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Case Report

Supraventricular Tachycardia as a Cardiopulmonary Stress Response to Traumatic Chylopneumothorax and Diaphragmatic Hernia: A Case Report

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Abstract

Background: Supraventricular Tachycardia (SVT) is a heart rhythm disorder characterized by an increased heart rate originating from the Atrioventricular (AV) node in the atrium. It is characterized by an Electrocardiography (ECG) tracing showing a narrow QRS complex (<120 ms) and a rapid, regular heartbeat. Blunt trauma to the thoracoabdominal region can cause diaphragmatic hernia and chylopneumothorax. Both conditions could escalate the progression of cardiopulmonary stress, such as heart rhythm disturbance (arrhythmia).

Case Illustration: We report a case of a 30-year-old woman who presented to the Emergency Room with sudden palpitations 1 day ago. Physical examination revealed a restless appearance (Glasgow Coma Scale [GCS] E4V5M6), blood pressure 146/94 mmHg, pulse rate 155 beats per minute (bpm), respiratory rate 30 bpm, temperature 36 °C, and saturation 95% on room air. In the thoracic region, dull percussion was found in the lower left hemithorax, decreased left lung base sounds, bowel sounds were heard, and tenderness was observed in the left costovertebral angle in the abdominal region. ECG examination showed the presence of SVT with a heart rate of 176 bpm. The patient was initially managed by performing a vagal maneuver, but it failed. The patient was then transferred to the Intensive Care Unit (ICU), underwent further diagnostic examinations, and received further therapy.

Conclusions: SVT in patients with thoracoabdominal trauma is not a primary cardiac abnormality, but rather an indicator of life-threatening trauma-related cardiopulmonary compromise. Clinicians need to recognize the importance of timely and appropriate intervention, along with adequate cardiopulmonary support, to improve patient outcomes.

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Keywords: Arrhythmia, Supraventricular tachycardia, Thoracoabdominal trauma, Chylopneumothorax, Diaphragmatic hernia

Introduction

The term 'SVT' literally indicates tachycardia [atrial rates >100 beats per minute (bpm) at rest], the mechanism of which involves tissue from the His bundle or above.[10] Supraventricular Tachycardia (SVT) is characterized by an Electrocardiography (ECG) showing a narrow QRS complex (less than 120 ms) and an increased heart rate. The heart rate exceeds 100 bpm in adults.[1]

Thoracoabdominal trauma may cause Blunt Cardiac Injury (BCI), including myocardial contusion or irritation of the cardiac conduction system. Mechanical impact to the anterior chest wall can transmit kinetic energy to the heart, leading to direct myocardial cell injury and electrical instability, which may disrupt normal impulse or conduction pathways. This process can result in various dysrhythmias, including SVT.[2-3]

Chylothorax could occur together with pneumothorax due to trauma to the chest wall, where the incidence occurs is about 0.2% following blunt thoracic trauma and 0.9% after penetrating trauma.[4-5]

Blunt trauma to the thoracoabdominal region can cause tears in the diaphragm, allowing intra-abdominal organs to migrate into the thoracic cavity, known as Traumatic Diaphragmatic Hernia (TDH). This condition often occurs on the left side because the liver provides protection on the right, which can lead to impaired ventilation and perfusion.[6] Thoracoabdominal trauma can also cause damage to the thoracic duct pathway, resulting in lymphatic fluid leakage into the pleural cavity and the development of a chylothorax, or if accompanied by air, it is called chylopneumothorax. Although rare in cases, chylopneumothorax has high morbidity if recognized late.[4,7]

Thoracoabdominal trauma is frequently associated with hypoxia. Hypoxia also stimulates sympathetic activation, which increases Atrioventricular (AV) nodal conduction and shortens refractory periods, thereby facilitating re-entrant arrhythmias such as SVT.[6-8]

This case report aims to describe the relationship between thoracoabdominal trauma and the emergence of complex cardiopulmonary complications and to emphasize the importance of multidisciplinary detection and management in trauma patients with cardiac manifestations.

Case Illustration

A 30-year-old woman presented to the Emergency Room of Dr. Ario Wirawan Lung Hospital with complaints of palpitations. The complaint began 1 day ago and did not improve with rest. Five days prior, the patient had fallen off her motorcycle and entered the bushes, but had no complaints afterward. The patient denied chest pain radiating to the back and left arm, severe headache, or loss of consciousness. However, she reported vomiting twice an hour before admission. Physical examination revealed a restless appearance (Glasgow Coma Scale [GCS] score E4V5M6), blood pressure 146/94 mmHg, pulse rate 155 bpm, respiratory rate 30 bpm, temperature 36°C, and saturation 95% on room air. In the thoracic region, dull percussion was found in the lower left hemithorax, decreased left lung base sounds, and bowel sounds were heard. In the abdominal region, tenderness was found in the left costovertebral angle. ECG examination showed the presence of an SVT rhythm with a heart rate of 176 bpm (Figure 1).

