Acute inferolateral myocardial ST segment elevation infarction hidden the type 1 ECG Brugada pattern in a patient with Brugada syndrome

Authors: Melina Terrana and Christophe Scavee
Service de Pathologie Cardiovasculaire, Cliniques Universitaires Saint-Luc, Bruxelles, Belgique

Comments and analysis modified by Andrés Ricardo Pérez-Riera & Raimundo Barbosa-Barros MD and Kjell Nikus, MD PhD

A 61-year old man admitted to emergence room (ER) for a prolonged chest pain.

The patient was diagnosed 3 years ago as an asymptomatic Brugada syndrome with spontaneous type 1 ECG Brugada patter and confirmed by positive genetic mutation (SCN5A Gly752Arg variant).

He did not show positive evidence of sudden death in his family history. Although, his father died at age 38 in a car accident in unclear circumstances. The ECG preformed 3 years ago we show in figure 1.

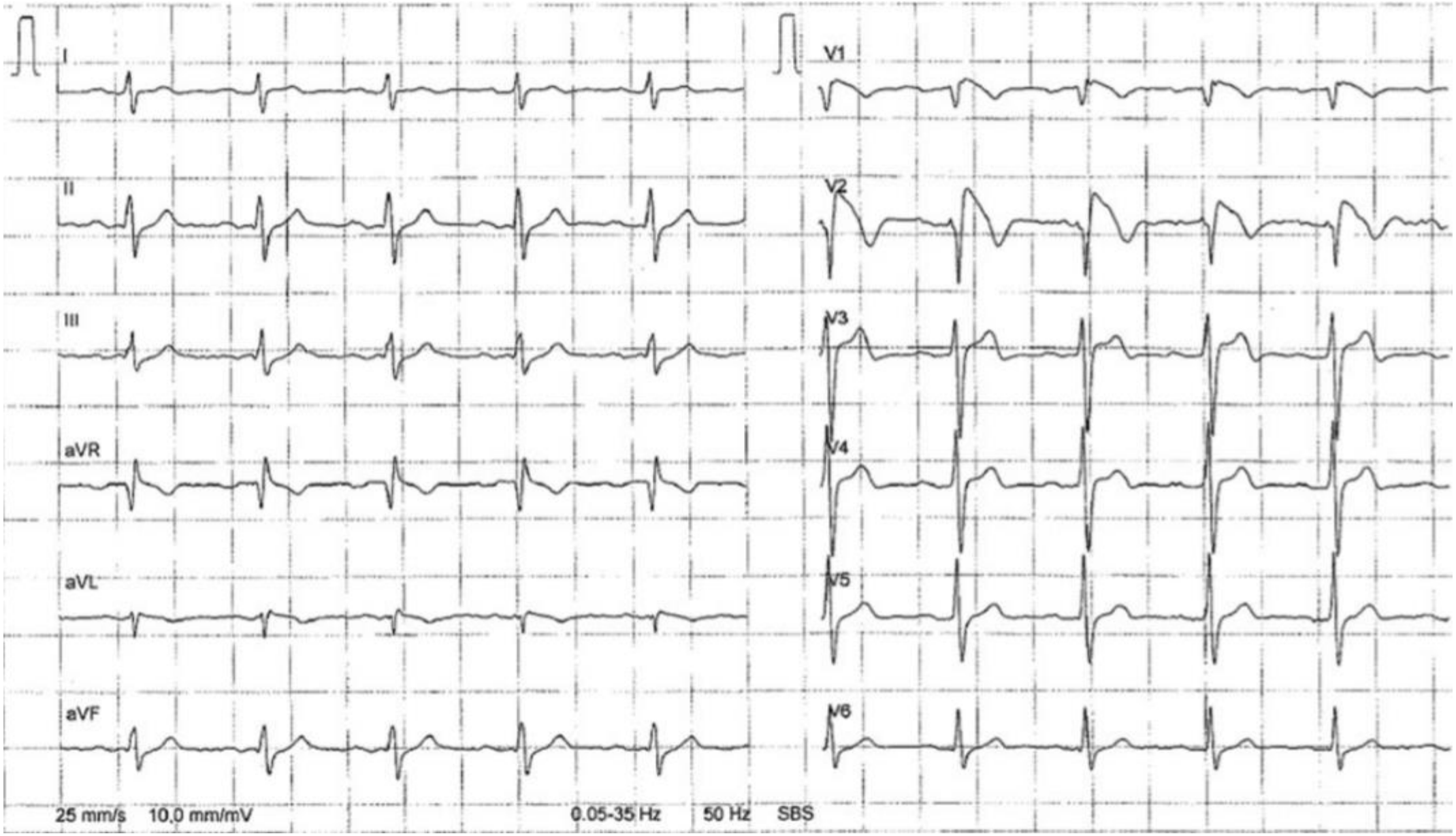
The figure 2 show the ECG preformed at admission on ER. Afterward, the patient developed ventricular tachycardia (figure 3) successfully treated by electrical cardioversion.

