

A Case of Chronic Alcoholism and Torsades de Pointes

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Abstract Torsades de pointes is a form of polymorphic ventricular tachycardia due to prolonged ventricular repolarization represented by a prolonged QT interval on electrocardiogram. Common causes of QT prolongation are usually considered in the clinical setting, such as electrolyte imbalances, medication side effects, congenital syndromes. As recent research has proposed, long-term excessive alcohol use may also be a contributing factor. The effects of alcohol on the heart have been studied extensively, showing that chronic exposure leads to cellular events and structural changes in the heart that may ultimately result in adverse clinical events. This case report presents a patient with a history of chronic alcoholism that experienced torsades de pointes and sudden cardiac death during hospitalization for a severe chronic obstructive pulmonary disease exacerbation. She was given macrolide antibiotics despite a prolonged QTc on electrocardiogram with a history of prior syncopal events. While macrolide antibiotics are known to cause QTc prolongation, chronic alcohol use has also been shown to be associated with QT prolongation. Moreover, QT prolongation may be a predictor of sudden cardiac death in alcoholics. In addition to common medication class side effects, the effects of commonly abused and readily accessible substances such as alcohol, should also be taken into consideration.

Keywords: torsades de pointes, prolonged QT interval, alcoholism, sudden cardiac death

Cite This Article: Daniel C. Sacher, "A Case of Chronic Alcoholism and Torsades de Pointes." *American Journal of Medical Case Reports*, vol. 6, no. 6 (2018): 117-120. doi: 10.12691/ajmcr-6-6-5.

1. Introduction

According to the 2015 National Survey on Drug Use and Health, 15.1 million adults in the United States had alcohol use disorder, 9.8 million men and 5.3 million women [1]. The adverse consequences of excessive and long-term alcohol exposure on the heart are well known, resulting in myocyte vacuolization, mitochondrial abnormalities, and myocardial fibrosis at the cellular level [2]. Acute alcohol ingestion can lead to supraventricular and ventricular arrhythmias, such as atrial fibrillation, premature ventricular complexes, ventricular tachycardia, and atrioventricular block [3]. Chronic excessive alcohol consumption has potentially toxic irreversible effects leading to cardiac dysfunction and ultimately alcoholic cardiomyopathy [4]. A recent population study showed that heavy alcohol consumption was associated with longer QTc intervals on electrocardiogram (ECG) [5], a known risk factor for developing a form of polymorphic ventricular tachycardia called torsades de pointes (TdP) [6].

2. Case Presentation

The patient of this report is a 46-year-old female with a past medical history of chronic obstructive pulmonary disease (COPD), active tobacco and daily alcohol use, who presented to the Emergency Room (ER) with a chief complaint of worsening shortness of breath, dry cough,

and rhinorrhea over the past 3 days. She described her symptoms as similar to previous COPD exacerbations, but unable to achieve relief from her home albuterol rescue inhaler. She denied any fevers, chills, sick contacts, chest pain, palpitations, abdominal pain, lightheadedness, or pre-syncope. She reported drinking approximately twenty-four 12-ounce cans of beer daily, her last drink being on the morning of admission.

On examination the patient was tachycardic but normotensive, afebrile with diffuse scattered expiratory wheezes heard over bilateral lung fields. She was somnolent and subsequently found to be hypoxic on room air with a pulse oximetry reading of 87%. Complete blood count with differential, comprehensive metabolic panel, urinalysis, and chest radiograph were all unremarkable. ECG showed sinus tachycardia to 101 beats per minute (bpm), normal axis, and normal intervals with a QTc of 493 milliseconds (ms). Arterial blood gas (ABG) showed an acute respiratory acidosis with significant hypercarbia, and the patient was placed on continuous bilevel inspiratory positive airway pressure (BiPAP). She received multiple albuterol-ipratropium treatments and steroids were given. Repeat ABG showed worsening hypercarbia despite these interventions, so she was admitted to the Progressive Care Unit for further monitoring.

