

A Case Report: Supraventricular Tachycardia Presenting As Atypical Abdominal Pain Following Exercise in A 72-Year-Old Diabetic Woman

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Abstract

Supraventricular tachycardia (SVT) is a common form of arrhythmia that typically presents with palpitations, dizziness, and chest discomfort. While these classic symptoms are well-recognized, atypical presentations such as abdominal pain are distinctly uncommon and can lead to diagnostic delays, particularly in elderly patients with comorbidities. We report the case of a 72-year-old woman with a history of type 2 diabetes mellitus who presented with an acute onset of sharp abdominal pain radiating to the back following physical exercise. Upon arrival, she was hemodynamically stable. An electrocardiogram revealed supraventricular tachycardia with an approximate rate of 170 beats per minute. Vagal maneuvers were attempted but failed to terminate the arrhythmia. Due to the unavailability of adenosine, the patient was treated with intravenous amiodarone, which successfully converted the rhythm to a normal sinus rhythm. She was admitted for monitoring and discharged on hospital day 3 in stable condition with oral bisoprolol maintenance therapy.

Introduction

Supraventricular tachycardia (SVT) is a broad category of arrhythmias originating at or above the atrioventricular node. The paroxysmal form (PSVT) is clinically characterized by an abrupt onset and termination, with heart rates typically ranging from 140 to 250 beats per minute. These arrhythmias usually present with narrow QRS complexes, unless aberrant conduction is present. Pathophysiologically, the majority of cases are driven by re-entry circuits involving the atrioventricular (AV) node, the atrium, or accessory pathways. In contrast, mechanisms involving enhanced automaticity or triggered activity are less common [1,2,3].

While SVT is frequently diagnosed in young adults, its incidence and clinical significance increase with age [1,3]. In the elderly population, physiological changes and comorbidities can complicate both the clinical presentation and the hemodynamic tolerance of the arrhythmia [1,2]. Classically, patients with SVT present with palpitations, lightheadedness, chest discomfort, or anxiety [1,4].

However, atypical presentations are not uncommon, particularly in patients with type 2 diabetes mellitus (T2DM). In these patients, autonomic neuropathy may alter visceral pain perception [5]. Furthermore, because cardiac and abdominal organs share overlapping sensory nerve pathways (visceral afferent convergence), cardiac arrhythmias can occasionally manifest as "angina equivalents," such as epigastric or abdominal pain. This can lead to misdiagnosis as gastrointestinal or acute surgical pathology [6].

Immediate management of stable SVT typically involves vagal maneuvers followed by intravenous adenosine as the first-line pharmacotherapy [1,3,7]. However, in resource-limited clinical settings where adenosine is unavailable, alternative antiarrhythmics like calcium channel blockers (diltiazem and verapamil) and beta-blockers (metoprolol) can be used [1,8]. If not available, alternative agents, such as amiodarone, may be considered [1,9].

This report presents the case of a 72-year-old diabetic woman who developed exercise-induced SVT presenting with the atypical symptom of acute abdominal pain radiating to the back. We report on the challenge of diagnosing atypical arrhythmias in the elderly and their treatment with amiodarone in the absence of adenosine.

Case Presentation

A 72-year-old female presented to a district hospital at approximately 08:45 AM with an acute onset of sharp abdominal pain radiating to her back, accompanied by palpitations and shortness of breath. The symptoms began approximately two hours prior, following 20 minutes of moderate exercise on a bicycle. The pain was constant and was not relieved by one hour of rest or by changes in position. Although the patient had a history of gastritis, she reported that the characteristics of this pain were distinctly different from her usual symptoms. She denied any prior history of chest pain, palpitations, or any episode of syncope.

The patient had a history of type 2 diabetes mellitus and routinely took glibenclamide. Her last visit to the doctor for diabetes was 6 months prior; she continued taking the drug but could not recall her last HbA1c or fasting blood glucose results. She had no history of hypertension, arrhythmia, or cardiac, lung, or kidney disease. The patient had a family history of hypertension but no family history of arrhythmia, sudden cardiac death, or ischemic heart disease. The patient had never smoked and did not consume alcohol. She had stopped consuming coffee around 20 years ago and denied taking over-the-counter supplements, herbal medicine, or narcotic drugs. She reported no known allergies.

The patient's vital signs were as follows: blood pressure of 100/70 mmHg, heart rate of 168 beats per minute, respiratory rate of 23 breaths per minute, and oxygen saturation (SpO₂) of 93% on room air. The patient was alert with a Glasgow Coma Scale (GCS) score of 15 (E4V5M6) and a numerical pain scale rating of 6/10.

