Case Report

Dramatic termination of rate-dependent supraventricular tachycardia aberrancy with midazolam in sympathetic overactivity.

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Abstract

Rationale: Paroxysmal supraventricular tachycardia is an episodic arrhythmia with variant electrocardiographic presentations. Patient concerns: A 30-year-old married male, heavy-smoker, Egyptian worker presented with unstable regular wide QRS-complex tachycardia after the anxiety event. Midazolam dramatically terminated both the presented supraventricular tachycardia with aberrancy and sympathetic overactivity. Diagnosis: Supraventricular tachycardia with aberrancy due to bundle branch block in the state of anxiety-related sympathetic excessive activity. Interventions: Electrocardiography, oxygenation, a trial of Valsalva maneuvers, intravenous sedation, and intensive care unit monitoring. Outcomes: Midazolam dramatically terminated both the presented supraventricular tachycardia with aberrancy and sympathetic overactivity. Lessons: Conversion of tachycardia related-aberrancy to sinus rhythm indicates rate dependency (RDBBB). Midazolam dramatically terminated both the presented supraventricular tachycardia with aberrancy and sympathetic overactivity. This reported case highlighted the role of short sedation in cases of supraventricular tachycardia with aberrant conduction in sympathetic overactivity.

Keywords: Rate-dependent supraventricular tachycardia, Supraventricular aberrancy, Midazolam, Sedation, Sympathetic overactivity
1. Introduction

Paroxysmal supraventricular tachycardia (PSVT) is an episodic cardiac arrhythmia with spontaneous onset and termination. Generally, PSVT is defined as any tachyarrhythmia that demands either atrial or atrioventricular (A/V) nodal tissue for its commencement and continuation. Alcohol consumption, caffeine drink, nicotine smoking, emotional stress, and Wolff-Parkinson-White (WPW) syndrome are included as risk factors. PSVT is rarely asymptomatic. Palpitations and severe symptoms are often the main presentations. Electrophysiological study (EPS) is an essential workup for exploring the origin of the conduction abnormalities and the pathophysiology associated changes in impulse generation and accessory pathways. It is preferable to treat the patients PSVT with Valsalva maneuvers, drugs, and occasionally electric shocks. Immediate synchronized cardioversion can be used with sedation in patients who are hypotensive with pulmonary edema, and ischemic chest pain, and who are unstable.

The term aberrations symbolize a temporary bundle branch block (BBB) with no QRS-complex abnormalities. The main causes are past bundle branch block, Wolff-Parkinson-White (WPW) syndrome, and drug adverse effects. Various mechanisms implicated in the presence of a temporary bundle branch block. These include; phase 3 block, phase 4 block, and concealed conduction. These mechanisms of aberration occasionally affect any part of the His-Purkinje system (HPS). This is different chronic BBB, due to shifting the site of the heart block during aberrancy. The right bundle branch block (RBBB) is the most frequent pattern of aberrancy. RBBB usually exists in 80% of aberrancy cases but up to 100% of aberration is happening in normal hearts. Several factors implicated in producing RBBB. An idiopathic RBBB occurs if there is no known heart disorder. So, the presence of alone and isolated RBBB in ECG does not certainly indicate the existence of underlying cardiac pathology. Indeed, despite all the above causes, RBBB considered a nonspecific abnormality. However, RBBB may be accompanied by several organic heart disease. All condition that affects the right side of the heart can be causing RBBB. These are including; atrial septal defect (ASD) with left-to-right shunting, a chronic pulmonary disease with pulmonary hypertension (PHT), and valvular heart diseases (VHD) “e.g., pulmonary stenosis (PS)”, cardiomyopathies (CMP), ischemic heart diseases (IHD), chronic degenerative changes involving the conduction system (especially older patients), post-cardiovascular surgery, acute pulmonary embolism (APE). The above abnormal conditions will result in acute right-sided heart overload which caused the delay in right ventricular conduction with sinus tachycardia. An alone and isolated RBBB does not indicate the necessary need for certain therapy. RBBB may be permanent or transient.

