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From Therapy to Trigger: Adenosine-Induced Polymorphic Ventricular Tachycardia During Treatment of Supraventricular Tachycardia

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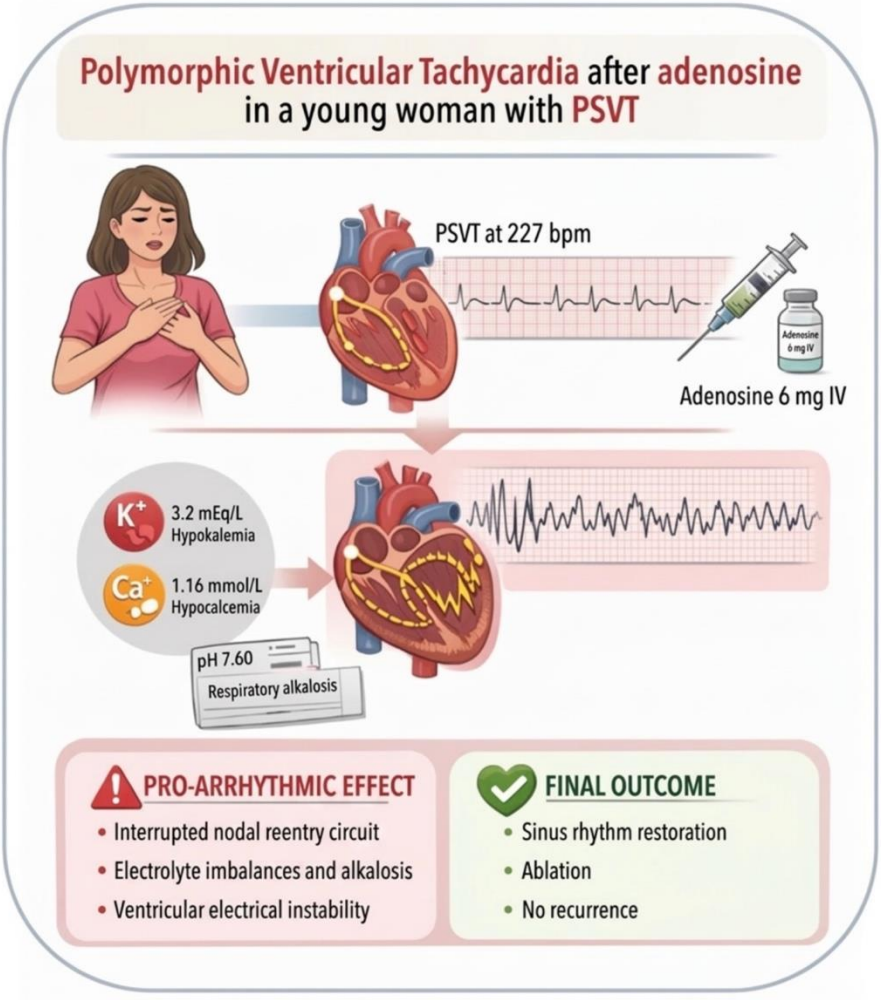
INTRODUCTION

Atrioventricular reciprocating tachyarrhythmias represent a constant challenge for emergency physicians, accounting for nearly 30% of arrhythmias observed in the emergency department [1]. In hemodynamically stable patients, the 2019 European Society of Cardiology (ESC) Guidelines recommend the use of vagal maneuvers followed, in case of failure, by intravenous administration of incremental boluses of adenosine (6 mg - 12 mg) [2].

From a pharmacodynamic perspective, adenosine is described as an endogenous nucleoside derived from Adenosine Triphosphate (ATP) acting primarily through activation of A1 receptors, widely expressed in the nodal conduction system (sinoatrial node - SAN- and atrioventricular node -AVN-). Interaction with these receptors leads to an abrupt reduction of the If current through cAMP depletion and an increase in potassium conductance, resulting in hyperpolarization of nodal cells generating negative chronotropic and dromotropic effects [3]. These properties explain its high efficacy in terminating Paroxysmal Supraventricular Tachycardia (PSVT).