On physical examination, the patient appeared anxious. Her cardiovascular examination revealed a regular tachycardia with normal S1 and S2 sounds and no murmurs, rubs, or gallops. Jugular venous pressure was normal at 5 + 2 cm H₂O when the patient was positioned at a 45-degree angle. Her lungs were clear to auscultation. The abdomen was soft, non-distended, and non-tender to palpation throughout. There was no peripheral edema.

Based on the vital signs, oxygen therapy was initiated via nasal cannula at 3 liters per minute, followed immediately by an electrocardiogram (ECG). The ECG captured a narrow complex regular tachycardia without visible P waves at approximately 170 beats per minute (Figure 1). The patient had a normal cardiac axis and a narrow QRS complex. The clinical presentation and ECG findings showed SVT typically consistent with atrioventricular nodal reentrant tachycardia (AVNRT), although definitive confirmation requires electrophysiological study.

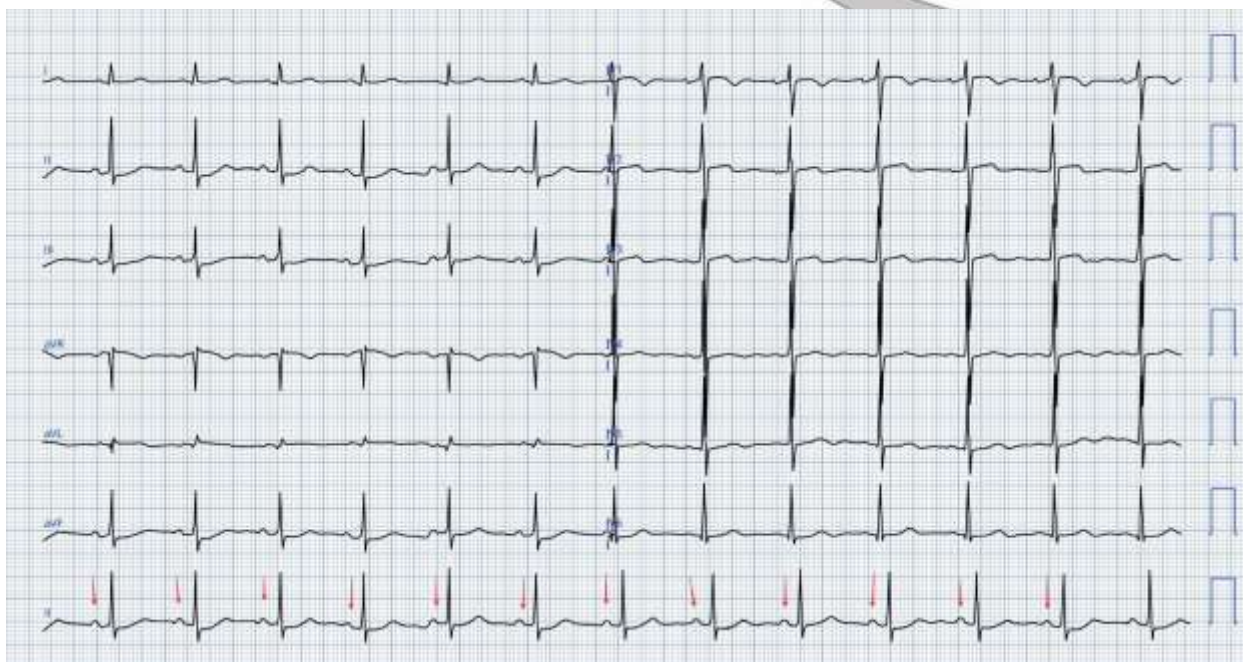


Figure 1: The initial ECG reveals a narrow complex tachycardia with absent P waves

The red arrows indicate a typical SVT pattern, characterized by the absence of P waves and regular tachycardia. Some of the beats appeared irregular due to motion artifact from the patient moving during the electrocardiogram.

The patient underwent a vagal maneuver with a standard Valsalva maneuver. This did not result in a change in heart rate, and the SVT did not convert to a normal sinus rhythm. Drug therapy was initiated. Because adenosine and verapamil were not available at the hospital, the patient received amiodarone 150 mg administered intravenously (IV) over 10 minutes.

Vital signs and an ECG were repeated at approximately 09:20 AM. The patient's follow-up vital signs were as follows: blood pressure of 100/60 mmHg, heart rate of 80 beats per minute, respiratory rate of 20 breaths per minute, and oxygen saturation (SpO₂) of 97% on 3 L/min of oxygen via nasal cannula. Her heart sounds (S1 and S2) were normal. The patient's complaints of palpitations and abdominal pain radiating to the back had resolved. Her repeat ECG showed a normal sinus rhythm with a normal cardiac axis, with each P wave followed by a narrow QRS complex (Figure 2). Notably, no Q waves, ST elevation, ST depression, or T-wave inversions were detected.