The ECG recorded after a thrombolytic reperfusion therapy on right coronary artery is showing in (figure 4).

In summary, the authors provide dynamic repolarization changes in the right precordial leads in a STEMI in a patient with an spontaneous Brugada type 1 ECG pattern. Remarkably, they illustrate a regression of the repolarization changes during the inferolateral myocardial infarction with lateral extension(posterior wall does not exist, followed by the recovery of the striking coved-type pattern after a successful thrombolytic reperfusion therapy.

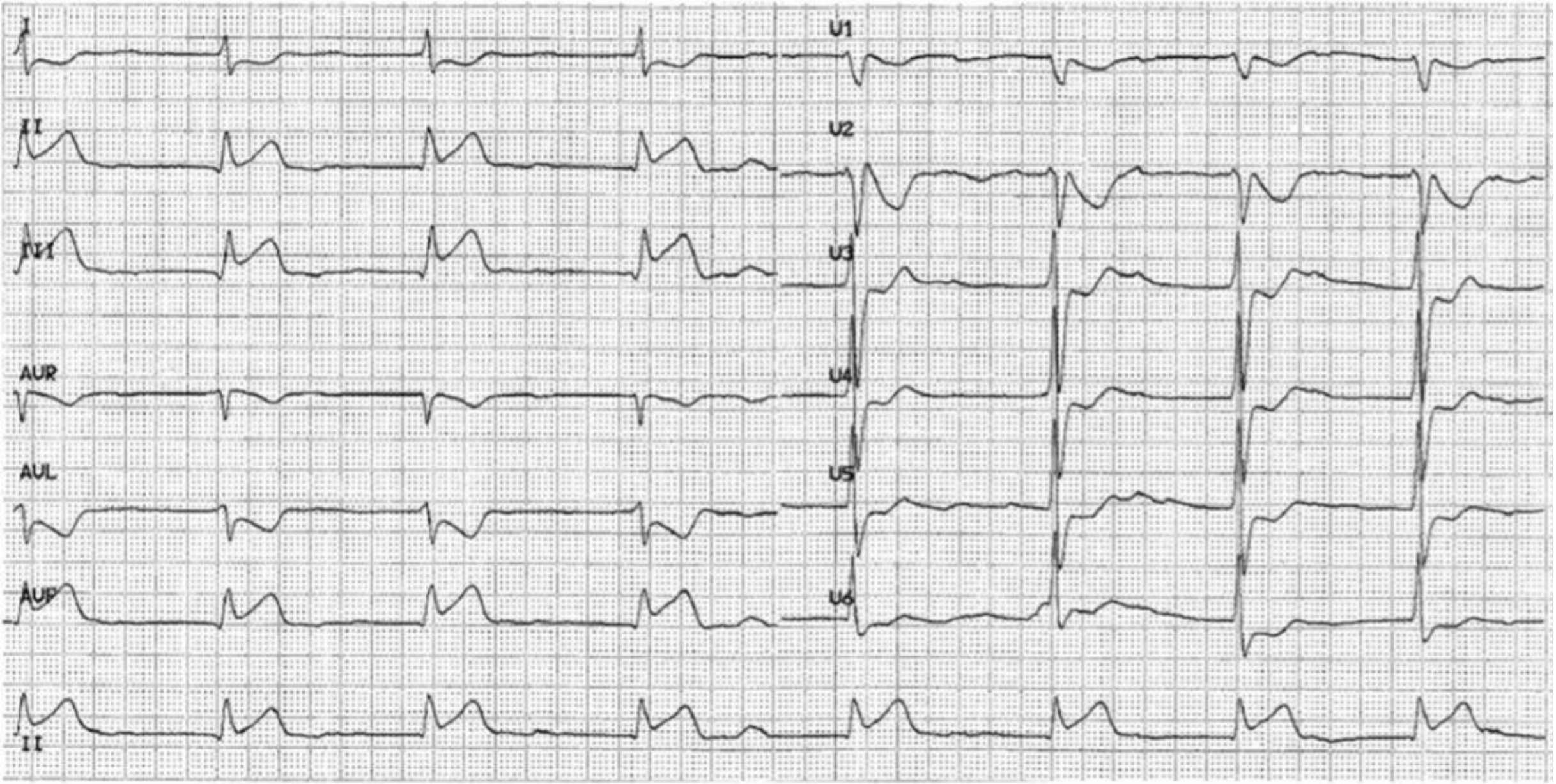
The authors hope that these illustrations will help to increase awareness for the management of Brugada patients with acute MI.