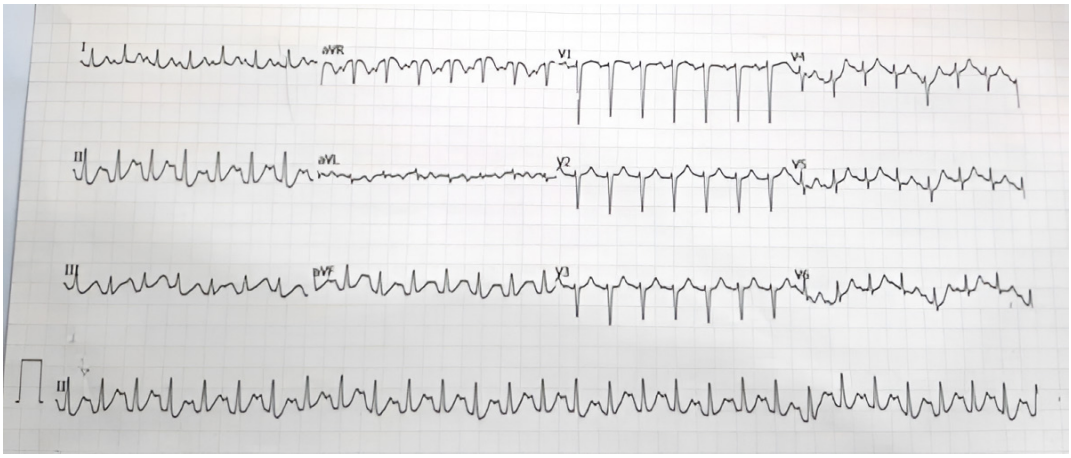


Figure 1. ECG image showing supraventricular tachycardia rhythm with recognizable P waves on Lead II. The PR interval and RP interval are 110 ms and 210 ms.

Based on the history, physical examination, and ECG, the patient was diagnosed with SVT with stable hemodynamics. Initial management was performed with a vagal maneuver, which failed, followed by administration of digoxin 0.5 mg; there was no resolution. The patient was then transferred to the Intensive Care Unit (ICU) and received O_2 via Non-Rebreathing Mask (NRM) at 10 L/min, fentanyl 200 mcg, and amiodarone 150 mg bolus. The bedside monitor showed that the patient's rhythm reverted to sinus rhythm, displaying a heart rate of 78 bpm. Unfortunately, the ECG for the sinus rhythm was not recorded. Administration of amiodarone was continued with a maintenance dose of 1 mg/min for 6 h and 0.5 mg/min for 18 h, as well as furosemide 20 mg/24 h, ampicillin-sulbactam 1.5 gr/8 h, methylprednisolone 62.5 mg/8 h, ketorolac 30 mg/8 h, tranexamic acid 500 mg/8 h, ivabradine 2×5 mg, and digoxin 1×0.25 mg.

The patient underwent other diagnostic examinations, including laboratory tests and thoracic radiography. Chest radiography showed homogeneous opacification in the left apicolaterobasal hemithorax, accompanied by a shift of the mediastinal and tracheal structures to the right (Figure 2a). Next, a Water-Sealed Drainage (WSD) was placed immediately (Figure 2b).