The patient's ethanol level resulted at 142, alcohol withdrawal monitoring was started, she was given thiamine, folate, multivitamins, and intravenous maintenance fluids. She was started on methylprednisolone, a course of azithromycin, and continued on her home medications of inhaled budesonide-formoterol and fluticasone-salmeterol. Upon re-evaluation the next morning, she was awake and

alert, less somnolent and off BiPAP. She was tolerating room air with a pulse oximetry of 96-97% and reported experiencing near complete resolution of her respiratory symptoms. A morning chest radiograph showed no acute pathology, and the decision was made to discontinue intravenous steroids. She exhibited no signs of alcohol withdrawal, and the plan was to transfer her to the general medical floors.

Shortly before transfer, nursing was alerted by telemetry that the patient was in ventricular tachycardia. At bedside patient was found in the fetal position, unresponsive to sternal rub and verbal commands. Pulseless electrical activity was detected with telemetry showing a rapid ventricular tachycardia (VT) to 180 bpm. Cardiopulmonary resuscitation was immediately started and return of spontaneous circulation and regular sinus rhythm was achieved after approximately three minutes of chest compressions without electrical shocks or medications being administered. Stat labs showed a mild hyponatremia of 132, normal ABG, potassium of 3.7, magnesium of 2.1,

with all other lab values unremarkable. The patient regained full consciousness without any memory of the event.

A 12 lead ECG obtained after cardiac arrest showed a ventricular rate of 69 bpm, normal sinus rhythm, with QTc 554 ms by Bazett's formula (Figure 1). An ECG obtained that same morning showed a prolonged QTc of 518 ms. The patient had received her second dose of azithromycin that day, for a total of 1000 milligrams since admission. Upon further review of telemetry, TdP was discovered (Figure 2). Azithromycin was promptly discontinued, and she was transferred to the Cardiac Care Unit (CCU) for further care.

During her stay in the CCU there were no significant events, few premature ventricular complexes were seen on telemetry, and serial ECGs showed her QTc subsequently decreased to 460-480 ms. An Echocardiogram was performed that showed mild global hypokinesis of the left ventricle, mildly reduced left ventricular systolic function, an ejection fraction of 45-50%, grade one diastolic dysfunction, and trace mitral and tricuspid regurgitation.

Vent. rate	69 bpm	Normal sinus rhythm
PR interval	164 ms	Prolonged QT
QRS duration	88 ms	Abnormal ECG
QT/QTc	520/557 ms	
P-R-T axes	74 16 32	

