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# Long QT Syndrome Masquerading as Seizure-like Episodes : A Case Report

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## ABSTRACT

Long QT Syndrome (LQTS) is a cardiac channelopathy characterized by delayed myocardial repolarization, predisposing patients to torsades de Pointes (TdP), ventricular fibrillation (VF), and sudden cardiac death, and it is frequently misdiagnosed as a neurological disorder due to seizure-like presentations. The objective of this case study is to illustrate the diagnostic challenges in differentiating cardiac arrhythmias from neurological events and to emphasize the importance of early electrocardiographic (ECG) evaluation in patients with unexplained syncope or convulsive episodes. A detailed clinical review was conducted of a 30-year-old woman admitted for an ulcerative colitis flare who developed recurrent seizure-like episodes during hospitalization. Diagnostic evaluation included serial ECGs, telemetry monitoring, neurological assessment, laboratory investigations, and review of QT-prolonging medications. Despite correction of metabolic abnormalities and adjustment of antiepileptic and psychotropic medications, her episodes persisted. A prolonged QTc interval was identified, and the patient subsequently experienced VF requiring resuscitation. Telemetry confirmed TdP, and retrospective history revealed prior stress-related syncopal episodes, suggesting a congenital predisposition. Management involved electrolyte optimization, beta-blocker therapy, magnesium and lidocaine administration, and implantation of a dual-chamber cardioverter-defibrillator (ICD) for secondary prevention. The patient demonstrated clinical stability on follow-up with no recurrent arrhythmias or device shocks. This case highlights the need for heightened clinical suspicion for LQTS in seizure-like presentations that lack typical postictal features and do not respond to neurological treatment. It underscores the value of early ECG screening, comprehensive history-taking, and multidisciplinary management, as timely recognition and appropriate therapy are essential to preventing life-threatening arrhythmias and improving long-term outcomes.

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## 1. Introduction

Long QT syndrome (LQTS) is a cardiac channelopathy characterized by delayed myocardial repolarization, predisposing affected individuals to malignant ventricular arrhythmias, syncope, and sudden cardiac death [1]. On electrocardiography (ECG), it manifests as a prolonged QT interval with associated T-wave abnormalities, including notched, broad-based or flattened waves, which may precipitate polymorphic ventricular tachycardia such as Torsades de Pointes (TdP) [2]. Although LQTS is considered an uncommon condition, its clinical significance lies in its potential lethality and its strong genetic basis, with approximately 80% of cases linked to identifiable autosomal dominant mutations in cardiac ion channel genes [3,4].

Despite established diagnostic criteria, LQTS continues to be underrecognized in clinical practice. It's typical symptoms including transient loss of consciousness, episodic collapse, or seizure-like activity frequently overlap with neurological disorders such as epilepsy, contributing to diagnostic delays and inappropriate treatment. Case reports and observational studies have demonstrated that individuals with congenital LQTS are often misdiagnosed with seizure disorders, particularly when interictal ECGs are not obtained or when symptoms are attributed to neurological or metabolic factors [5]. These diagnostic challenges are further compounded by the potential for QT prolongation to be exacerbated by medications, electrolyte abnormalities, or physiological stressors, which may obscure the underlying congenital abnormality. Consequently, many patients remain undiagnosed until they experience a significant arrhythmic event, such as TdP or ventricular fibrillation (VF), highlighting a persistent gap in clinical awareness and risk stratification [6].

The objective of this case report is to demonstrate the diagnostic complexity of LQTS presenting as recurrent seizure-like episodes, highlight the importance of integrating ECG evaluation early in the assessment of unexplained syncope or convulsive events, and emphasize the role of multidisciplinary collaboration in achieving accurate and timely diagnosis.

## 2. Case Report

A 30-year-old pleasant lady with a 7-year history of severe ulcerative colitis (UC) on infliximab, complicated by recurrent flares and prior CMV colitis, as well as depression and a remote seizure disorder managed with levetiracetam, presented to our emergency department, in September 2025, with a 5-day history of worsening bloody diarrhea, abdominal pain and weight loss. She was admitted with an acute UC flare and concern for CMV reactivation. Initial management included intravenous corticosteroids, valganciclovir, fluid resuscitation and supportive care.