Despite its rapid efficacy, extremely short half-life (approximately 10 seconds) and favorable safety profile, adenosine is not exempt from pro-arrhythmic effects, like all anti-arrhythmic drugs. In addition to the well-known induction of atrial fibrillation and the risk of degeneration into ventricular fibrillation in the presence of ventricular pre-excitation, cases of polymorphic ventricular tachycardia, including torsade's de pointes, have been reported in literature, even in the absence of structural heart disease [4-7]. These unintended clinical observations have led to the description of the bifaceted electrophysiological behavior of adenosine, effectively referred to as "Janus face", reflecting its capacity to exert both antiarrhythmic and, under unfavorable precipitating conditions, proarrhythmic effects [8].

Given the widespread use of a drug that is both complex and delicate from an electrophysiological standpoint, we present the case of a young woman with PSVT that degenerated into non-sustained polymorphic ventricular tachycardia immediately after adenosine administration (Central Figure).



Central Figure

CASE REPORT

A 28 years old young woman presented to the Emergency Department (ED) of our institution (Policlinico Umberto I, Rome, Italy) via the territorial emergency medical service for sudden-onset palpitations associated with chest tightness, which had begun approximately 20 minutes earlier. The Electrocardiogram (ECG) recorded in the ambulance documented a regular narrow-QRS tachyarrhythmia at 207 bpm with an RP interval < 70 ms (Figure 1a). On arrival, the patient was alert, oriented, hemodynamically stable and mildly tachypneic. The ECG obtained in the ED confirmed these findings and was strongly suggestive of Atrioventricular Nodal Reentrant Tachycardia (AVNRT) (Figure 1a).

The patient's medical history was notable for similar self-limiting episodes. She denied allergies, medication use, substance abuse or relevant comorbidities. Arterial blood gas analysis revealed respiratory alkalosis (pH 7.60, pCO2 18 mmHg) with metabolic compensation (HCO3- 17.7 mEq/L, BE -3.9), mild hyperlactatemia (2.4 mmol/L), mild hypokalemia (3.2 mEq/L) and mild hypocalcemia (1.16 mmol/L); respiratory parameters were otherwise within normal limits.

Following failure of vagal maneuvers (carotid sinus massage performed under continuous monitoring), as recommended by ESC guidelines, a 6 mg intravenous bolus of adenosine was administered. Immediately after infusion, a polymorphic wide-complex tachycardia occurred (Figure 1b), which spontaneously terminated within 18 seconds. Throughout the episode, the patient remained conscious and hemodynamically stable. The arrhythmia was followed by a brief sinus pause and restoration of sinus rhythm at approximately 100 bpm, with sporadic ventricular extrasystoles and subsequent stabilization (Figure 1b). Shortly thereafter, the patient reported complete symptom relief.

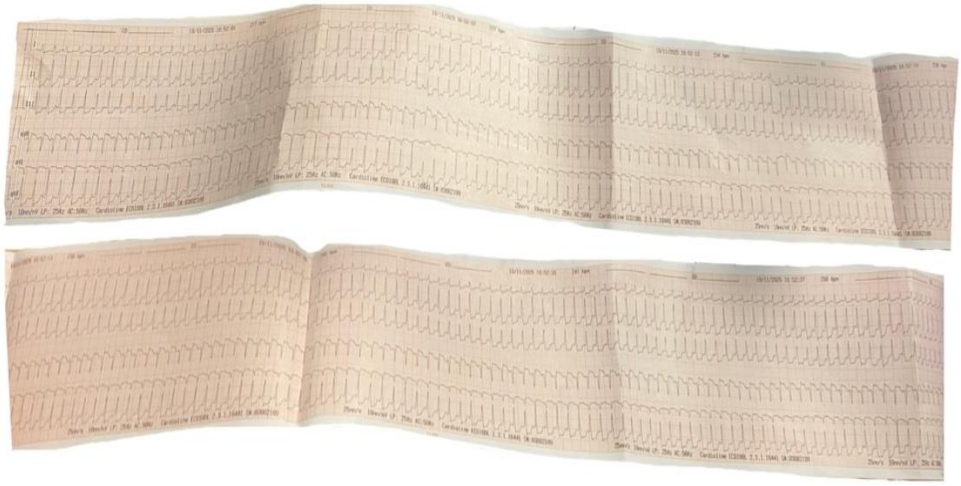


Figure 1a: ECG recorded in the ED at baseline and during carotid sinus massage.