Figure 1 ECG preformed 3 years ago



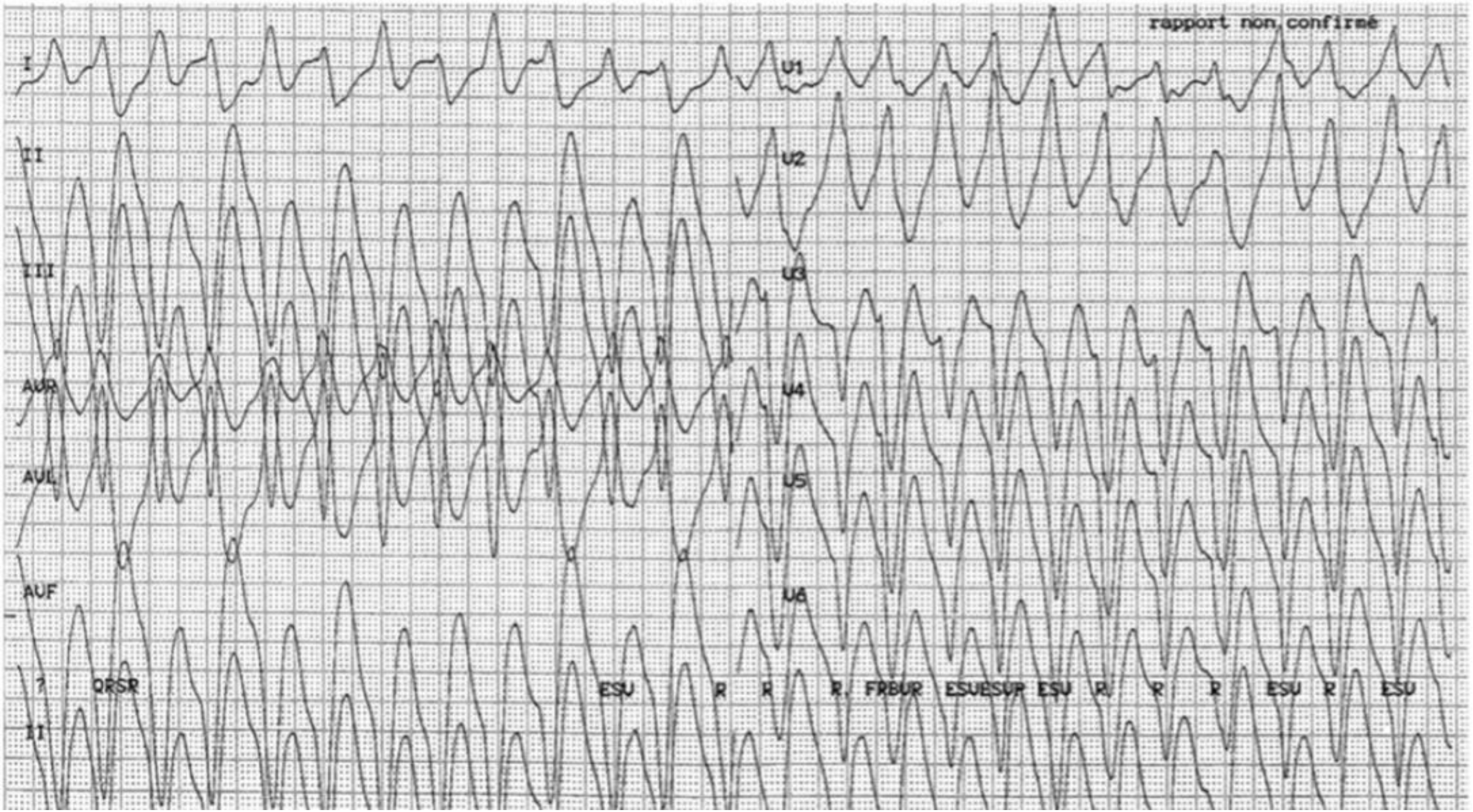
The QRS axis is hard to determinate (Indeterminate axis): near perpendicular to the frontal plane. Spontaneous Type 1 ECG Brugada pattern.