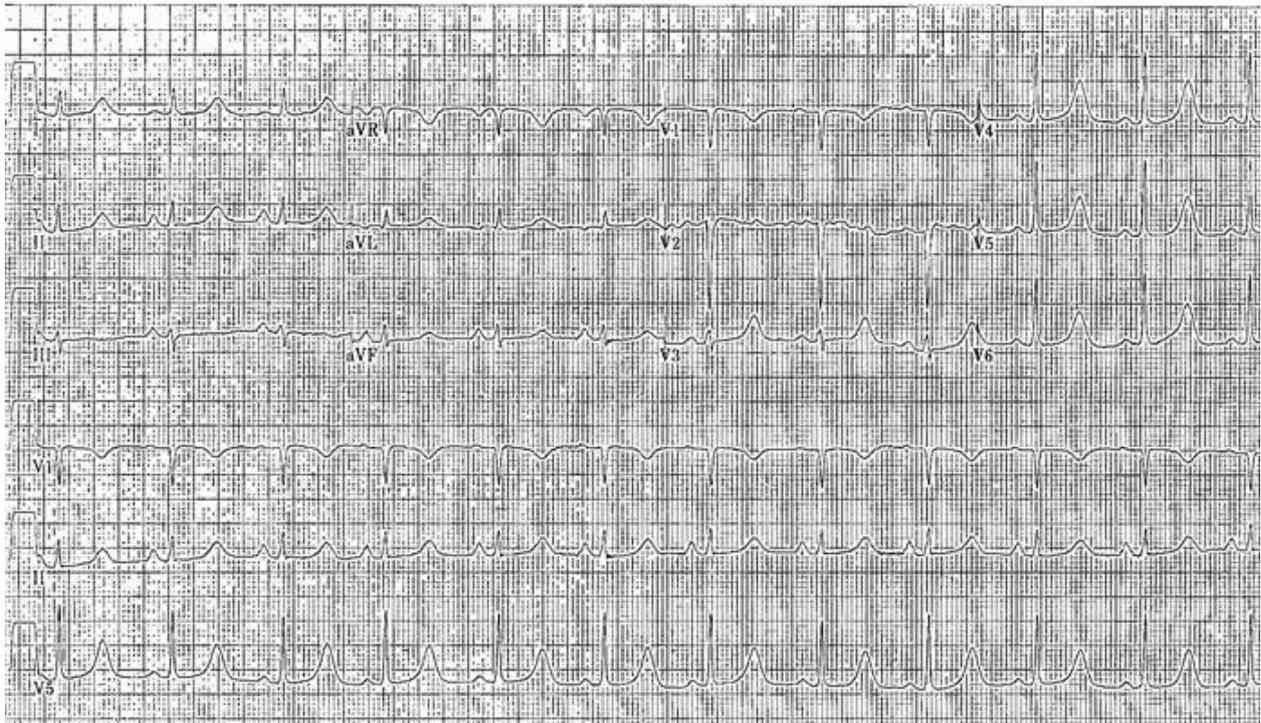


Figure 1.

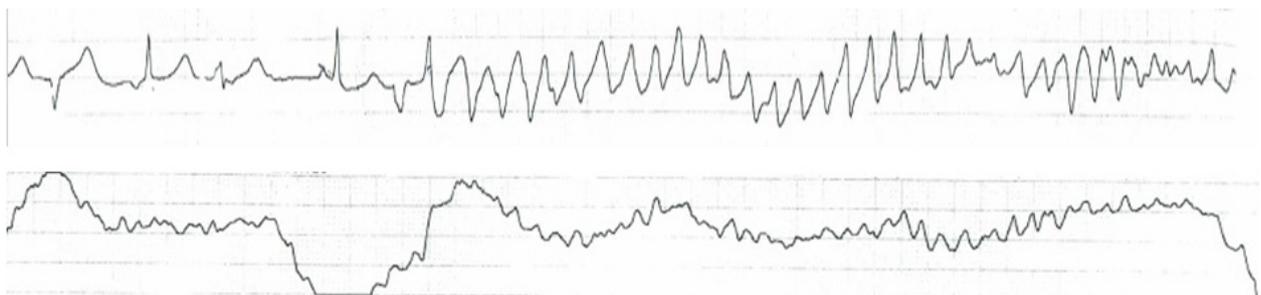


Figure 2.

The patient was transferred to the general medical floor in stable condition. She was seen by Cardiac Electrophysiology who recommended a left heart catheterization (LHC), followed by an Implantable Cardioverter Defibrillator (ICD) for secondary prevention of sudden cardiac death (SCD). She had a LHC that revealed no significant coronary artery disease, and the next day underwent successful placement of a dual-chamber ICD. She was started on metoprolol succinate daily and scheduled for outpatient follow up with Electrophysiology. All home medications with the potential to cause QT prolongation were discontinued, and she was advised to avoid any potential QT prolonging medications in the future.

Further investigation into the patient's medical history revealed that she had previously visited the ER two and a half weeks earlier after experiencing a syncopal episode while at work. She reported that she had lost consciousness without any memory of the event. The patient also recalled a similar syncopal event that had occurred about six months prior. At this ER visit her ECG showed a mildly prolonged QTc of 480 ms, ethanol level was zero and patient reported no alcohol use that day. She was held for brief observation, did not display seizure activity, no arrhythmias were recorded, and was discharged home. She denied any knowledge of a family history of SCD or arrhythmias. Of note, older ECGs from a previous admission for COPD exacerbation a few years prior showed a prolonged QT, with an average QTc of 490 ms.

3. Discussion

A prolonged QT interval can be acquired either from drugs or electrolyte imbalances, or congenital in origin, such as in Long QT syndrome. In adults, women have a longer QTc with a normal range of 450-470 ms, while a normal QTc in men is considered less than or equal to 450 ms [7]. TdP is a type of polymorphic VT that is the result of prolonged ventricular repolarization, represented by a prolonged QT interval on ECG. Polymorphic VT is defined as a ventricular rhythm faster than 100 bpm with unique QRS complexes that change with each beat due to a continuously changing ventricular activation sequence. TdP is a distinctive pattern of rapid and irregular QRS complexes that twist around an isoelectric baseline as the mean QRS axis changes. This arrhythmia may eventually degenerate into ventricular fibrillation but is usually of short duration and non-sustained. TdP has a strong association with electrolyte imbalances, such as hypokalemia, hypomagnesemia, or hypocalcemia, as well as certain medications that delay ventricular repolarization. When QT prolongation resulting in TdP is observed any underlying causes should be thoroughly investigated and swiftly corrected [6,8].

