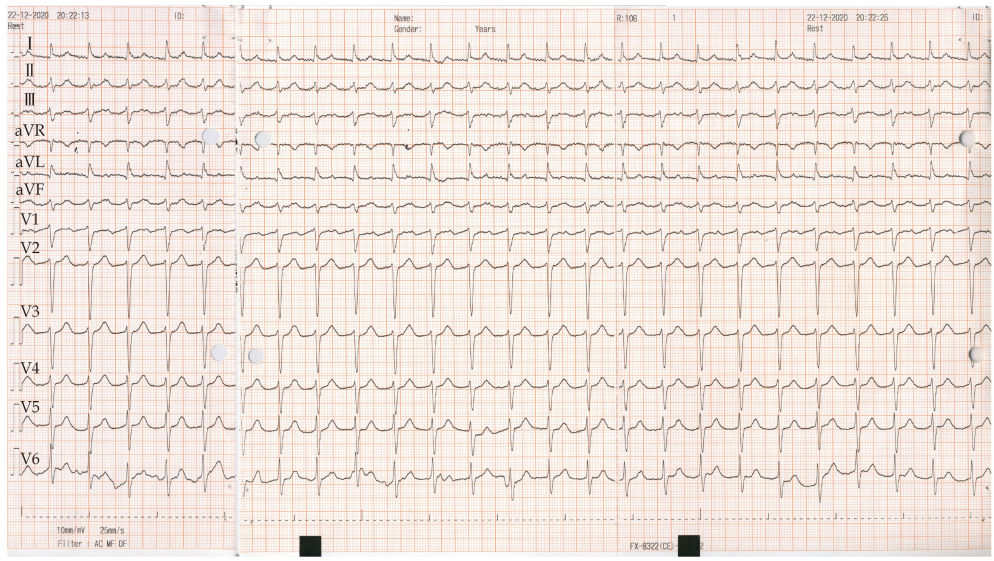

Figure 1 Electrocardiogram taken upon admission showing narrow complex tachycardia at 106 beats/min without obvious P-waves. There is a waving pattern without an isoelectric baseline, best appreciated in lead V1.

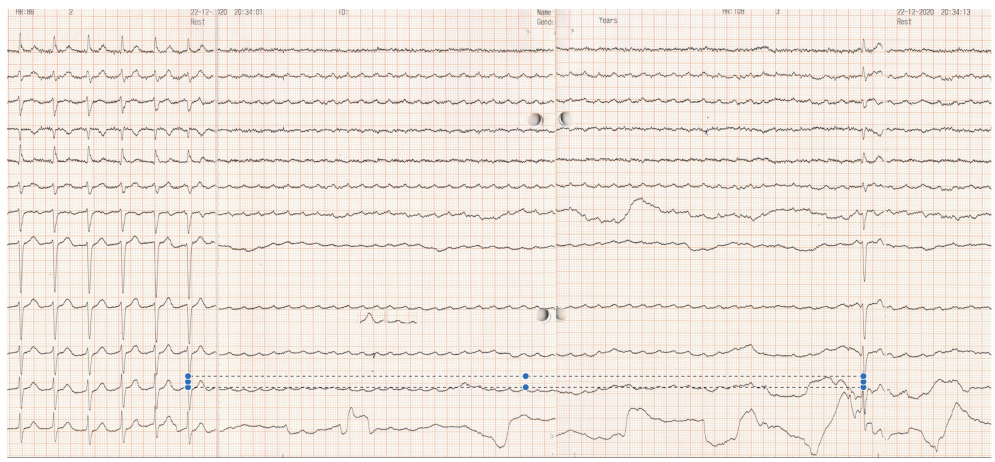

Figure 2 Electrocardiogram after administration of adenosine depicts prolonged complete atrioventricular block and ventricular standstill. The characteristic sawtooth waveform of atrial flutter is visible.

oventricular-blocking drugs,[4] as in our subject. Second, patients with underlying structural heart diseases as prior cardiac surgery or ablation procedures have a predisposition to develop atypical AFL, which unlike the more common typical AFL, is independent of the cavotricuspid isthmus. Atrial septal defect closure may result in focal tissue scaring and changes in left atrial geometry, serving as a substrate and facilitating the formation of focal macro-reentry.[3] In order to determine the exact location and mechanism of the arrhythmia electrophysiologic study is required. With that being said, a trained eye should have been able to suspect AFL based on the waving pattern without an isoelectric baseline, best appreciated in lead V1. Moreover, given the proximity of beta-blockers and calcium-channel blockers administration, adenosine should have been postponed until their effect subsided. These agents exert both negative chronotropic and dromotropic effect, suppressing the sinoatrial and atrioventricular node, respectively. Their effect possibly suppressed myocardial tissues and impaired the generation of escape rhythm, in addition to the suppression of the His-Purkinje system by adenosine.[2] There was no necessity in jeopardizing the patient, particularly considering her stable hemodynamics and suspected tachycardia-bradycardia syndrome.

Sinus node dysfunction (SND) is primarily an age-dependent pathology, resulting from degenerative changes at the level of the sinus nodal tissue.[5] SND can develop at any age, however it is more frequent among older individuals, especially those over 65 years old. The clinical ma-

nifestation of SND is diverse and may include; the "tachy-brady syndrome" which is identified by bradycardia alternating with paroxysmal supraventricular arrhythmias, sinus bradycardia, sinus pause or arrest, sinoatrial exit block, atrial fibrillation with slow ventricular response, and "chronotropic impotence", the failure to increase the heart rate in response to metabolic demands. A similar fibrotic degenerative process may involve the atrioventricular conduction system in these patients.[6]

In summary, we report an elderly women who developed prolonged AVB and ventricular standstill following adenosine injection. Our case highlights the importance of judicious use of adenosine. We argue that its use for diagnostic purpose in the above-described clinical setting was unnecessary, as AFL could have been identified by the combination of patient's history and presenting electrocardiogram. Extreme caution should be practiced when considering adenosine administration in cases of suspected of SND, particularly tachycardia-bradycardia syndrome who are treated with combination of beta-blockers and calcium-channel blockers.

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