

# Acquired Complete Heart Block with Long QT Interval and Recurrent Polymorphic Ventricular Tachycardia: A Case Report

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## Abstract

We are reporting a case of acquired complete heart block and long QT interval (a dispersion of repolarization that leads to polymorphic ventricular tachycardia) that has presented with loss of conscious and proved to be due to torsade de pointes. The patient responded well to cardiac pacing and beta blocker therapy. The association of complete acquired heart block and long QT interval is quite rare.

## Keywords

Heart Block Long QT

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## 1. Introduction

Long QT syndrome (LQTS) is an uncommon cardiac disorder of repolarization leading to prolonged QT interval and T-wave abnormalities on the ECG, thus predisposing to *Torsades de Pointes* (TdP) and sudden cardiac death [1]. A study by Schwartz *et al.* [2] reported the prevalence of LQTS to be 1 in 2000 people. Pseudo 2:1 AV block when sinus intervals are shorter than the ventricular refractory period has been reported with long QT syndrome (LQTS) [2], but only few reports of acquired complete heart block, and few reports of congenital complete heart block with prolonged QT interval [3]-[6].

## 2. Case Report

We report a 29-year-old gentleman, with positive family history of sudden cardiac death, and no cardiac symptoms before, who presented to our center with syncope, his ECG showed complete heart block with an escape

1/18/2015 6:29:07 AM

Rate 88 . Age not entered, assumed to be 50 years old for purpose of ECG interpretation  
 . Accelerated junctional rhythm  
 . Ventricular tachycardia  
 . Left bundle branch block

