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# Correcting the QTc and fixing torsade's, what can't steroids do!

Yasir Ahmed, Mustafeez ur Rahman, Muhammad Rafique, Sajjad Ahmad, Ghulam Mustafa Awan

## ABSTRACT

**Introduction:** Long QT syndrome (LQTS) is the congenital or acquired prolongation of the QT interval on an electrocardiogram (ECG). It is well-known that the QT interval is prolonged in adrenal insufficiency (AI) but rarely prolonged enough to cause Torsade de Pointes (TdP).

**Case Report:** Here we report a case of TdP and cardiac arrest in a patient with adrenal insufficiency. The patient had a return of spontaneous circulation after a successful cardiopulmonary resuscitation (CPR). Electrocardiograms persistently showed prolonged QT corrected for heart rate (QTc) prior to and at the time of cardiac arrest, some exceeding 600 milliseconds (ms). The QT interval improved significantly upon administration of steroids and the episodes of TdP resolved.

**Conclusion:** This case highlights the importance of recognizing adrenal insufficiency (AI) as a potentially reversible cause of prolonged QT and TdP.

**Keywords:** Adrenal insufficiency, Cardiac arrest, Torsade de pointes

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## INTRODUCTION

Electrocardiogram abnormalities in AI were reported by Sommerville in the 1950s [1, 2]. Flat T-waves, prolonged QT, low voltage, and prolonged PR or QRS interval, and depressed T waves are the usual changes seen on ECG. It is well-known that the QT interval is prolonged in AI but reports on QT sufficiently prolonged to cause TdP are rare [3].

## CASE PRESENTATION

A 64-year-old female with hypertension, diabetes mellitus type 2, hypothyroidism, sick sinus syndrome, and status post remote pacemaker placement for a bradyarrhythmia during pregnancy was electively admitted for surgical resection and mediastinal node biopsy for newly diagnosed limited-stage small cell lung cancer.

The evening before surgery, the patient was found by nursing staff to be altered and pulseless. Cardiopulmonary resuscitation (CPR) was initiated immediately. She had a return of spontaneous circulation after 2 minutes of CPR. She did not require any medications.

Telemetry showed an episode of TdP 1 to 2 minutes prior to the event (Figure 1). Metabolic and infectious workup obtained at the time of event was normal, and

cardiac markers were negative. Electrocardiogram showed normal sinus rhythm and QT prolongation with a QTc of 577 milliseconds (ms). Multiple additional episodes of TdP were recorded, all of them self-limiting. Electrocardiograms persistently showed prolonged QTc, even recorded above 600 ms (Figure 2). Occasional episodes of hypotension, hypoglycemia, and hypokalemia were noticed, raising the suspicion of AI. Cortisol levels were reported low before and after the cosyntropin stimulation test, 2.3 and 6.9 mcg/dL respectively (normal >18–20 mcg/dL after stimulation). Adrenocorticotropic (ACTH) hormone level was <1 (normal 7.2–63.3 pg/mL). This confirmed AI.

The patient was started on intravenous steroids (hydrocortisone), and QTc improved over the next few days to 462 ms (Figure 3). Transthoracic echocardiography (TTE) showed a left ventricular ejection fraction (EF) of 25–30% and hypokinesis which improved upon administration of steroids to 60–65% in 5 days. Left heart catheterization had shown moderate non-obstructive disease.

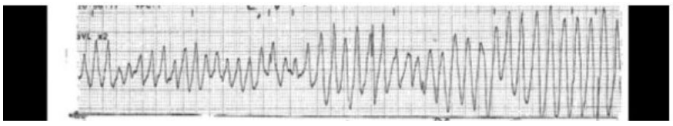

Figure 1: Telemetry strip just prior to the event, showing Torsade de pointes.