Figure 2 ECG preformed at admission during prolonged precordial pain



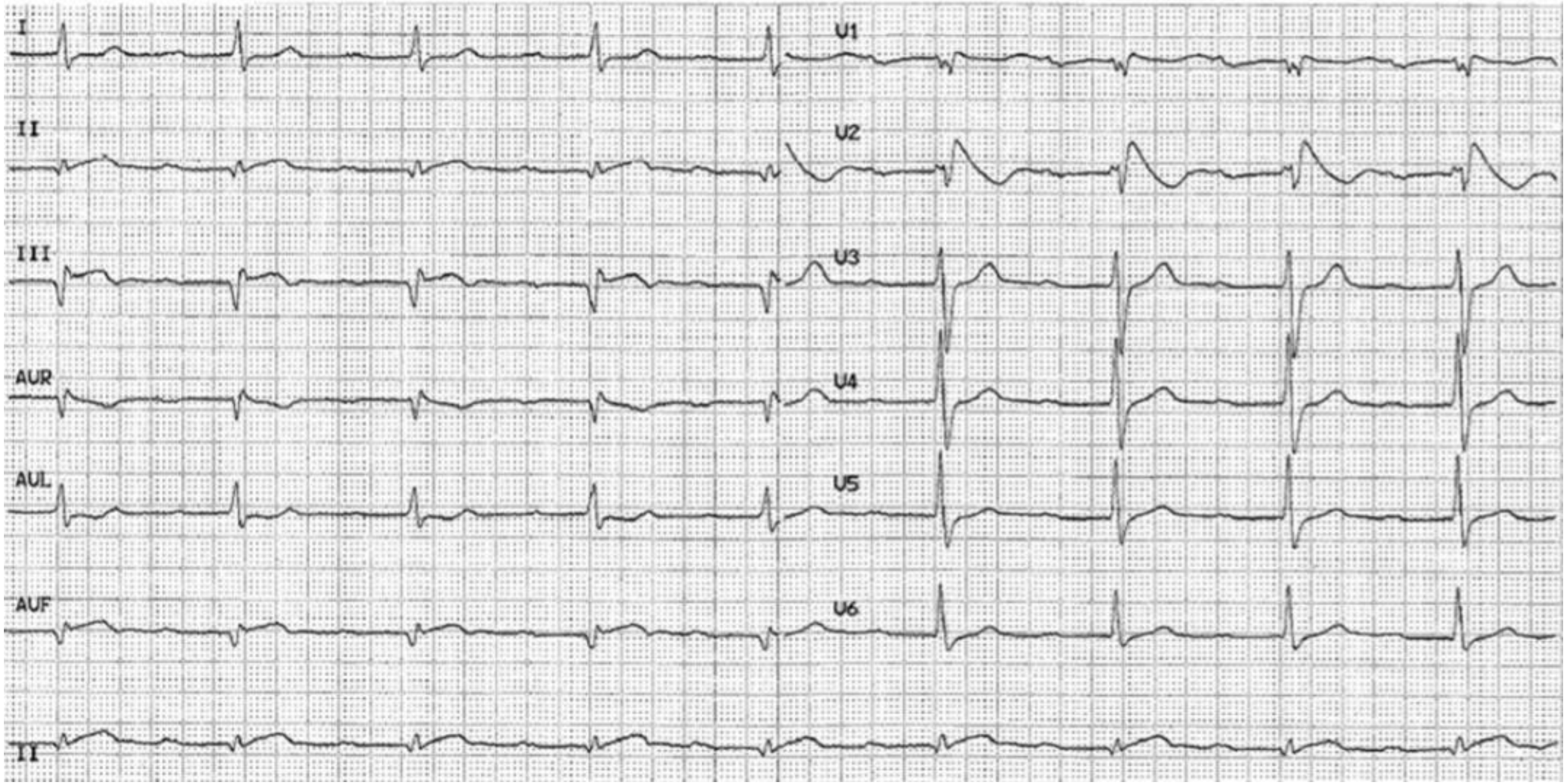
Acute STSE inferolateral MI (old dorsal) (**Bayés de Luna A 2006**) complicated with complete AV block. ST segment depression from V_1 to V_4 are reciprocal changes of inferior leads (mirror image). A ST-segment depression in right precordial leads probably hidden the type 1 ECG BrP.