At approximately 10:30 AM, the patient's laboratory examination results were as follows: Complete blood count (CBC) showed a hemoglobin level of 14.8 g/dL (normal value: 12 - 16 g/dL), hematocrit of 45.1% (normal value: 38 - 42%), white blood cell count of $10.17 \times 10^9/L$ (normal value: $4.7 - 11.3 \times 10^9/L$), and platelets of $257 \times 10^9/L$ (normal value: $150 - 450 \times 10^9/L$).

Cardiac marker examination showed a troponin I level of 4 ng/L (normal value: <29 ng/L). Fasting blood glucose was 215 mg/dL (normal value: <140 mg/dL). Her serum creatinine was 0.59 mg/dL (normal value: 0.6 - 1.1 mg/dL), BUN was 10.9 mg/dL (normal value: 10 - 20 mg/dL), AST was 13 U/L (normal value: 8 - 31 U/L), and ALT was 12 U/L (normal value: 6 - 31 U/L).

Electrolyte levels were within normal limits: Sodium was 137 mmol/L (normal value: 136 - 145 mmol/L), Potassium was 4.1 mmol/L (normal value: 3.5 - 5.1 mmol/L), and Chloride was 102 mmol/L (normal value: 97 - 111 mmol/L).

A posteroanterior (PA) chest X-ray was performed to assess the patient's symptoms. The chest X-ray demonstrated a normal cardiac silhouette size and contour. The lung fields were clear, with no evidence of pulmonary edema, infiltrates, or consolidation. No bony abnormalities were noted. The overall chest X-ray findings were unremarkable (Figure 3).



Figure 3: The posteroanterior chest X-ray image of the patient

After the SVT converted to a normal sinus rhythm, the patient was hospitalized in the Intensive Cardiac Care Unit (ICCU) for close monitoring. She received a therapeutic dosage of amiodarone 300 mg per day orally. Supportive therapies consisted of intravenous hydration with 1000 mL of normal saline every 24 hours, intravenous pantoprazole once daily, and diazepam 2 mg twice daily.

The patient was later discharged on the third day in stable condition with normal vital signs and no complaints of palpitations or arrhythmia. Upon discharge, the amiodarone and other inpatient medications were discontinued, and she was transitioned to oral bisoprolol 5 mg once daily for long-term maintenance therapy. The patient was scheduled for echocardiography at the follow-up outpatient appointment.

Discussion

Supraventricular tachycardia (SVT) is a general term describing a group of arrhythmias that originate at or above the atrioventricular node. Paroxysmal supraventricular tachycardia (PSVT) is clinically characterized by a sudden onset and termination, with atrial rates typically ranging between 140 and 250 beats per minute and narrow QRS complexes, unless aberrant conduction is present. Pathophysiologically, PSVTs are most commonly driven by re-entry circuits involving the AV node, atrium, or accessory pathways, rather than mechanisms of enhanced automaticity. In the case of atrioventricular nodal reentrant tachycardia (AVNRT), the AV node functionally dissociates into two pathways: a "fast" pathway with a long refractory period and a "slow" pathway with a shorter refractory period. A premature beat can block the fast pathway and travel down the slow one, initiating a re-entry

loop [1,2,3]. This case report describes a 72-year-old woman presenting with SVT precipitated by moderate exercise, characterized by an atypical presentation of acute abdominal pain radiating to the back.

The classic clinical presentation of SVT typically involves palpitations, dizziness, chest discomfort, and dyspnea [1,4]. While SVT is frequently diagnosed in young adults, its incidence and clinical significance increase with age [1,3].

The classic clinical presentation of cardiac ischemia or severe arrhythmia typically involves discomfort referred to the T1 through T5 dermatomes, affecting the chest and medial aspect of the arms. These symptoms are usually rapid in onset, crescendo briskly, and may leave the patient with a profound "feeling of doom" that does not wane with rest [3,4]. However, atypical presentations can occur. In this patient, the chief complaint was sharp abdominal pain radiating to the back, mimicking gastrointestinal or aortic pathology. The mechanism of this referred pain involves visceral afferent convergence. Cardiac sensory fibers typically enter the spinal cord at the T1 to T5 thoracic levels. However, these visceral pathways frequently overlap with somatic and visceral fibers from the upper abdomen, which generally enter the spinal cord between the T6 and T9 levels [6].