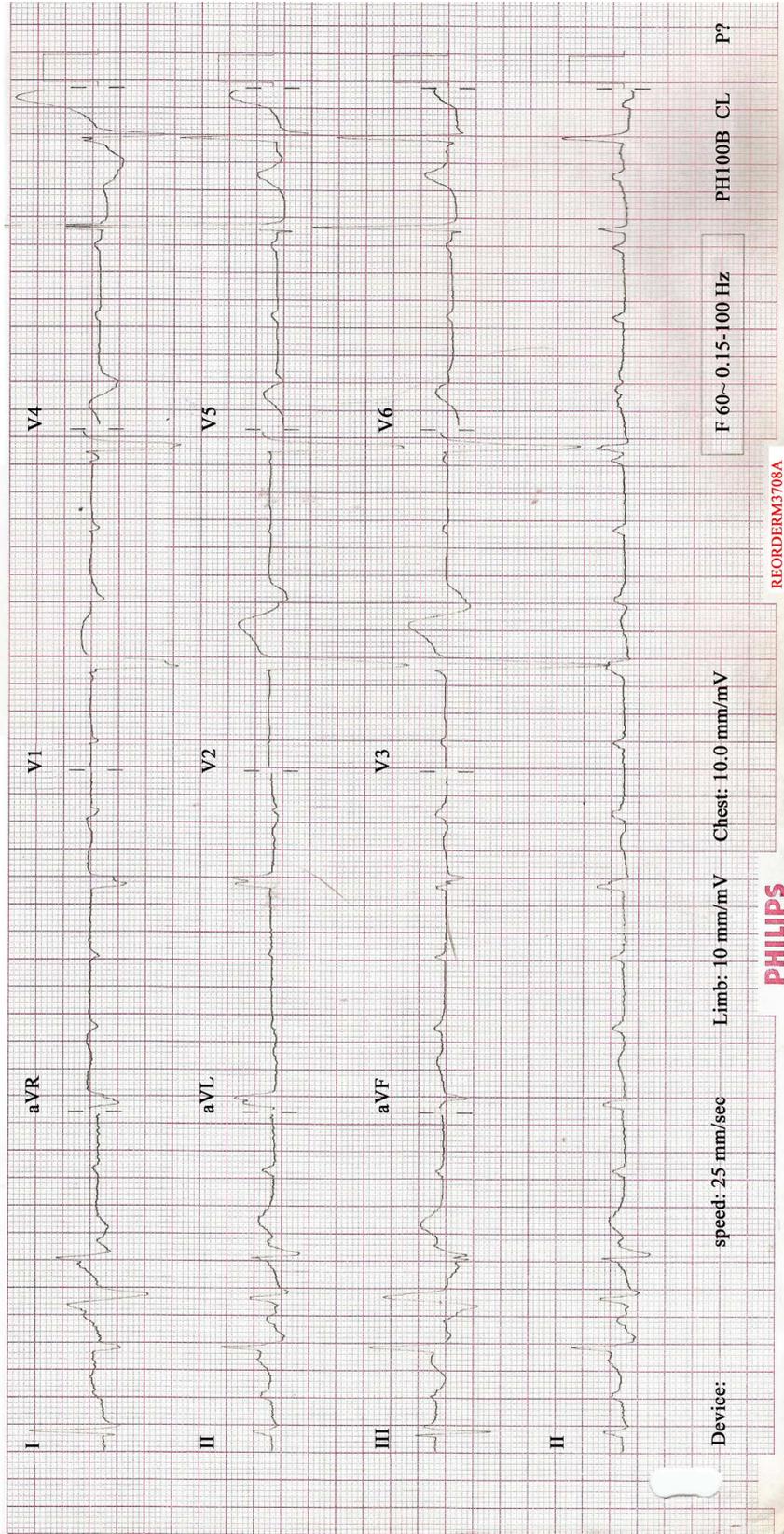
10604 58 14



--AXIS--  
 P -16  
 QRS 60  
 T 60

12 Lead; Standard Placement

Unconfirmed Diagnosis



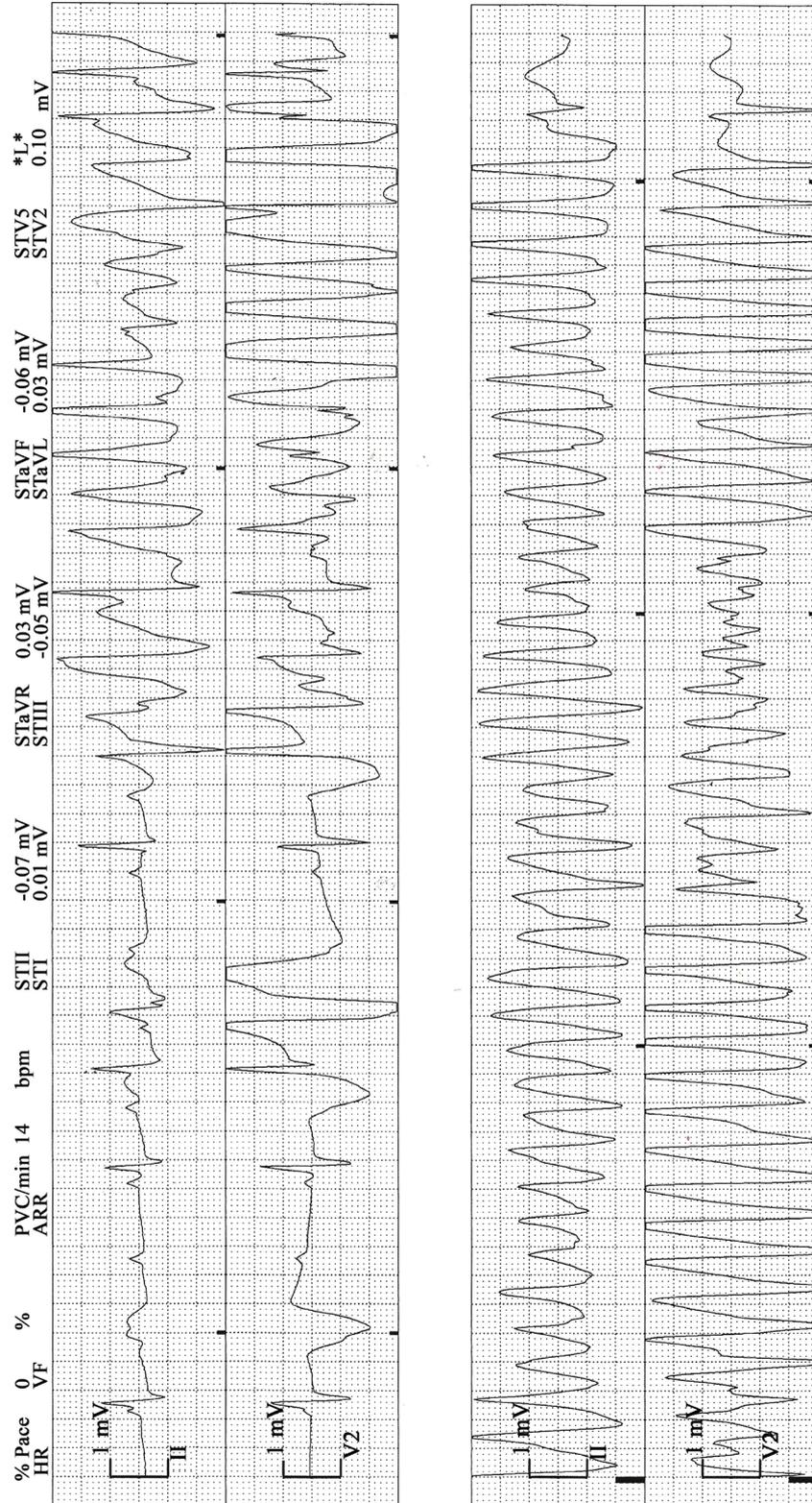
Device: speed: 25 mm/sec Limb: 10 mm/mV Chest: 10.0 mm/mV F 60-0.15-100 Hz PH100B CL P?