Figure 3 ECG preformed during acute phase of inferolateral MI



Sustained monomorphic ventricular tachycardia, broad QRS complexes (> 120 ms) of varying duration, HR near 150bpm, monophasic R wave in V_1-V_2: focus arising from a single focus on left ventricle free wall: RBB like morphology V_1-V_2, several fusion and capture beats are observed.

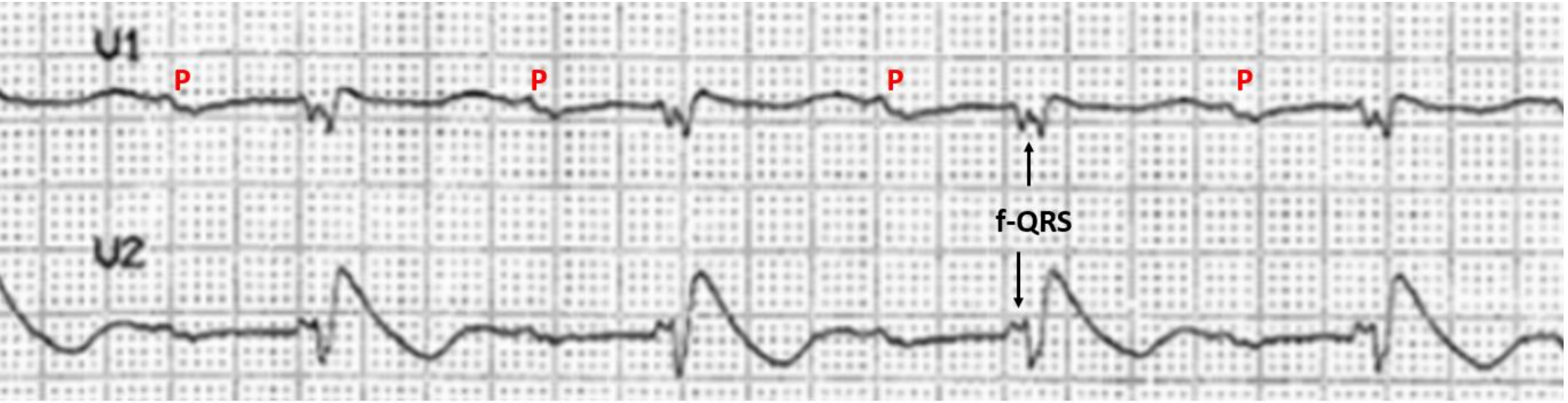
Figure 4 ECG preformed after a thrombolytic reperfusion therapy