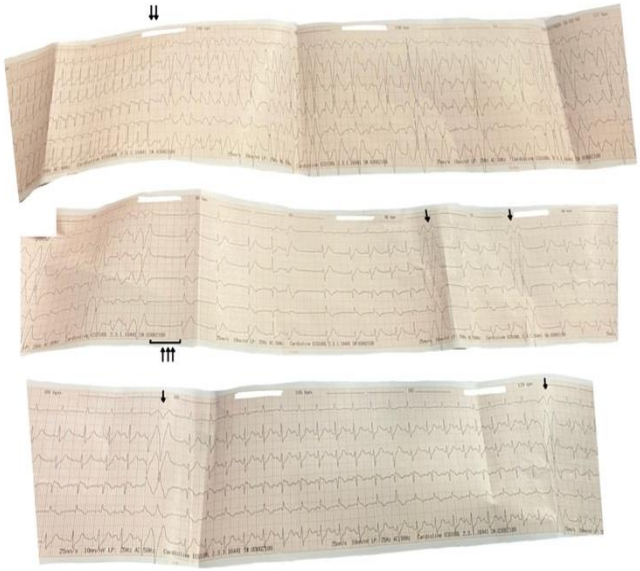


Figure 1b: ECG tracing recorded during continuous monitoring. The two arrows indicate the moment of adenosine administration and the simultaneous onset of polymorphic wide-complex ventricular tachycardia. The three arrows indicate the brief pause following the spontaneous termination of ventricular tachycardia. Single arrows indicate sporadic ventricular premature beats after resolution of torsade's de pointes.

The patient was then transferred to the Cardiac Intensive Care Unit (CICU) for further evaluation. Electrolyte abnormalities were corrected and transthoracic echocardiography showed no evidence of structural heart disease. Clinical, laboratory and telemetry follow-up in the following days did not document any significant anomalies.

Considering the presenting supraventricular tachyarrhythmia, the patient was referred for an invasive Electrophysiological Study (EPS) with planned catheter ablation. During EPS, ventricular incremental pacing demonstrated concentric decremental retrograde conduction, supporting the reasonable exclusion of Atrioventricular Reentrant Tachycardia mediated by an accessory pathway (AVRT). During programmed atrial stimulation with double extrastimuli (S1 500 ms, S2 400 ms, S3 380 ms), an anterograde "jump" was observed, associated with induction of a narrow-QRS tachycardia at 210 bpm recognized by the patient as identical to the arrhythmia that prompted hospitalization. These findings further supported the clinical suspicion of AVNRT. Programmed ventricular stimulation with double extrastimuli up to refractoriness, both under baseline condition and during isoproterenol infusion, did not induce ventricular tachyarrhythmias.

An electroanatomical mapping of the triangle of Koch was then performed using the CARTO system, documenting slow-pathway potentials at the base of the triangle. Three radiofrequency applications (45 W, 50°C) were delivered using an irrigated SmartTouch SF catheter, producing multiple retrogradely conducted junctional beats. Repeat EPS approximately 20 minutes after the last application confirmed elimination of nodal duality and non-inducibility of the arrhythmia, both at baseline and during isoproterenol infusion.

The patient was discharged the following day in stable hemodynamic condition and good general health. At 3-month follow-up, she remained asymptomatic, with no recurrence of arrhythmia and no impairment in quality of life.

METHODS

To contextualize the clinical case and explore its clinical and physio pathological implications, a systematic review of literature was conducted on the PubMed/MEDLINE, Embase and Cochrane databases, using combinations of the following keyword, indexed as "Title/Abstract" and/or "MeSH Terms": "adenosine", "supraventricular tachycardia", "ventricular tachycardia", "torsade's de pointes", "proarrhythmic effects", "accessory pathway", "electrolyte imbalance". The search was conducted through December 2025.

Original articles, case reports, case series, systematic reviews and meta-analyses published in English were included if they reported proarrhythmic events, especially ventricular, associated with adenosine administration in patients with or without structural heart disease. Studies without an abstract, non-English publications, and preclinical studies were excluded.

Study screening and selection were independently performed by two reviewers (B.M. and T.R.), with discrepancies resolved by consensus. For each study, data regarding patient clinical characteristics, the type and duration of induced ventricular arrhythmia, the presence of predisposing factors (accessory pathway, long QT, electrolyte imbalance), and clinical outcomes were subsequently extracted.

Given the heterogeneity of study designs and the predominance of case reports, the methodological quality of observational studies was assessed descriptively. Findings were critically integrated into the discussion. The PRISMA flow diagram of the systematic search is shown in Figure 2.