REORDERM3708A

PHILIPS

Figure 1. Showing complete heart block, prolonged QT interval and deep T wave inversion in pericardial leads and one spike of failed temporary pacing.

<b>Event Disclosure Strip Report</b> Delay: 10 Seconds Speed: 25mm/Second	
Patient: 29-3 Care Unit: CCUTELE Bed: T254_02 Admit Date: 18-Jan-2015	Event Time: 05:46:40 Event Date: 18-Jan-2015 VENTRICULAR FIBRILLATION! Generated: 08:55 18-Jan-2015
Patient ID: Date of Birth:	Pacer/ICD/PCD:



COMMENTS: Signature:	Date:
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Figure 2. Showing the polymorphic ventricular tachycardia induced by PVC.

1/21/2015 3:16:15 PM

Rate 119 . . . Age not entered, assumed to be 50 years old for purpose of ECG interpretation  
 PR 130 . . . Atrial-sensed ventricular-paced rhythm . . . . .ventricular pacing tracks p-waves  
 QRS 130 . . . No further analysis attempted due to paced rhythm  
 QT 372  
 QTc 524

--AXIS--  
 P 55  
 QRS -84  
 T 89  
 12 Lead; Standard Placement  
 - ABNORMAL ECG -

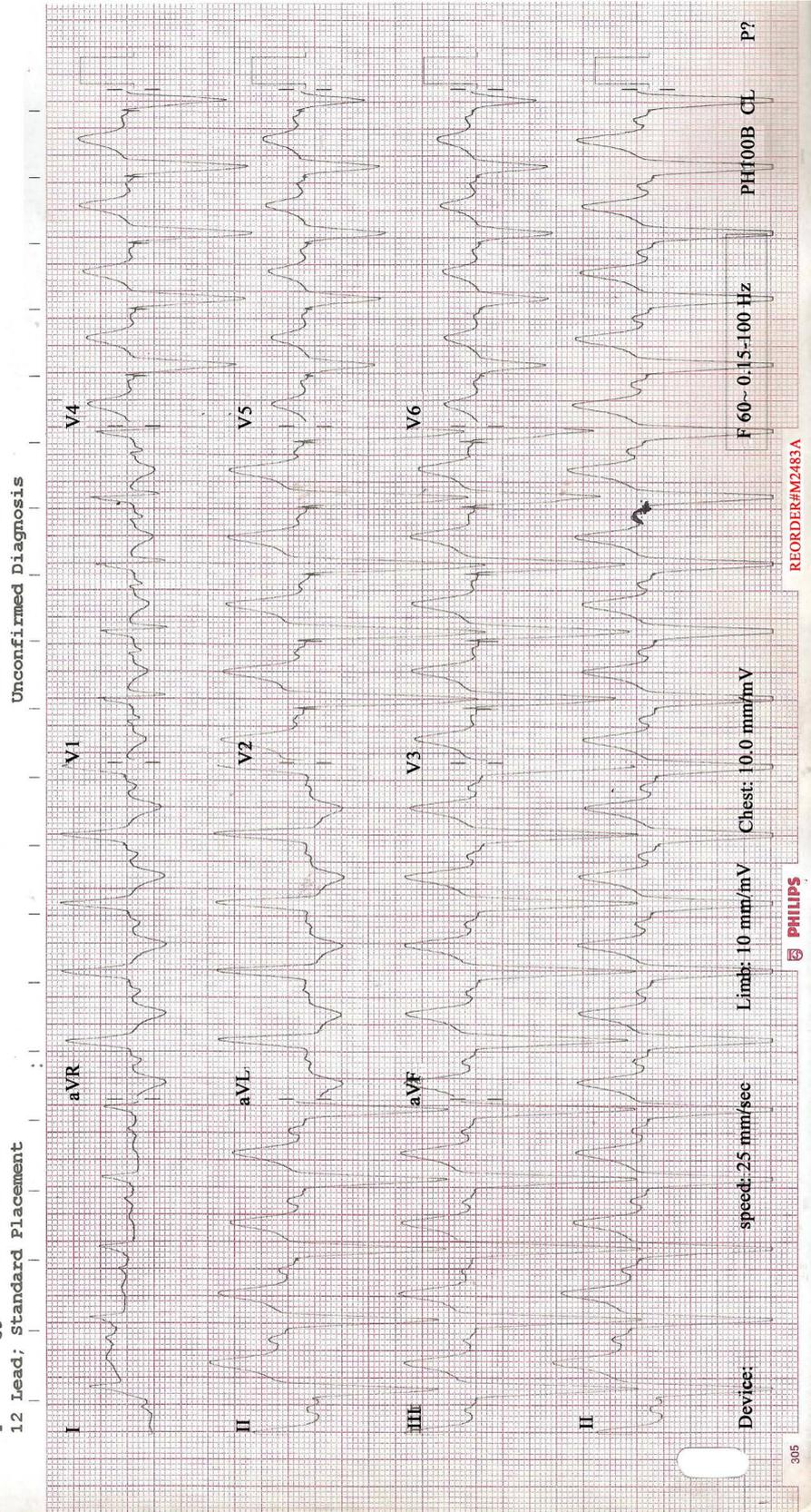


Figure 3. Showing shortening of the QT interval after pacing with complete suppression of the arrhythmia.

rhythm of wide QRS, at a rate of 35 beats per minutes, We had no previous records of the patient as he has recently arrived to the country but this was his first time to have such a complaint, inpatient work up did not reveal any reversible cause of complete heart block, his QT interval was prolonged (QT 680 ms, QTc bazett method 534 ms, QTc Hodges method 640 ms) (we used the Hodges method because the patient was tachycardiac after DDD pacing as the bazett method would overestimate the corrected QT when the patient heart rate above 110 BPM [6]), with deep T wave inversion in the precordial leads (**Figure 1**). On telemetry he started to have recurring polymorphic ventricular tachycardia which was initiated by a premature ventricular beat (**Figure 2**) and led to loss of consciousness. A temporary pacemaker was inserted which suppressed the arrhythmia and led to shortened QT. His echocardiogram, serum electrolyte including potassium and coronary angiogram were normal. A permanent dual chamber pacemaker was implanted the following day with higher basal rate (**Figure 3**) and the patient was started on beta blocker. QT and QTc were significantly reduced after permanent pacing (QT 400 ms QTc bazett 573 ms (overestimated as heart rate is 119 BPM), QTc Hodges 503 ms) [7]. He remained arrhythmia free and discharged in good condition. At six weeks follow up the patient was seen doing well, without symptoms or recorded arrhythmia, and normal pacemaker function.