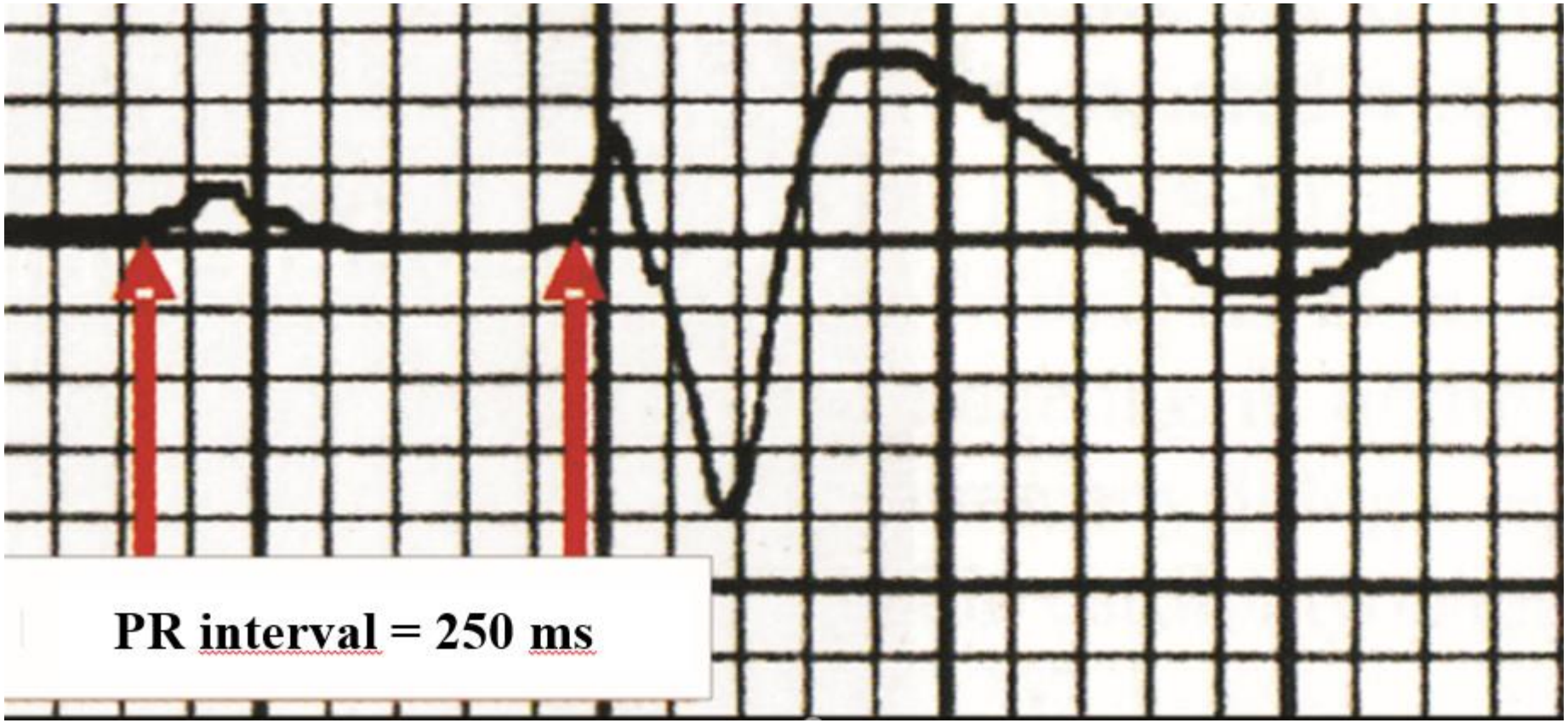
Sinus bradycardia (HR 53bpm), very prolonged PR interval: first degree AV block (PR interval 440ms), Inferior myocardial infarction sequelae, minimal ST segment elevation on inferior leads followed by biphasic "plus-minus T wave in III and aVF, resurgence of the type 1 Brugada ECG pattern as previously observed before the right coronary occlusion, and fragmented QRS on right precordial leads.

Fragmented QRS (fQRS) is a convenient marker of myocardial scar evaluated by 12-lead ECG recording. fQRS is defined as additional spikes within the QRS complex in at least two contiguous leads. In patients with CAD, fQRS was associated with myocardial scar detected by single photon emission tomography and was a predictor of cardiac events. fQRS was also a predictor of mortality and arrhythmic events in patients with reduced LVEF. fQRSs, which include various RSR' patterns, without a typical bundle-branch block are markers of altered ventricular depolarization owing to a prior myocardial scar. fQRS improve the ability to detect a prior MI compared with Q waves alone by ECG. **(Das MK 2000)** The fQRS is a marker of a prior MI, defined by regional perfusion abnormalities, which has a substantially higher sensitivity and negative predictive value compared with the Q wave. The fQRS is an independent predictor of cardiac events in patients with CAD. It is associated with significantly lower event-free survival for a cardiac event on long-term follow-up **(Das MK 2007)**.

