Journal of Arrhythmia 31 (2015) 118–120

Contents lists available at ScienceDirect

Journal of Arrhythmia

journal homepage: www.elsevier.com/locate/joa

Case Report

# QRS–ST–T triangulation with repolarization shortening as a precursor of sustained ventricular tachycardia during acute myocardial ischemia

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ARTICLE INFO

*Article history:*
Received 13 June 2014
Received in revised form
8 August 2014
Accepted 19 August 2014
Available online 20 September 2014

*Keywords:*
Ventricular arrhythmia
Myocardial ischemia
Mechanisms of re-entry
ST segment elevation
Vasospastic angina

ABSTRACT

We present segments from a 24-hour 12-lead digital Holter recording in a 48-year-old man demonstrating transient ST elevations in the inferior leads that triggered sustained ventricular tachycardia/ventricular fibrillation (VT/VF) requiring cardioversion. The onset of VT was preceded by a gradual increase in the ST with marked QRS broadening that lacked distinction between the end of the QRS and the beginning of the ST (QRS–ST–T "triangulation"), and shortening of the QT interval not caused by an increased heart rate. This is a relatively rare documentation of the mechanisms immediately triggering sustained ventricular arrhythmias during acute myocardial ischemia obtained with 12-lead ECG.



## 1. Case report

We present excerpts from a 24-hour 12-lead Holter recording acquired in a 48-year-old man during his hospitalization for an unexplained blackout. His previous medical history included repeated blackouts and exertional angina. One month earlier, he has been hospitalized for chest pain with transient ST segment elevation in the inferior leads, and he underwent percutaneous coronary angioplasty to the mid-LAD coronary artery. Subsequently, he was treated with aspirin, clopidogrel, atorvastatin, ramipril, isosorbide mononitrate, and bisoprolol.

The Holter recording documented several transient episodes of ST elevation in leads II, III, and aVF, with reciprocal ST depression in leads V5 and V6 during chest discomfort. One of the episodes triggered ventricular premature beats followed by sustained ventricular tachycardia/ventricular fibrillation (VT/VF) requiring cardioversion. Ventricular arrhythmia (VA) onset was preceded by a gradual elevation in the ST compared to previous segments (Fig. 1A) with marked QRS broadening, most pronounced in leads III and aVF (Figs. 1B–F) where the QRS–ST–T complexes immediately preceding the arrhythmia appeared "triangulated" and lacked distinction between the end of the QRS and beginning of the ST segment (Fig. 1F).

Fig. 2A presents a continuous tracing in lead III of the last 3 min preceding VA onset. Note that the shortening of the ventricular repolarization not caused by an increased heart rate is clearly revealed by the sinus complex preceding VA in the last row when compared to an earlier sinus complex (arrows). In Fig. 2B, the early (solid line) and late (dotted line) complexes are superimposed and aligned by the ascending QRS limb (arrow). Note the clear separation by $>40$ ms of the descending T wave limbs of both complexes (measured arbitrarily and marked by a horizontal dotted line).

## 2. Discussion

During the acute phase of myocardial infarction (MI), marked ST elevation with "lambda-like patterns" or monophasic QRS–ST–T complexes similar to those reported here have been strongly associated with VF [1]. Such abnormal ventricular complexes reflect the ischemia-induced slowing of intra-ventricular conduction and abnormal augmentation of repolarization dispersion, as indicated by various alterations in the shape and duration of the action potentials (AP) across the ischemic myocardium (including triangulated APs), which initiates re-entry arrhythmias [2]. These QRS–ST–T patterns ("monophasic" and "triangulated," among others) are most likely not specific for myocardial ischemia. Arrhythmogenic "lambda-like" ST elevation patterns have been reported in "atypical Brugada syndrome (BrS)" (Brugada changes in the inferior leads) and in acute MI complicated with VF [3]. Moreover, similarities exist between the mechanisms of ST elevation and VF triggers (phase 2 re-entry) during acute ischemia and BrS [4]. Similar "triangulated" (phase 3 prolongation) and

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http://dx.doi.org/10.1016/j.joa.2014.08.006
1880-4276/