### 2.1. Hospital course

The patient experienced a brief convulsive seizure the following morning, lasting less than two minutes, which resolved spontaneously without any residual neurological effects. Her routine electroencephalogram (EEG) was normal. Laboratory tests showed mild hypokalemia (3.3 mmol/L), which was corrected. The EEG revealed a 510-ms QT prolongation, as shown in Figure A1, attributed to her medications (escitalopram and levetiracetam oxalate), which were subsequently discontinued. The neurology team diagnosed her with syncope, taking into account severe dehydration and hypotension. Her husband noted that this seizure differed from her usual episodes due to the absence of post-seizure confusion. Therefore, the neurology department maintained her levetiracetam dosage at 750 mg twice daily.

The next day, she experienced another seizure-like episode lasting 2 minutes with mild post-ictal lethargy but no focal deficits. The event occurred shortly after receiving tramadol for abdominal pain, which is known to lower seizure threshold, and her levetiracetam dose was

increased to 1000 mg twice daily as per neurology recommendations. On day 3, she had another brief event lasting 30 seconds with mild post-ictal drowsiness witnessed by her husband. A temporal pattern emerged, with all events occurring at the time of valganciclovir administration, and the medication was held over concern for valganciclovir-induced lowering of seizure threshold.

### 2.2. ICU admission

Over the next few days, she had two additional brief seizure-like episodes, each lasting less than 30 seconds, with upper extremity stiffness but no eye rolling or tongue biting. She was placed on a non-rebreather mask and transferred to the neurological intensive care unit (ICU) for continuous EEG monitoring. While in the ICU, she experienced another seizure-like episode that progressed to ventricular fibrillation (VF) arrest. Cardiopulmonary resuscitation (CPR) was initiated and return of spontaneous circulation (ROSC) was achieved after one cycle with a single defibrillation. Amiodarone was administered. Post-arrest ECG showed a QTc of 398ms and laboratory studies revealed hypokalemia (3.2mmol/L), which was corrected. Continuous EEG did not show any epileptiform activity. In true epileptic seizures, EEG typically demonstrates topographical epileptiform discharges originating from focal or generalized cortical regions; however, in episodes caused by cardiac arrhythmias, EEG usually shows non-topographical changes such as generalized slowing or attenuation due to transient cerebral hypoperfusion. This distinction has been described in the literature and assists in differentiating convulsive syncope from epileptic seizures [7,8].

On day 6, she developed recurrent syncopal episodes with posturing. ECG demonstrated a prolonged QTc greater than 570ms and telemetry captured TdP, which reverted to sinus rhythm following brief CPR. She was treated with magnesium sulfate and lidocaine infusions. An echocardiogram revealed global hypokinesia with an ejection fraction of 40%. Additional history-taking revealed prior stress-related syncopal episodes in her twenties and a remote diagnosis of possible postpartum cardiomyopathy. Continuous monitoring of the corrected QT interval is essential in patients presenting with recurrent syncope, seizure-like episodes, or exposure to QT-prolonging medications, as dynamic QTc changes help identify those at risk of malignant ventricular arrhythmias. Prolongation of QTc increases susceptibility to early after depolarizations and the development of TdP, particularly in the presence of electrolyte disturbances or bradycardia. Acute management focuses on aggressive correction of hypokalemia and hypomagnesemia, discontinuation of QT-prolonging medications, administration of intravenous magnesium sulfate for TdP, and use of isoproterenol or temporary pacing for bradycardia-induced TdP [9, 10]. Recognition and treatment of recurrent or refractory QT prolongation are crucial to prevent progression to VF and sudden cardiac death. Based on these findings, cardiology initiated nadolol while tapering lidocaine.

During her ICU stay, she developed persistent bradycardia of approximately 50 beats per minute. Isoproterenol infusion was started to increase her heart rate and nadolol was temporarily held. In hindsight, her seizure-like episodes were recognized as ventricular tachycardia (VT) events precipitated by QT prolongation, raising strong suspicion for congenital long QT syndrome (LQTS) given the documented prolonged QTc and TdP.