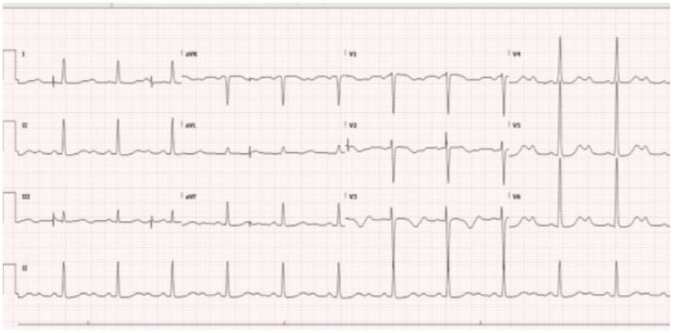

Figure 2: Normal sinus rhythm with a heart rate of 71 bpm, T-wave changes and a QTc of 602 milliseconds. Intermittent pacer spikes with non-sense or capture.

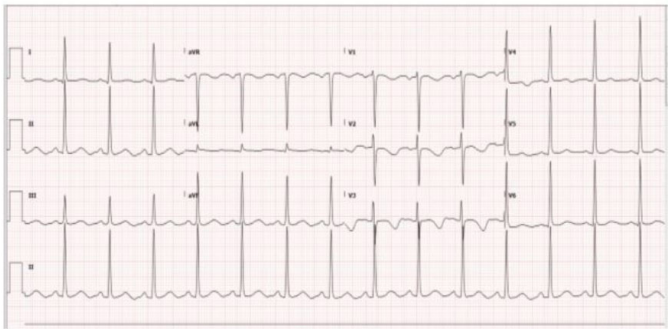

Figure 3: Normal sinus rhythm with a heart rate of 87 bpm, left ventricular hypertrophy and T-wave changes and a QTc of 462 milliseconds (improved after steroid administration).

The patient is being followed up as outpatient, maintained on a chronic steroid therapy (with oral prednisone) and no further episodes of prolonged QT on ECG or TdP reported at 18 months.

## DISCUSSION

The QT interval represents the onset of ventricular depolarization through repolarization of the cardiac myocytes [3]. QT interval is prolonged in LQTS and can be due to congenital or acquired conditions. Ion channel's dysfunctions due to genetic mutations have been shown to cause congenital LQTS [4]. Medications, electrolyte abnormalities, intracranial events, cardiac and endocrine disorders are well-known causes of acquired LQTS [5–8].

The mechanism of prolonged QT in AI is poorly understood but is believed to be related to potassium and calcium channels. Glucocorticoids were found to be responsible for the upregulation of the potassium channels in the ventricles of a rat's heart. The absence of this upregulation due to AI is thought to affect the slow inactivating potassium current, thus causing QT prolongation [9]. Rats who underwent adrenalectomy were found to have decreased the accumulation of calcium in the sarcoplasmic reticulum due to altered velocity of calcium transport across the channels, which is thought to be another mechanism responsible for QT prolongation. This effect was significantly reversed after administration of steroids [10].

Treatment with glucocorticoids results in normalization of the QT interval in these patients, as shown in several other cases. [3, 11, 12]. Patients can have associated hypothyroidism due to hypopituitarism, and the resultant severe hypothyroidism has also been found to be a cause of QT prolongation [13, 14].

## CONCLUSION

Adrenal insufficiency should be considered in differentials when working up a cause of prolonged QT and TdP, especially when patients have other features of AI present. Steroids can improve or completely resolve prolonged QT and TdP, hence recognition is very important for a potentially reversible etiology.

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## Author Contributions

Yasir Ahmed – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Interpretation of data, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Mustafeez ur Rahman – Acquisition of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Muhammad Rafique – Conception of the work, Analysis of data, Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Sajjad Ahmad – Interpretation of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Ghulam Mustafa Awan – Conception of the work, Design of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

## Guarantor of Submission

The corresponding author is the guarantor of submission.

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## Consent Statement

Written informed consent was obtained from the patient for publication of this article.

## Conflict of Interest

Authors declare no conflict of interest.

## Data Availability

All relevant data are within the paper and its Supporting Information files.