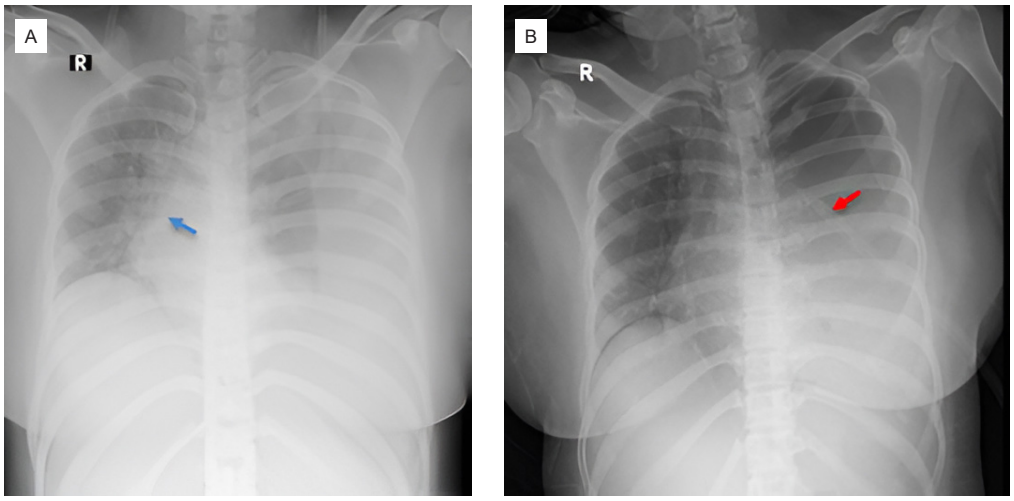


Figure 2. Chest X-ray image showing: (a) Opacity is seen in the left hemithorax accompanied by mediastinal shifting to the right (blue arrows), (b) A chest tube is seen in the left hemithorax of Intercostal Space (ICS) V post-WSD procedure (red arrows).

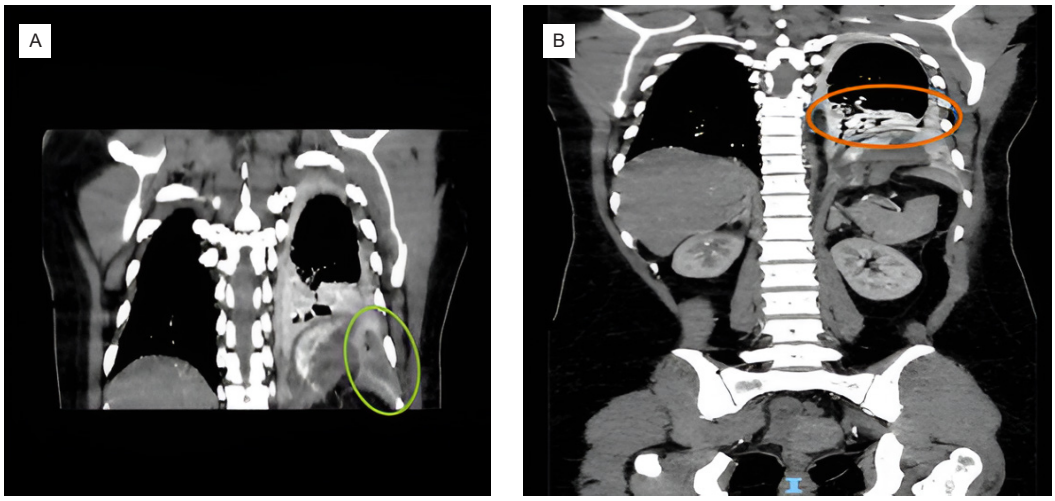


Figure 3. CT scan image showing: (a) A chest tube is seen in the left hemithorax with the tip in the left posterior of the intercostal space (ICS) V and left chylopneumothorax accompanied by collapse of the inferior lobe of the left lung (green circle), (b) A defect in the left hemidiaphragm accompanied by herniation of bowel structure into the left hemithorax (orange circle).

A WSD fluid specimen was obtained, with triglycerides 728 mg/dl and no malignant cells found. Blood test results obtained: blood glucose 290 mg/dl, leukocytes 17.03x103/µl, urea 48.7 mg/dl, creatinine 1.43 mg/dl, pH 7.503, pCO_2 18.4 mmHg, pO_2 174.8 mmHg, HCO_3- 14.3 mmol/L. CT scan examination of the thoracoabdominal region with contrast showed the appearance of a left chylopneumothorax accompanied by collapse of the inferior lobe of the left lung and pulmonary contusion (Figure 3a), as well as a defect in the left hemidiaphragm accompanied by herniation of the bowel structure into the left hemithorax (Figure 3b).

The patient was subsequently referred to another hospital for further management and treatment by a Thoracic, Cardiac, and Vascular Surgeon for the left diaphragmatic hernia.

Discussion

SVT is a heart rhythm disorder characterized by an increased heart rate originating from the tissue above the ventricles, namely in the atrium or, more precisely, in the AV node. SVT is characterized by an ECG examination showing a narrow QRS complex (less than 120 ms) and an increased heart rate.[1]

In the present case, the patient experienced SVT and according to algorithm above (bold arrows), a focal AT could be likely consider although the other form like Atrioventricular Reentrant Tachycardia (AVRT) and Atrioventricular Nodal Re-entrant Tachycardia (AVNRT) could not be ruled out as the heart rate is reguler and exceeds 100 bpm (regular tachycardia) with visible uniform P waves, 1:1 of atrial ventricular rate with PR interval above 70 ms, and RP interval (210 ms) is longer than PR interval (110 ms).

