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Case Report

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Test the Termination of Ischemic Ventricular Tachycardia and QT-Prolongation in Hypokalemic Hypertensive Patient with Nitroglycerine; A Case Report

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Abstract

Rationale: Ventricular tachycardia is one of the most serious arrhythmias. It is arrhythmia of death. Ischemic heart disease considered the most common cause of ventricular tachycardia. No doubt that hypokalemia and QT-prolongation are important causes. Nitroglycerine was the new therapeutic key for the current case.

Patient concerns: A 90-year-old, Egyptian male, farmer, heavy cigarette smoker patient presented in the Emergency room with ventricular tachycardia and prolonged QT in the presence of hypokalemia, hypertension, and Parkinson's disease with history of ischemic heart disease.

Diagnosis: Ischemic ventricular tachycardia with prolonged QT over hypokalemia and hypertension.

Interventions: Electrocardiogram and nitroglycerine intravenous infusion.

Outcomes: Dramatic termination of ventricular tachycardia with normalization of QT-interval.

Lessons: Regards the dramatic response of ischemic ventricular tachycardia and QT-prolongation to nitroglycerin, it may be used as a new therapeutic and diagnostic antiarrhythmic agent in like-these cases. Hypokalemia is a precipitator for both ventricular tachycardia and QT-prolongation.

Keywords: Test the termination, Ischemic ventricular tachycardia, QT-prolongation, Hypokalemia, Hypertension, Nitroglycerin

Abbreviations

AAD : Antiarrhythmic drug
AMI : Acute myocardial infarction
ECG : Electrocardiogram
HTN : Hypertension
ICU : Intensive care unit

IHD : Ischemic heart disease
IVI : Intravenous infusion
PVC : Premature ventricular contraction
PD : Parkinson's disease
SCD : Sudden cardiac death
SHD : Structural heart disease
VF : Ventricular fibrillation
VT : Ventricular tachycardia

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Introduction

Ventricular tachycardia (VT) considered the myocardial arrhythmias under His bundle branch and myocardial conduction fiber [1]. Ventricular tachycardia or ventricular fibrillation (VF) represent most of the sudden cardiac death (SCD) in the United States (US). The estimated rate of about 300,000 deaths per year [2]. VT is defined as any rhythm faster than 100 (or 120) beats/min, with three or more consecutive spontaneous premature ventricular contraction (PVCs) in a row, arising distal to the bundle of His [2-4]. If there are ≥ 6 of rapid PVCs (frequency $>100/\text{min}$) are considered as persistent VT (≥ 30 seconds), while less than 6 beats as non-persistent VT (≤ 30 seconds). Persistent VT is very serious, as it can induce VF and SCD1. Patients with PVC couplets are more prone to the development of VT than with single PVC4 PVCs is a Precursor for VT and VF [5]. Hypokalemia is an important VT inducer, followed by hypomagnesemia [2]. Acquired channelopathies, most commonly from drugs that prolong the QT-interval may also promote VT2. Patients with ischemic heart disease (IHD) are at increased risk of incident or recurrent VT, VF, and sudden cardiac death [6].

The following causes implicated in VT [2,7]:

- IHD (the most common cause).
- Structural heart disease (SHD) with disruption of normal conduction patterns e.g. non-ischemic cardiomyopathy.
- Congenital SHD e.g. tetralogy of Fallot.
- Acquired channelopathies e.g. drugs that prolong the QT-interval e.g. phenothiazine's.
- Inherited channelopathies e.g. long QT-syndrome.
- Electrolyte imbalances eg, hypokalemia.
- Sympathomimetic agents e.g. cocaine.
- Systemic diseases causing infiltrative cardiomyopathy or scar eg, sarcoidosis.