Bifascicular heart block is a result of blockage of any 2 of the 3 fascicles. Right bundle branch block plus the left posterior fascicular block (LAFB) attains right bundle branch block configuration with right axis deviation or RAD (on condition that other differentials of RAD are excluded). Bifascicular heart blocks are possibly considerable due to the ventricular conduction based on the remaining fascicle. Further damage of the remaining fascicle is meaning indicates the presence of atrioventricular block or (trifascicular heart block).

Tachycardia-dependent block is often present due to the disappearance of the conduction defect with restoring the normal conduction. This is will be causing prolongation in cycle length mostly from 620 to 5,160 ms using either the Valsalva maneuver or carotid sinus massage or. The pathogenesis for interrupted BBB is still a controversial issue. Valsalva maneuvers are indicated to revealed normal conduction and phase 4 BBB. Tachycardia-dependent block occasionally happens only if the ventricular rate overrides a certain critical level (rate-related RBBB). But, it is a non-diagnostic finding.

Midazolam is the commonest used benzodiazepine (BDZs) in the critical care medicine due to its shorter half-time (t1/2) than the most other members of BDZs, water (H2O) solubility, and proportionality for intravenous infusion and the absence of active metabolites for it. It has become sedative of choice for most procedures needing sedation. Several factors proposed its use: 1. It is easy in giving. 2. It has expected effects. 3. It has outstanding safeness.
4. Rapid onset and short duration are characteristic. Its large hemodynamic stability. All these factors are accompanied by improved compliance\(^{10,11}\). It is also linked with shorter convalescent time, less associated vomiting, and respiratory depression. So, it the sedative of choice in a conscious patient\(^{11}\). Midazolam sharing the other members of BDZs in causing peripheral vasodilatation and subsequent hypotension\(^{12}\). Midazolam is primarily used preoperatively as an anxiolytic medication\(^{12}\). Its half-time is about 90-150 minutes\(^{13}\). Midazolam is frequently used for conscious sedation procedures expected to last less than one hour\(^{13}\). The initial dose for IV procedural sedation, such as transcutaneous pacing (TCP) or cardioversion, is often 0.5-1 mg via intravenous route over 2 minutes (maximum 2.5 mg/dose). Afterward, the physician should be waiting 2-3 minutes after each dose to assess its sedation influence. A total dose of more than 5 mg usually is not necessary to reach the desired sedation\(^{14,15}\). Doses are repeated every 3–5 minutes\(^{15}\).

Overactivity of the sympathetic nervous system (SNS) manifested as diaphoresis, and tachycardia, and hypertension\(^{15,16}\). The sympathetic nervous system is the way for the manifestations of anxiety\(^{12}\). However, increased sympathetic activity is claimed to be involved in the inducing of anxiety\(^{12}\). Benzodiazepines are used to reducing anxiety, and for preoperative sedation\(^{13}\). The GABA A receptor increases Cl\(^–\) conductance\(^{16,17}\) that is the target site for benzodiazepines action\(^{16}\). Benzodiazepines bind to gamma-aminobutyric acid (GABA) receptors and work as muscle relaxants reducing anxiety and sympathetic overactivity\(^{15}\). Hypotension and slow heart rate with a slow down of high respiratory rates are adverse effects of benzodiazepines\(^{13}\).