### 3. Discussion

The ventricular tachyarrhythmia that underlies the cardiac events of LQTS is Torsades-de-Pointes, a curious type of ventricular tachycardia that most of the time is self-limiting and produces transient syncope but that can also degenerate into ventricular Fibrillation and cause cardiac arrest or sudden death [1]. The association of acquired complete heart block and long QT interval in literature is quite rare, only few reports about congenital complete heart block and long QT interval [3] [4]. Indik [8] had also reported a similar case study of a 65-years old female who developed dizziness and found to have complete heart block with QT interval of 800 ms and markedly abnormal T wave. She was demonstrated to have episodes of TdP on Holter monitoring. After insertion of the pacemaker, the T-waves normalized and QT interval shortened and arrhythmia suppressed. It is generally accepted that in the long QT syndrome, the irritability of the ventricle increases due to the exaggerated dispersion of ventricular repolarization and unilateral changes in sympathetic tone of the heart (functional distribution of right and left stellate innervation to the ventricles). The appearance of polymorphous ventricular tachycardia mostly of the torsade de pointes type can frequently be detected in the syndrome of QT prolongation. Contrary to congenital complete heart block, the acquired complete heart block can have more frequent attacks of loss of conscious. In our work, we went in agreement with other authors [3]-[6] that pacing shortened the QT interval and prevented the provocation of the torsade de pointes. In searching for the etiology of our condition, a study by Kurita *et al.* [8] investigated fourteen patients with complete atrioventricular block. They demonstrated that there was significant prolongation of the QT and QTc intervals among patients with complete heart block who developed TdP as compared to those who did not develop TdP:  $753 \pm 57.5$  vs  $635 \pm 78.4$  ms ( $p < 0.01$ ) and  $585 \pm 44.8$  vs  $476 \pm 58.3$  ms ( $p < 0.01$ ) which might point to the mechanism of arrhythmia in our case.

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