Nitroglycerin (NTG) is the most common and eldest prescribed short-acting anti-anginal drug. Although the drug was added in the clinical applications since 1879, there are some defects in the education of both patients and health care providers on the different benefits of short-acting nitrates. Nitrates are vasodilators with marked venous effects on large capacitance vessels. Indeed, nitrates increase coronary collateral circulation, increase aortic compliance and conductance and blood flow to ischemic areas of the myocardium [7]. Nitrates alleviate anginal symptoms by directly influencing the coronary arteries, coronary collateral

circulation, aortic compliance and conductance, and blood flow to ischemic areas of the myocardium [8]. Short-acting nitrates like- NTG are beneficial in acute IHD [9]. Myocardial ischemia is the result of an imbalance between myocardial O_2 demand and myocardial O_2 supply. Despite NTG is clinically effective in the therapy of this condition, its exact mechanism of action is still uncertain [10]. Because of clinical observations suggesting that NTG may suppress PVCs during acute myocardial ischemia, a study was undertaken to assess the effect of NTG on the incidence of PVCs in patients with acute myocardial infarction (AMI) [11]. NTG yield a significantly more rapidly decreasing in ventricular arrhythmias: 6 hours after onset of recording, the number of PVCs had declined to 39% of the baseline value [12]. NTG is one of the most useful anti-ischemic agents. IHD may manifest and presented with ventricular arrhythmias like PVCs. Despite NTG is not known as an antiarrhythmic drug (AAD) but Suzanne B. and her fellows in reported that the clinical observations suggesting that NTG may suppress PVCs during acute IHD [13]. The corresponding author also reported a case of PVCs-bigeminy with a dramatic response of bigeminy to NTG infusion [1]. The data indicate that NTG may decrease the number of PVCs for up to 3 hours in patients with AMI [14]. NTG is one of the most useful AADs [15]. But it is not known as AAD [15]. In the last two years, nitroglycerin was introduced by the author as a newer antiarrhythmic agent in two case reports. The first reported case (2017) [15] was a case of ischemic PVCs-bigeminy had shown a dramatic response to a trace dose of intravenous NTG infusion. The second reported case (2019) [16,17] was another case of ischemic PVCs-quadrigeminy had shown a dramatic response to a trace dose of intravenous NTG infusion. However, recently the author in his observational retrospective study, reported fourteen cases of various PVCs were effectively treated with intravenous NTG infusion [5].

Case Presentation

A 90-year-old, heavy cigarette smoker, farmer, Egyptian male patient presented in the Emergency room with dizziness, chest pain, dyspnea, and tremor. Profuse sweating was associated symptom. The patient is a Parkinson's disease. The patient gave a past history of hypertension (HTN). He is a heavy cigarette smoker (40 cigarettes for about 40 years). Upon general physical examination; generally, the patient was masked faces, dyspneic, severe sweaty, and had cold extremities. The heart rate was regular of 110 bpm, blood pressure of 170/90 mmHg, respiratory rate of 24 bpm, the temperature of 36.3 °C, pulse oximeter of O_2 saturation of 94% and tachycardia on heart auscultation. No more relevant clinical data were noted during the clinical examination. The initial ECG tracing showed T-wave in inversions in I, II, aVL and aVF leads with generalized tremor artifacts (**Figure 1A**). Within 5 minutes of the first ECG, the second tracing showed runs of ventricular tachycardia in I, II, aVR and aVF leads with prolongation of QTc-interval (494 ms) in V4-6 leads (**Figure 1B**).



Figure 1: Serial ECG tracings; A. tracing showing T-wave in inversions in I, II, aVL and aVF leads (green arrows) with generalized tremor artifacts (red arrows), and sudden movement artifacts (blue arrows). B. tracing showing runs of ventricular tachycardia in I, II, aVR and aVF leads (green arrows) with prolongation of QTc-interval (494 ms) in V4-6 leads (brown lines), ST-segment depressions in V4-6 (blue arrows), and still some artifacts (brown arrows).

O₂ inhalation 100% using the nasal mask (5 l/m) was given. The physician had started to giving of nitroglycerin IVI in very low dose (10 mg/50 ml solvent, 5 ug/min, with 3 drops/min) relatively for 30 minutes only as a therapeutic and diagnostic test [5,15,16]. The third ECG tracing was taken after nitroglycerin IVI and during patient sleep to avoidance the tremor artifact. It showed complete disappearance of above runs of VT with normalization of QT-interval (**Figure 2**).