Autonomic neuropathy in a patient with type 2 diabetes mellitus (T2DM) can alter pain perception, causing patients to experience "angina equivalents" such as dyspnea, nausea, or epigastric/abdominal pain rather than classic retrosternal chest pressure [4,5].

The precipitation of SVT in this case was linked to physical exertion. Exercise induces a sympathetic surge and increases catecholamine levels, which can enhance automaticity or trigger re-entry circuits in susceptible individuals [2]. While sinus tachycardia is the most common physiological response to exercise, the sudden onset of symptoms and the heart rate exceeding the patient's maximum predicted heart rate raised the suspicion of a pathological arrhythmia [1,2].

The initial ECG captured a narrow complex tachycardia without visible P waves. The clinical presentation and ECG findings showed SVT typically consistent with AVNRT, although definitive confirmation would require an electrophysiological study. In AVNRT, retrograde atrial depolarization occurs simultaneously with ventricular depolarization, causing the P waves to be buried within the QRS complexes and rendering them invisible on the surface ECG [3].

Management of acute SVT in stable patients begins with vagal maneuvers. The Valsalva maneuver is the standard first-line intervention, though its success rate varies between 17% and 43% [1,7]. In this case, the vagal maneuver failed, necessitating pharmacological cardioversion. Current guidelines recommend adenosine as the first-line drug of choice due to its rapid onset and short half-life [1,3,7]. However, in resource-limited settings where adenosine is unavailable, alternative antiarrhythmics like calcium channel blockers (diltiazem and verapamil) or beta-blockers (metoprolol) can be considered [1,8]. Verapamil and metoprolol were not available at the hospital, so amiodarone was chosen as the treatment for this patient.

While non-dihydropyridine calcium channel blockers (e.g., verapamil, diltiazem) or beta-blockers are typically the preferred second-line agents for narrow-complex SVT, amiodarone is a Class III antiarrhythmic that is effective for a wide range of arrhythmias, including those refractory to other treatments [1,9].

Although amiodarone has a slower onset of action compared to adenosine and carries a risk of hypotension, bradycardia, acute hepatotoxicity, and phlebitis, it remains a viable option in specific clinical contexts, particularly in patients with structural heart disease or ischemic potential, as it is less likely to depress myocardial contractility compared to calcium channel blockers [9]. Once the patient was discharged, she was transitioned to an oral beta-blocker (bisoprolol) for recurrent SVT prevention [1].

Crucially, the clinical course and investigations helped exclude other serious etiologies. The initial ECG captured a narrow complex tachycardia without visible P waves. The second ECG, obtained after amiodarone treatment, showed conversion to a normal sinus rhythm. Notably, no Q waves, ST elevation, ST depression, or T-wave inversions were detected, supporting the exclusion of acute coronary syndrome (ACS) [10].

Tachycardia increases myocardial oxygen demand, which can lead to demand ischemia (Type 2 MI), especially in elderly patients. The patient's normal cardiac silhouette on chest X-ray and normal Troponin I level (4 ng/L) helped exclude acute myocardial necrosis and heart failure [10].

Because the patient presented with atypical arrhythmia symptoms characterized by sharp abdominal pain, differential diagnoses such as aortic dissection or acute pancreatitis needed to be considered. However, a CT scan was not performed to exclude these pathologies because the ECG immediately identified the arrhythmia as the likely etiology of the pain.

Furthermore, the immediate cessation of pain upon the restoration of sinus rhythm provided critical diagnostic confirmation. Had the etiology been a primary abdominal pathology, such as acute pancreatitis or cholecystitis, the

pain would likely have persisted independently of the heart rate due to ongoing inflammation or obstruction. The complete disappearance of pain immediately following cardioversion with amiodarone confirmed that the abdominal symptoms were a referred manifestation of the arrhythmia ('angina equivalent'), validating the decision to forgo extensive abdominal imaging.

Regarding the disposition of the patient, the restoration of sinus rhythm was prioritized over immediate transfer to a tertiary facility. Transporting a symptomatic elderly patient with a heart rate of 170 bpm posed significant risks of hemodynamic deterioration. In this resource-limited setting, the decision to utilize intravenous amiodarone allowed for immediate stabilization. Given the patient's uncomplicated recovery and the lack of arrhythmia recurrence, the clinical team decided to continue observation at the current facility rather than initiating a transfer.