On day 9, she experienced VF arrest requiring CPR for two cycles before achieving ROSC. She received lidocaine and midazolam, with electrolyte correction. She underwent an urgent dual-chamber implantable cardioverter-defibrillator (ICD) insertion for secondary prevention. Post-procedure, she remained hemodynamically stable on lidocaine infusion.

Over the next several days, she was successfully weaned off lidocaine, and her QTc stabilized around the 510ms range. Her antidepressants were adjusted to alternatives with a lower risk of QT

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prolongation following psychiatry review. She was transferred to the general ward and discharged home on day 16 as shown in Figure A2.

### 2.3. Follow Up

At her initial follow-up, she was stable on nadolol with no major complaints. Two months after discharge, she was hospitalized abroad for severe VT and VF episodes. Her antiepileptic therapy was subsequently tapered and possibly switched due to concerns about its role in ventricular arrhythmias. At her six- and twelve-months' follow-ups, she was asymptomatic, performing her usual daily activities with no further VT or VF episodes nor ICD discharges.

## 3. Results and Discussion

LQTS is a genetic condition that disrupts myocardial repolarization manifesting as a prolonged QT interval on an ECG and predisposing patients, particularly in young individuals, to life-threatening ventricular arrhythmias such as torsades de pointes and ventricular fibrillation [11]. The condition can be congenital or acquired, or may represent interaction between underlying genetic susceptibility and external triggers, including medications or electrolyte abnormalities [12]. Patients typically present with syncope, seizure-like episodes, cardiac arrest or sudden cardiac death. Because LQTS-related events often involve transient loss of consciousness with convulsive features, they are frequently misdiagnosed as neurological disorders, particularly epilepsy, resulting in substantial delays in diagnosis [13]. This diagnostic overlap has been well described, as convulsive syncope due to ventricular arrhythmias can closely mimic epileptic seizures and contribute to misdiagnosis [14].

In our case, the absence of classic postictal symptoms such as tongue biting, incontinence, or focal neurological deficits raised suspicion of a non-epileptic cause. The diagnostic challenge was complicated by several confounding factors including severe dehydration, hypotension, hypokalemia, and the use of medications known to lower seizure threshold or prolong the QT interval such as tramadol, escitalopram, and levetiracetam. Psychiatric medications, particularly selective serotonin reuptake inhibitors, have been recognized as contributors to acquired QT prolongation in susceptible individuals [15]. Additionally, chronic inflammatory disorders such as ulcerative colitis may independently increase susceptibility to seizure-like activity due to neuroimmune activation and oxidative stress, as demonstrated in experimental and clinical studies [16]. These factors have been described as potential triggers in patients with latent congenital LQTS. Initially, convulsive syncope remained the leading diagnosis; however, the persistence of recurrent episodes despite electrolyte correction and medication adjustments suggested an alternative explanation.

In evaluating recurrent seizure-like events, it is also important to consider psychogenic non-epileptic seizures, particularly in individuals with psychological comorbidities or chronic systemic illness. PNES frequently coexist with epilepsy and may resemble true seizures clinically, creating diagnostic uncertainty [17]. However, in patients with underlying cardiac disease, seizure-like episodes may instead represent transient cerebral hypoperfusion due to malignant arrhythmias. Unlike epileptic seizures, which show epileptiform discharges on electroencephalography, arrhythmia-induced convulsive syncope produces non-specific generalized slowing. Prior reports have documented patients initially labeled as having epilepsy or pseudo-seizures who were later found to have long QT syndrome or other ventricular arrhythmias as the true underlying cause of their events [18]. Therefore, careful distinction among epileptic seizures, psychogenic episodes, and arrhythmia-induced convulsive events is essential, as misclassification may delay recognition of potentially life-threatening cardiac pathology.

Valganciclovir administration also contributed to diagnostic complexity, as it coincided with several events and has been associated with lowering seizure threshold. Once QT prolongation was identified on ECG, medication-induced QTc prolongation was suspected, prompting systematic review of all QT-prolonging drugs. Telemetry confirmation of TdP, along with recurrent VF, established the diagnosis of LQTS. Retrospective history revealed stress-induced syncope and a prior possible postpartum cardiomyopathy, both of which may represent early manifestations of congenital LQTS. Prior case reports have described similar presentations where seizure-like activity was the initial clinical manifestation of undiagnosed LQTS [19].