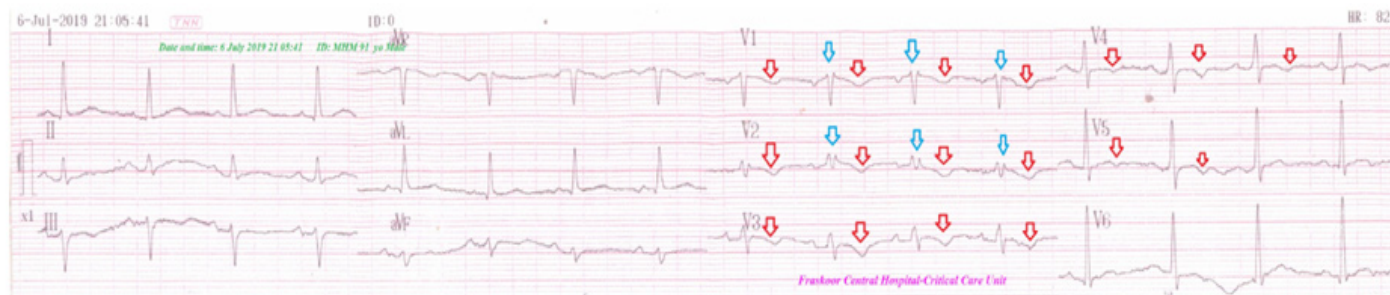


Figure 2: ECG tracings after nitroglycerin IV infusion showing complete disappearance of above runs of ventricular tachycardia with normalization of QT-interval. There is an appearance of incomplete RBBB (blue arrows) and T-wave inversions in V1-5 (red arrows).

The requested workup was: Electrolytes profile show: Na^+ : 141 mmol/L, K^+ : 2.6 mmol/L, ICa^{++} : 1.2 mmol/L, Hb: 15.2 gm/dl, Random blood glucose: 111 mg/dl. Troponin test was positive (368 ng/mL). Thyroid function tests were normal. The echocardiographic report showed evidence of IHD with inferior regional wall motion abnormality with systolic dysfunction and low ejection fraction (53%, that was measured with 2-D). Potassium chloride was urgently given with IV infusion (15% 10 mEq/h for about 12 hours) judged with serial potassium level. Standard anti-ischemic drugs (Aspirin 75 mg tablet; OD, nitroglycerin retard (2.5 capsules; BID), atenolol (50 mg tablet; OD) and atorvastatin (20 mg tablet; OD) were given to the case for about 3 days then discharged with no any problem.

Discussion

Overview: An elderly parkinsonian patient presented with ventricular tachycardia with prolonged QT in the presence of hypokalemia and HTN with a history of IHD. I can't compare the current case with similar conditions. There are no similar or known cases with the same management for near comparison. The primary objective: for my case study was the presence of ischemic VT with prolonged QT in the presence of hypokalemia and HTN. Hypokalemia is a precipitator for both VT and QT-prolongation. The secondary objective for my case study was the dramatic response of ischemic VT and QT-prolongation to NTG.

Limitations of the study: There are no known limitations in the study. But, contraindications of NTG like hypotension, and pulseless VT are possible limitations.

Conclusion

My study report regards the ischemic VT and QT-prolongation responsive to NTG IVI. Testing the dramatic response of NTG IVI on ischemic VT and QT-prolongation to differentiate the ischemic cause from the others. Regards the dramatic response of ischemic VT and QT-prolongation to nitroglycerin, it may be used therapeutically and diagnostically as a new AAD in like-these cases. Nitroglycerin may have an indirect effect through increasing myocardial perfusion not due to documented clear antiarrhythmic effect. This test may decrease the overload uses of AADs and their adverse effects. It is recommended to widening the research area in the therapeutic effects of NTG on ischemic non-VT and QT-prolongation. Regards the dramatic response of ischemic VT and QT-prolongation to NTG, it may be used as a new therapeutic and diagnostic AAD in like-these cases.

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