There are three main types of congenital Long QT syndromes differentiated by gene mutations in cardiac potassium or sodium channels, each with different triggers, ECG characteristics, and mortality. In individuals without a congenital Long QT syndrome, many researchers have suggested that QT prolongation is likely a gene-environment interaction, and not purely either acquired or genetic [9,10]. Many patients without congenital long QT syndrome are

given known QT prolonging medications and never develop a prolonged QT as a result or may develop a prolonged QT but never experience TdP. This observation that some individuals are more prone to develop a prolonged QT suggests the possibility of clinically silent gene mutations, or an underlying genetic predisposition for QT prolongation after exposure to QT prolonging agents. Predisposing factors to QT prolongation resulting in a higher risk of TdP are female gender, hypertension, bradyarrhythmia's, electrolyte abnormalities, left ventricular hypertrophy, reduced left ventricular ejection fraction, paroxysmal atrial tachyarrhythmia's, ischemia, and advanced greater than 60 years old [10,11,12].

Many medications are known to cause acquired long QT syndromes, including Class 1A (disopyramide, procainamide), Class IC (flecainide) and Class III (sotalol, amiodarone) antiarrhythmics, antibiotics (notably macrolides, quinine, pentamidine, trimethoprim-sulfamethoxazole), histamine receptor antagonists, certain serotonin receptor antagonists and inhibitors, cholinergic antagonists, certain antipsychotics (haloperidol, chlorpromazine and other phenothiazines) and antidepressants (amitriptyline and other tricyclic antidepressants), and poisons like arsenic and organophosphates [6,10,11]. Non-medication related causes include bradyarrhythmia's such as complete atrioventricular block or transient bradycardia such as during adenosine injection. Some states of starvation can cause long QT, such as anorexia nervosa, or surgical changes like gastrectomy and ileocecal bypass, all resulting in sustained electrolyte abnormalities. Nervous system injury has also been shown to cause a prolonged QTc interval, seen in spontaneous subarachnoid hemorrhage as well as in brainstem hemorrhage and left thalamic hematomas [10,13].

Research has demonstrated that alcohol disrupts transport of sodium and potassium ions across cell membranes, disrupting cardiac depolarization and repolarization, causing T wave abnormalities, prolongation of the PR interval, and frequently observed QT prolongation on ECG [14,15,16]. A recent published population study that focused on heavy drinkers, especially women who drink greater than one drink (15 grams of ethanol) a day showed that they had longer QTc intervals compared to nondrinkers. This study concluded that heavy alcohol consumption is a risk factor for a prolonged QTc interval [5]. Another study looking at the relationship between QT prolongation, alcoholic liver disease, and SCD demonstrated that chronic alcohol intake is associated with QT prolongation, and that QT prolongation may predict the risk of SCD in chronic alcoholics [17]. Exactly how alcohol influences QT prolongation remains unclear. It may be related to direct alcoholic myocardial damage, stimulation of catecholamine secretion from the adrenal medulla, alteration of cell membrane permeability, or by directly inhibiting the myocyte's NaK-ATPase [15,18,19].

4. Conclusion

The patient of this case report had over a 30-year history of heavy and excessive alcohol abuse with a history of prolonged QTc on ECG and two previous syncopal events. The azithromycin she was given, in

conjunction with her prolonged QTc at baseline was likely the inciting factor that led to torsades de pointes and sudden cardiac death. It should be taken into consideration, as discussed above, that her heavy and consistent alcohol use may have also contributed to prolongation of her QT interval putting her at higher risk for sudden cardiac death. Furthermore, future considerations should be made to prevent the recurrence of similar events in the future. In cases where patients present with alcohol intoxication, or have a history of chronic alcoholism, it would be prudent to review current and past electrocardiograms for any abnormalities, and if a prolonged QTc interval is present, to consider avoiding any QT prolonging medications during their hospitalization.

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