Management of LQTS consists of pharmacological and non-pharmacological therapies. Beta-blockers such as nadolol remain first-line therapy due to their protective effect against adrenergically mediated arrhythmias [20]. High-risk patients—those with QTc greater than 500ms, recurrent syncope, or specific genotypes (LQT2 or LQT3)—may require additional interventions such as acute antiarrhythmic therapy. In our case, lidocaine infusion was used temporarily for ventricular arrhythmia suppression. The patient ultimately underwent implantable cardioverter-defibrillator (ICD) placement for secondary prevention, which is recommended in guidelines for individuals with documented TdP or VF [21]. Genetic counselling and testing are also essential to confirm suspected congenital LQTS and to enable screening of first-degree relatives [22].

## 4. Conclusion

LQTS is a disorder of myocardial repolarization that can present with syncope, seizure-like activity, or cardiac arrest, making early recognition essential to prevent life-threatening arrhythmias. This case illustrates the diagnostic complexity encountered when cardiac arrhythmias mimic neurological or psychogenic conditions, particularly in patients with chronic inflammatory disease and concurrent use of QT-prolonging medications. The absence of postictal signs, persistence of episodes despite electrolyte correction, and subsequent documentation of torsades de pointes and ventricular fibrillation were key elements that established the diagnosis. Accurate differentiation between epileptic seizures, psychogenic non-epileptic events, and arrhythmia-induced convulsive syncope is critical, as misdiagnosis may delay life-saving cardiac evaluation. Management included removal of QT-prolonging medications, beta-blocker therapy, acute antiarrhythmic support, and ultimately implantable cardioverter-defibrillator placement for secondary prevention. This case underscores the importance of early ECG assessment, thorough history-taking, and multidisciplinary collaboration to optimize outcomes and guide long-term management in patients presenting with unexplained seizure-like episodes.

**Patient Consent:** written informed consent was obtained from the patient for publication of her clinical history and case details. A signed consent form is available upon request.

**Author Contribution:** All authors contributed equally to the main contributor to this paper. All authors read and approved the final paper.

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**Conflicts of Interest:** "The authors declare no conflict of interest."

## Appendix

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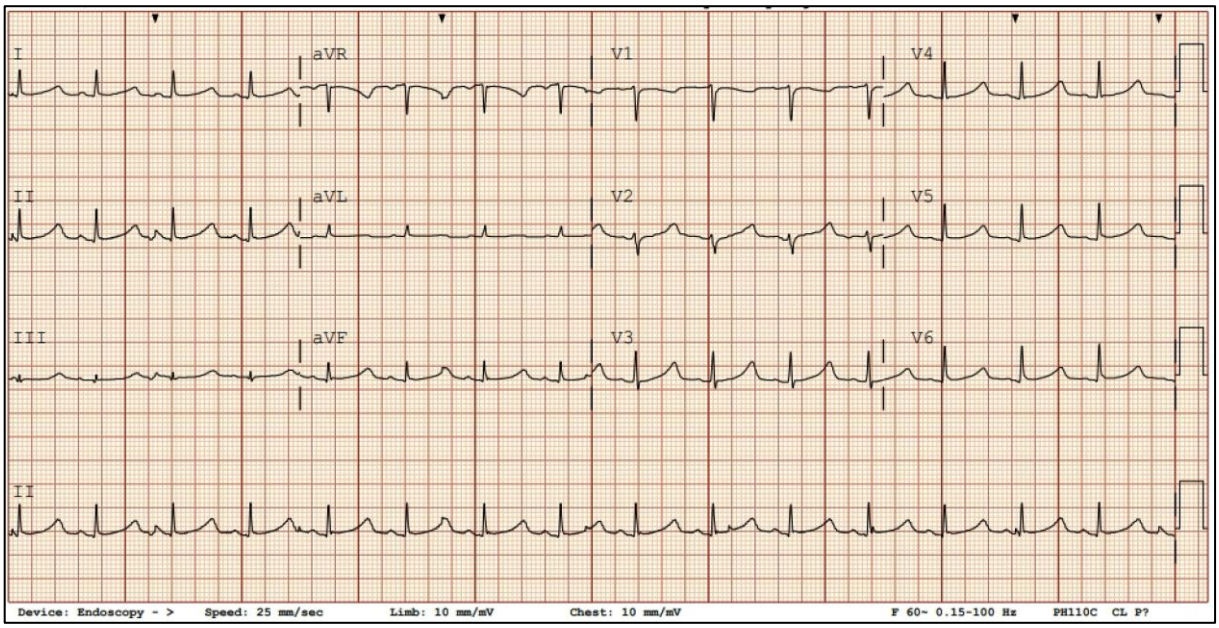