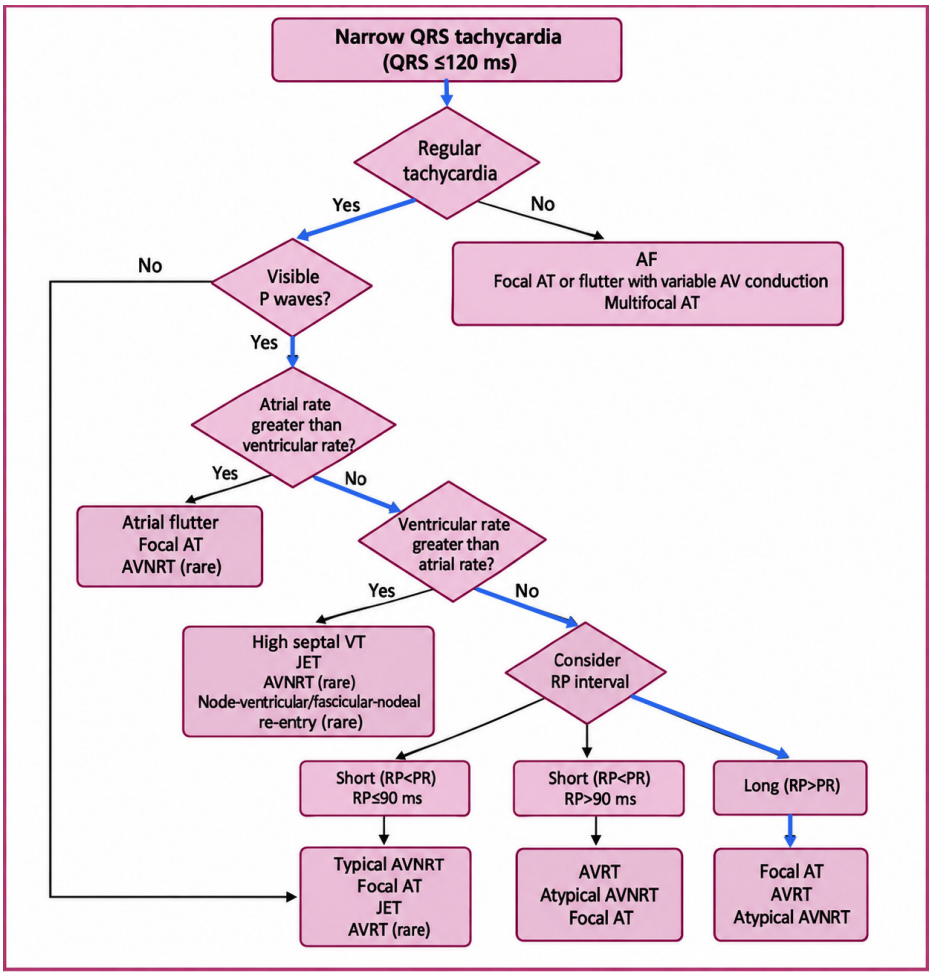


Figure 4. Differential diagnosis of narrow QRS tachycardia.[9]
Abbreviation: AF = atrial fibrillation; AT = atrial tachycardia; AV = atrioventricular; AVNRT = atrioventricular nodal re-entrant tachycardia; AVRT = atrioventricular reentrant tachycardia; JET = junctional ectopic tachycardia; RP = RP interval; VT = ventricular tachycardia.

Generally, the mechanisms responsible for cardiac arrhythmias are abnormal impulse formation (i.e., increased automaticity or triggered activity) and conduction disturbances, such as re-entry. The risk factors for arrhythmia in this case are abnormal impulse formation (i.e., increased automaticity or triggered activity) and conduction disturbances, such as re-entry. Abnormal impulse formation may arise from enhanced automaticity or triggered activity caused by early or delayed afterdepolarizations, which generate ectopic electrical impulses within the myocardium. Conduction disturbances, particularly reentry, occur when an electrical impulse continuously circulates through myocardial tissue due to unidirectional block and heterogeneity of conduction velocity and refractory periods, allowing repeated re-excitation of previously activated tissue. These electrophysiological abnormalities create both the trigger and the substrate necessary for the initiation and maintenance of clinically significant arrhythmias such as SVT.[11]

Focal Atrial Tachycardia (AT) is a form of SVT that originates from a single ectopic focus within the atrium but outside the sinus node, with impulses spreading centrifugally throughout the atrial tissue. The atrial rate in this condition typically ranges from 100 to 250 bpm and can be persistent or non-persistent.[12]