2. Case report

A 30-year-old married male, heavy-cigarette smoker, Egyptian worker presented to the emergency room with chest pain, dizziness, and sensation of the heart beating. Symptoms had happened after the anxiety event. The patient had a history of PSVT for a year ago and on oral verapamil tablet (240 mg, once daily). The patient denied a history of other related diseases. Upon examination, the patient appeared irritable, sweating profusely, pale, tachypneic, and anxious. The primary vital signs were as follows: BP of 150/90 mmHg, the heart rate of 220/bpm and regular, the body temperature of 37.7°C, and respiratory rate of 22/min. The initial workup was: electrocardiogram (ECG), that showed regular wide QRS complex tachycardia at 220 beats/min with right bundle branch block configuration and right axis deviation (RAD) (Figure 1). The patient was admitted to ICU and managed for PSVT. Unfortunately, Valsalva maneuvers were tried with no response. Oxygen inhalation (5 L/min ) was given. Intravenous midazolam (2.5 mg over 5 minutes) was urgently given aiming for direct current cardioversion (DCC) shock procedural preparation. Before performing the DCC, the ECG recordings showed an abrupt disappearance of supraventricular tachycardia aberration with complete sedation. The patient was already connected to the ICU monitor for vitals and O\(_2\) saturation follow up. No DCC shock was given. ECG tracing was repeated after the SVT aberration disappearance and showed slight sinus tachycardia (Figure 2). The investigations done were: Troponin test, electrolyte profiles, full blood picture, thyroid function tests, random blood sugar, and echocardiography with no detectable abnormal results. The entire recovery had accomplished and the patient was discharged within 6 hours from admission with no problem. Midazolam 7.5 mg once daily oral tablet at night was prescribed only for one-week post-discharge. Planning for the future electrophysiological study was recommended.
Figure 1: A 12-lead ECG tracing upon arrival to the ICU department showing wide-complex tachycardia with a ventricular rate of 220 bpm, indicating RBBB and LPFB morphology (bifascicular block) with marked RAD (131°).

Figure 2: ECG tracing after midazolam administration showing sinus tachycardia with a ventricular rate of 115 bpm with the disappearance of bifascicular block.

3. Discussion

Sympathetic overactivity was the main presentation for the current case. Unstable supraventricular tachycardia with aberration was relieved dramatically with midazolam sedation. DCC shock therapy was unintentionally avoided in the management of unstable supraventricular tachycardia with an aberration attack due to the normalization of above arrhythmia with a single dose of midazolam. Sympathetic overactivity manifestation disappeared quietly with complete midazolam sedation. There were no similar published cases for comparison with the current case.

The following implicated in differential diagnoses; 1. Right ventricular outflow tract tachycardia or RVOT (LBBB morphology in V1 plus an inferior axis, sporadic PVCs, non-sustained ventricular tachycardia,
sustained ventricular tachycardia (VT), and both aVR and aVL leads are remarkable negative taking a QS-pattern). 2. **Left anterior fascicular ventricular tachycardia** or LAFVT (typical RBBB configuration, inferior axis, QRS-alternans, short QRS-complexes duration. 3. **Monomorphic ventricular tachycardia** (MVT); with only using Brugada algorithm, morphology criteria, and Vereckei aVR algorithm (the following criteria; atrial ventricular dissociation, appearance of fusion beats, presence of capture beats, absent RS-configuration in chest leads, presence of an initial R-wave in lead aVR, an initial small r or q-wave of > 40 ms width in aVR lead, notching on the descending limb of a negative onset, dominantly negative QRS-complex in lead aVR, monomorphic positive or negative concordance in chest leads, Q-waves in chest leads, extreme right axis, QRS width >160 milliseconds favor ventricular tachycardia). 4. **Polymorphic ventricular tachycardia** or PVT (an irregular, >1 morphologically distinguished QRS-complex happening through the same attack of VT, extremely rapid ventricular rate, QRS-complexes seem to be twisting around the isoelectric baseline, frequently non-sustained, abnormal-looking with constantly changing QRS-complexes, and progressive axis deviation with twisting of points). 5. **Ventricular fibrillation** or VF (chaotic tachycardia without consistently identifiable QRS-complexes). 6. **Supraventricular tachycardia with preexcitation** (regular, delta wave, retrograde P-wave may be concealed within the QRS-complex, producing a pseudo-R’-wave pattern in V1, and a pseudo-S-pattern in the II, III, and aVF leads) [18-23].

4. Conclusion

The reported case highlighted the pivotal role of sedation with a short-acting benzodiazepine; midazolam, in terminating an acute attack of unstable supraventricular tachycardia with aberrancy due to RBBB, specifically; bifascicular block. Sympathetic overactivity manifestations should be considered during the management of unstable supraventricular tachycardia with the aberrant attack. Sedation with midazolam will avoid DCC shock therapy and its hazards.

Conflicts of interest

There is no potential conflict of interest was reported by the author.

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References


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