**Figure. A1**. ECG on the patient's first day of admission revealing a prolonged QTc of 510 ms

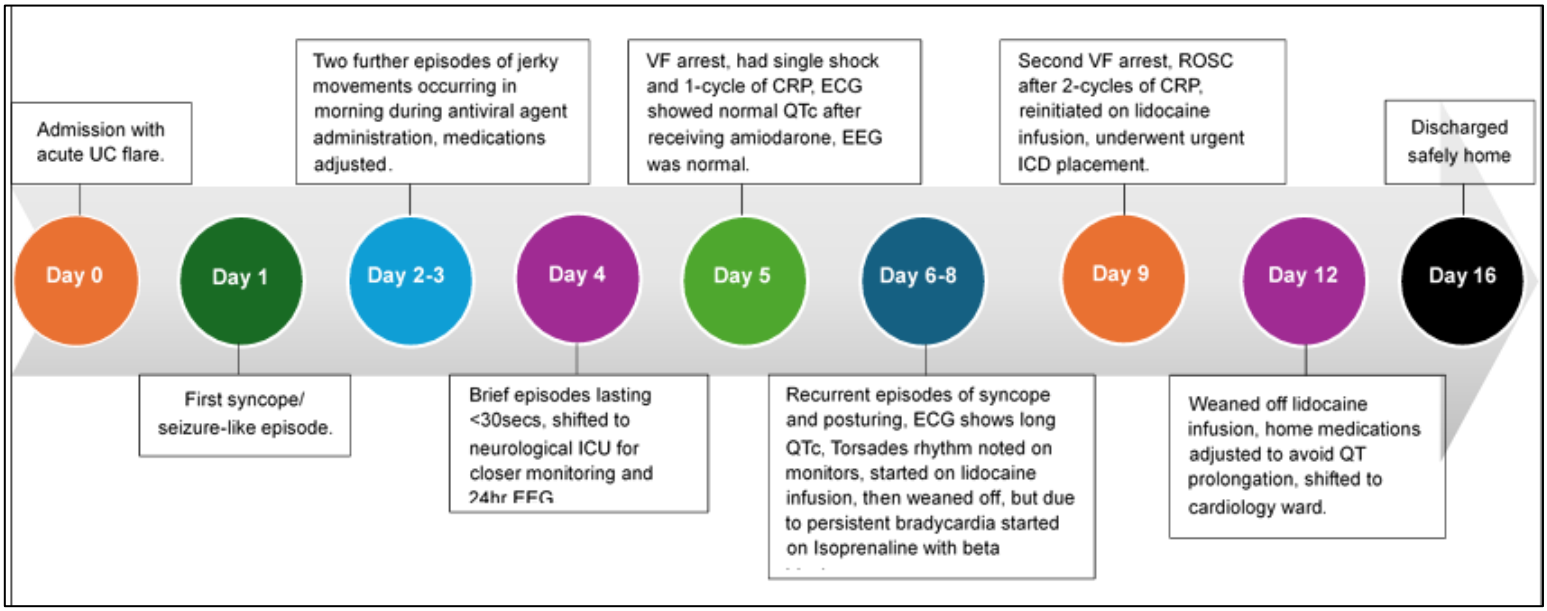


**Figure. A2**. Timeline of events during the patient's admission

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