From an electrophysiological perspective, trauma itself does not create new arrhythmogenic substrates such as dual AV nodal physiology or new reentry circuits. Instead, the physiological stress associated with trauma likely acts as a trigger, activating pre-existing but clinically asymptomatic electrophysiological substrates. In this patient, the severe respiratory distress caused by diaphragmatic rupture and chylopneumothorax likely resulted in hypoxemia and sympathetic activation. This increased atrial myocardial excitability and facilitated the development of Premature Atrial Complexes (PACs). PACs can serve as trigger impulses that initiate focal AT or activate latent supraventricular arrhythmia mechanisms.[8]

Blunt trauma to the thoracoabdominal region can cause a sudden increase in intra-abdominal pressure, resulting in diaphragmatic rupture, which can lead to post-TDH, particularly on the left side. Diaphragmatic defects allow abdominal viscera, such as the small intestine or colon, to herniate into the thoracic cavity, thereby compressing the lungs and mediastinal structures. It can trigger sudden changes in intrathoracic pressure and asymmetry of pulmonary perfusion and can also trigger activation of the sympathetic nervous system, which ultimately triggers the onset of supraventricular arrhythmias.[4,7,13]

Thoracic duct leakage can result from direct tearing due to blunt trauma or secondary traction from mediastinal organ shifting due to herniation. Accumulation of chylous fluid in the pleural cavity, accompanied by air from alveolar rupture, creates a chylopneumothorax, which typically shows pleural fluid triglyceride levels >110 mg/dl, as seen in this patient (728 mg/dl).[6,13-14] This condition worsens respiratory distress and increases cardiopulmonary strain.

Arrhythmias after blunt chest trauma are not always immediate and may occur hours to days after the initial injury due to evolving myocardial injury or secondary physiological changes within the thoracic cavity.[11] In the present case, SVT did not occur immediately after the trauma but developed on the fifth day of hospitalization. This delayed presentation may be explained by the gradual accumulation of chylothorax, which can progressively increase intrathoracic pressure and subsequently lead to cardiopulmonary compromise. Increased intrathoracic pressure may influence cardiac filling pressures and atrial stretch, which in turn may predispose patients to SVT.[15]

It can be concluded that SVT in patients with thoracoabdominal trauma is a warning sign of significant respiratory or structural impairment, not simply a primary dysrhythmia. Simple diagnostic examinations, such as chest radiography and pleural fluid analysis, are crucial for establishing the initial diagnosis, while a thoracoabdominal CT scan is necessary to determine the etiology and plan treatment. Early detection, appropriate cardiopulmonary stabilization, and multidisciplinary coordination are the main keys to reducing morbidity and mortality in trauma cases with complex cardiopulmonary complications such as this.

Conclusion

This case demonstrates that SVT in patients with thoracoabdominal trauma is not a primary cardiac abnormality, but rather an indicator of life-threatening trauma-related cardiopulmonary compromise. Clinicians need to recognize the importance of timely and appropriate intervention, along with adequate cardiopulmonary support, to improve patient outcomes.

List of Abbreviations

AF	Atrial Fibrillation
AT	Atrial Tachycardia
AV	Atrioventricular
AVNRT	Atrioventricular Nodal Re-entrant Tachycardia
AVRT	Atrioventricular Reentrant Tachycardia
BCI	Blunt Cardiac Injury
bpm	beats per pinute
ECG	Electrocardiography
GCS	Glasgow Coma Scale
ICU	Intensive Care Unit
ICS	Intercostal Space
JET	Junctional Ectopic Tachycardia
NRM	Non-Rebreathing Mask
PACs	Premature Atrial Complexes
SVT	Supraventricular Tachycardia
TDH	Traumatic Diaphragmatic Hernia
VT	Ventricular Tachycardia
WSD	Water-Sealed Drainage

Ethical Clearance

Not applicable.

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

The first author (IPBM) conceived the case report, collected the clinical data, drafted the initial manuscript and coordinated. The second author (IKP) supervised clinical management and critically revised the cardiological interpretation and manuscript content. The remaining authors (WP,NT,NAA) contributed literature review, data verification, fugure preparation manuscript drafting, and reference formatting. All authors read, critically revised, and approved the final manuscript.

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None.

Conflict of Interest

The authors declared that they have no competing interests.

Availability of Data and Materials

None.

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None.

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Generative AI and AI-Assisted Technologies in the Writing Process

Authors acknowledge that artificial intelligence (AI) tools were only used to assist in language editing and did not generate or alter the scientific content, analyses, or conclusions presented in this manuscript.

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