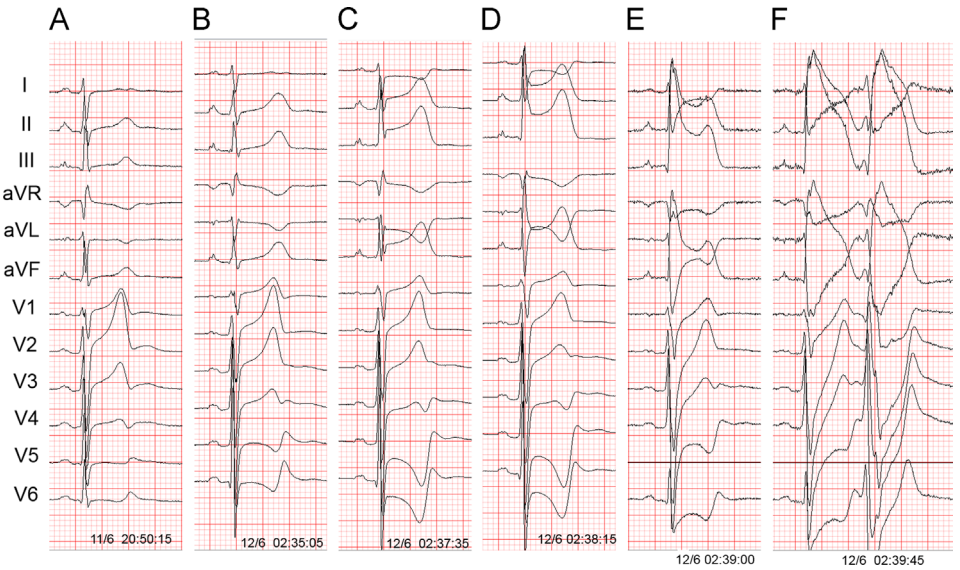


**Fig. 1.** Excerpts from a 12-lead Holter recording (25 mm/s, 1 cm/mV) demonstrating ST segment elevation in leads II, III, and aVF with reciprocal ST depression in leads V4 to V6 (B)–(F). Note the gradual broadening of the QRS with almost no distinction between the QRS end and ST beginning preceding ventricular arrhythmia onset. Lead V5 did not record during the last minute preceding VA onset, probably owing to a cable disconnection.

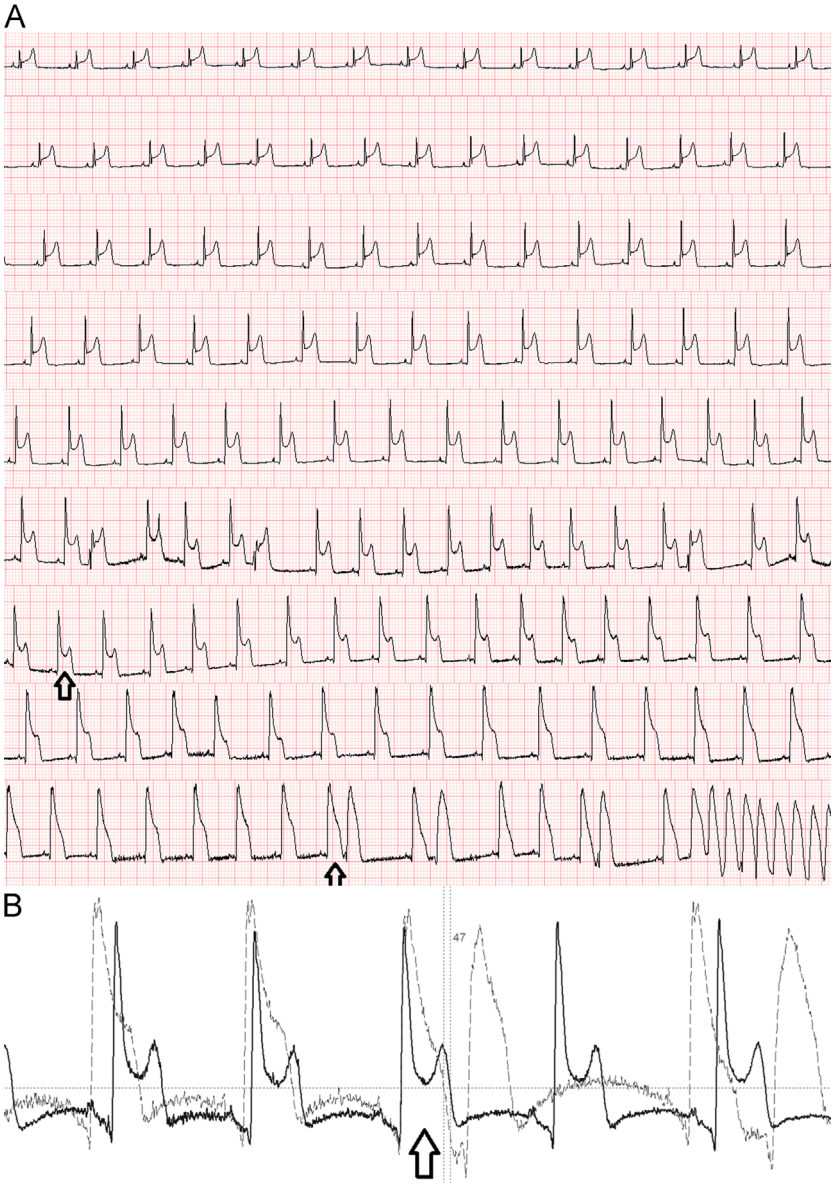


**Fig. 2.** (A) Continuous recording (lead III: 12.5 mm/s, 1 cm/mV) of the last minute preceding ventricular arrhythmia onset. Note that the gradual shortening of the repolarization segment not caused by an increase in the heart rate is best visible when the final sinus ECG complex preceding VA onset is compared to an earlier (45 s) ECG complex (marked by arrows). Both complexes are preceded by similar RR intervals. (B) The earlier (solid line) and later (dotted line) sinus ECG complexes from the top panel are superimposed and aligned by the ascending QRS limb to better display the shortening of the repolarization segment in the later ECG complex.

unstable (beat-to-beat variability) APs have been induced in experimental studies with Langendorff-perfused hearts treated with AP prolonging or HERG-channel inhibitors [5]; they were predictors of VA [5].

QT shortening of the "triangulated" QRS–ST–T complexes immediately preceding VT onset was possibly important for arrhythmia initiation, because both the AP duration prolongation and shortening in the presence of AP triangulation and instability have been shown to be markedly pro-arrhythmic [5].

Thus, the observed repolarization shortening was clearly visible only when consecutive ECG complexes were superimposed and aligned. These observations would have been less reliable if the QT intervals were measured in the same ECG complexes.

Our observations reveal an important mechanism that triggers sustained ventricular arrhythmias during acute myocardial ischemia.

## Conflict of interest

The authors have no conflict of interest.

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