We acknowledge the limitations inherent in this case report. First, the diagnosis of AVNRT remains presumptive based on surface ECG criteria (regular narrow complex tachycardia with invisible P waves); definitive confirmation of the dual-pathway mechanism would require an electrophysiological study (EPS), which was not performed. Second, we relied on a single Troponin I measurement; while serial troponins are standard, the absence of specific ischemic changes on the post-cardioversion ECG and immediate symptom relief supported a non-ischemic etiology [11,12]. Finally, echocardiography and ambulatory rhythm monitoring were not available during the initial admission. The absence of these diagnostic tools limits our ability to fully assess the patient's structural heart disease status and risk of recurrence.

Conclusions

Supraventricular tachycardia presenting as acute abdominal pain is an atypical clinical entity that can lead to diagnostic delays, particularly in elderly diabetic patients. The awareness of this possibility following physical exertion, combined with the initiation of an early ECG, could prevent misdiagnosis as a gastrointestinal disorder. We acknowledge the limitations inherent in this case report. The diagnosis of AVNRT remains presumptive based on surface ECG criteria; definitive confirmation of the arrhythmia mechanism would require an electrophysiological study, which was not performed. Regarding the disposition, the decision to treat the patient locally rather than transferring immediately was made to avoid the hemodynamic risks of transporting a symptomatic elderly patient. Local observation was continued due to the patient's sustained stability and absence of arrhythmia recurrence. Intravenous amiodarone was administered solely because adenosine was unavailable. This represents a deviation from standard first-line guidelines. Due to amiodarone's potential toxicity and slower onset, it should be used with caution, and adenosine remains the preferred choice when available. Finally, the absence of echocardiography and ambulatory rhythm monitoring limits our assessment of structural heart disease and recurrence risk, underscoring the need for further follow-up.

References

1. Brugada J, Katritsis DG, Arbelo E, et al.: 2019 ESC Guidelines for the management of patients with supraventricular tachycardia The Task Force for the management of patients with supraventricular tachycardia of the European Society of Cardiology (ESC): Developed in collaboration with the Association for European Paediatric and Congenital Cardiology (AEPC). *Eur Heart J.* 2020, 41:655-720. 10.1093/eurheartj/ehz467
2. Link MS: Clinical practice. Evaluation and initial treatment of supraventricular tachycardia. *N Engl J Med.* 2012, 367:1438-48. 10.1056/NEJMcp1111259
3. Lilly LS: Pathophysiology of heart disease: an introduction to cardiovascular medicine 7th Edition. Leonard S. Lilly. Leonard S. Lilly (ed): Wolters Kluwer, Philadelphia; 2021. 12:315-320.
4. Yetkin E, Ozturk S, Cuglan B, Turhan H: Clinical presentation of paroxysmal supraventricular tachycardia: evaluation of usual and unusual symptoms. *Cardiovasc Endocrinol Metab.* 2020, 9:153-8. 10.1097/XCE.0000000000000208
5. Vinik AI, Ziegler D: Diabetic Cardiovascular Autonomic Neuropathy. *Circulation.* 2007, 115:387-97. 10.1161/CIRCULATIONAHA.106.634949
6. Foreman RD: Mechanisms of cardiac pain. *Annu Rev Physiol.* 1999, 61:143-67. 10.1146/annurev.physiol.61.1.143
7. Appelboom A, Reuben A, Mann C, et al.: Postural modification to the standard Valsalva manoeuvre for emergency treatment of supraventricular tachycardias (REVERT): a randomised controlled trial. *The Lancet.* 2015, 386:1747-53. 10.1016/S0140-6736(15)61485-4

8. Kotadia ID, Williams SE, O'Neill M: Supraventricular tachycardia: An overview of diagnosis and management. Clin Med Lond Engl. 2020, 20:43-7. 10.7861/clinmed.cme.20.1.3
9. Fox DJ, Tischenko A, Krahm AD, Skanes AC, Gula LJ, Yee RK, Klein GJ: Supraventricular tachycardia: diagnosis and management. Mayo Clin Proc. 2008, 83:1400-11. 10.4065/83.12.1400
10. Thygesen K, Alpert JS, Jaffe AS: Fourth Universal Definition of Myocardial Infarction (2018). Circulation. 2018, 138:618-51. 10.1161/CIR.0000000000000617
11. Ben Yedder N, Roux JF, Paredes FA: Troponin Elevation in Supraventricular Tachycardia: Primary Dependence on Heart Rate. Can J Cardiol. 2011, 27:105-9. 10.1016/j.cjca.2010.12.004
12. Chow GV, Hirsch GA, Spragg DD, Cai JX, Cheng A, Ziegelstein RC, Marine JE: Prognostic Significance of Cardiac Troponin I Levels in Hospitalized Patients Presenting With Supraventricular Tachycardia. Medicine (Baltimore). 2010, 89:141-148. 10.1097/MD.0b013e3181dddb3b