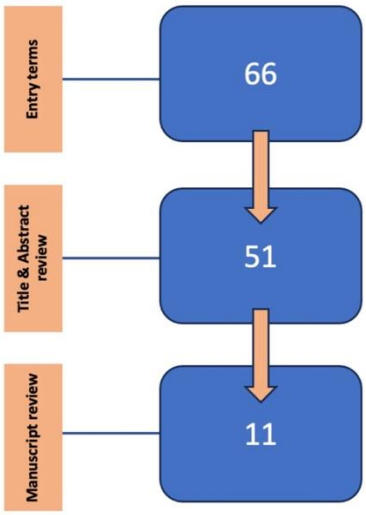


Figure 2: PRISMA flow diagram of the literature search.

DISCUSSION

Adenosine is the first-line therapy for hemodynamically stable PSVT, with reported success rates exceeding 85% [2,9]. The most common adverse effects - flushing, chest discomfort, nausea - are generally transient and benign, although unpleasant for the patient. Nevertheless, as with all antiarrhythmic agents, adenosine has proarrhythmic effects, including ventricular tachyarrhythmias, which are rare but documented in literature [4,6,10].

A systematic review by Feng and colleagues highlighted that most adenosine-induced ventricular tachycardia episodes are non-sustained and self-limiting [9]. However, cases of torsade's de pointes and degeneration into ventricular fibrillation, even in structurally normal hearts, have been reported [5,7,11,12]. In the present case, the close temporal relationship between adenosine administration and the onset of polymorphic ventricular tachycardia strongly suggests a direct causal role.

From a pathophysiological standpoint, several mechanisms may have contributed to the arrhythmic degeneration. Adenosine primarily induces transient NAV block without significantly interfering with conduction through potential accessory pathways, underscoring the need for caution when administering this pharmacologic agent if ventricular pre-excitation is suspected. In our patient, subsequent EPS excluded the presence of accessory pathways with sodium-dependent electrophysiological properties. Sudden interruption of the tachycardia circuit can, however, accentuate local conduction anisotropy in the presence of a diseased substrate and, less commonly, trigger arrhythmias *via* autonomic imbalance in structurally normal hearts.

The concept of adenosine's "Janus face" captures this duality: antiarrhythmic at the nodal level but potentially proarrhythmic at a ventricular level when a predisposing substrate or trigger is present [8]. In our case, electrolyte disturbances and respiratory alkalosis may have predisposed to arrhythmic degeneration. These conditions are, in fact, known to promote QT interval prolongation and early depolarizations, key mechanisms underlying ventricular arrhythmias such as torsade's de pointes. Additionally, recent data suggest how ionized calcium alteration could be identified as an independent determinant of ventricular electric instability in acute arrhythmic contexts [11]. In this regard it is interesting to note that non-dihydropyridine calcium channel

blockers may constitute an alternative therapy and have a comparable efficacy to adenosine even though they present a different safety profile, as reported in Feng et al.'s meta-analysis [9]. Nevertheless, adenosine retains as of today a clear advantage in terms of pharmacokinetics, rapidity, and predictability, which supports its continued recommendation as first-line therapy [2].

This case does not intend to challenge the central role of adenosine in the management of hemodynamically stable SVT, but to emphasize the importance of an integrated clinical approach and assessment and to the need for it to be administered in a controlled setting, with continuous ECG monitoring and immediate defibrillation availability, especially when an accessory pathway is suspected. In case of significant electrolyte disturbances, it is prudent and reasonable to correct modifiable abnormalities - when feasible - prior to adenosine administration to reduce risk of ventricular arrhythmic degeneration.

CONCLUSION

Adenosine-induced polymorphic ventricular tachycardia in structurally normal hearts is a rare but recognized event. The presence of an accessory pathway and electrolyte disturbances may act as a facilitating substrate. Awareness of the potential "Janus face" of adenosine should encourage the clinician to maintain a high level of vigilance during its administration, without diminishing its established role as a first-line therapeutic cornerstone in the management of hemodynamically stable PSVT.

ACKNOWLEDGEMENT

Patient consent statement

Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

IRB approval statement

Ethical approval was not required for this study in accordance with local regulations for single case reports.

Declaration of generative AI use

During the preparation of this work the authors used the artificial intelligence software ChatGPT 5.0 as an aid in the creation of the image presented as the Central Figure. After using this tool, the authors reviewed the content and take full responsibility for the content of the published article.

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