V_1-V_2 of an ECG preformed after PCI



Very prolonged PR interval(440ms), type 1 Brugada ECG pattern, fragmented QRS. First-degree atrioventricular block is frequently encountered in clinical practice and is generally considered a benign process. However, there is emerging evidence that prolonged PR interval is associated with significant increases risk of atrial fibrillation, heart failure and adverse cardiovascular outcomes and mortality(**Kwok CS, 2016**). In Brugada syndrome PR interval prolongation consequence of prolonged HV split or HV is a bad prognosis marker. Multivariate analysis revealed that a PR interval ≥170 ms and T-wave amplitude <105 µV in lead V1 are independent risk stratifiers of life-threatening events. (**Miyamoto A 2011**).



The figure shows a tracing of a symptomatic patient with Brugada syndrome after intravenous ajmaline injection. First-degree atrioventricular block (PR interval = 216 ms) and Brugada type-1 ECG pattern in V_1 lead (positive test).

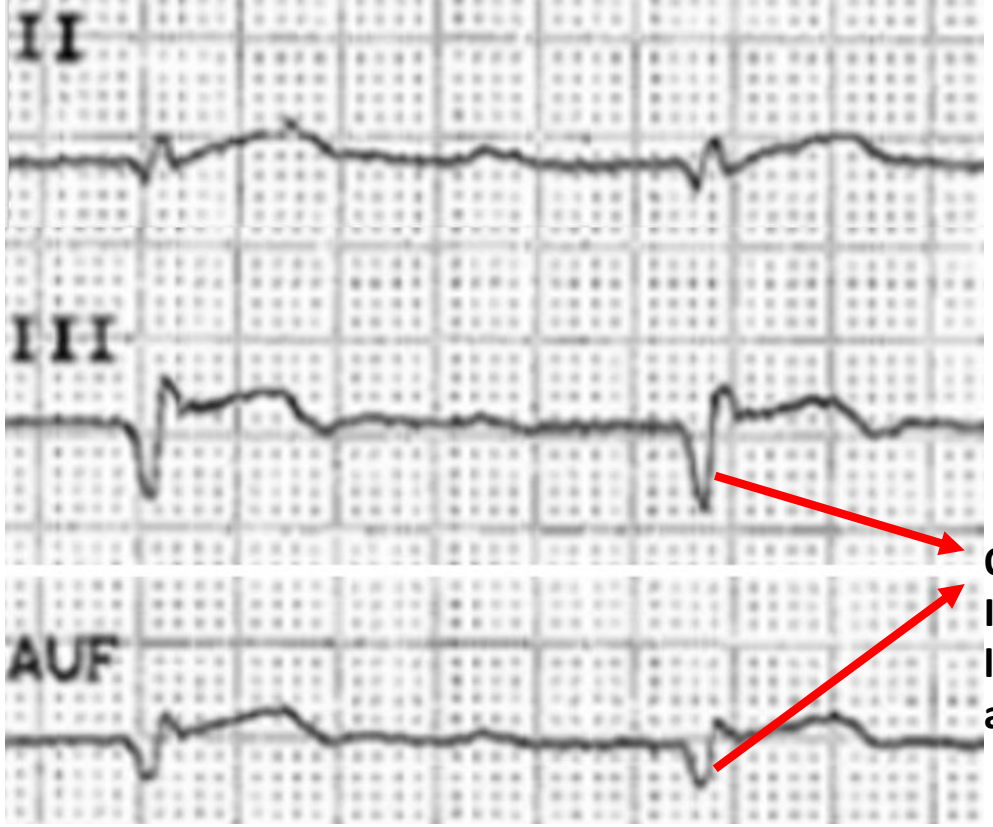
In BrS the PR interval of ECG and the His bundle electrogram in approximately 50% of the cases are prolonged, even reaching sometimes figures of 100 ms (**Yokokawa 2007**). This prolongation of the PR interval is observed predominantly in cases where the SCN5A gene mutation can be proven (carriers). The presence of a prolongued HV interval is possible in HBE by the existence of intra-His or infra-His block.

PR prolongation consequence of HV split or HV prolongation is considered another ECG risk marker (**Miyamoto 2011**).

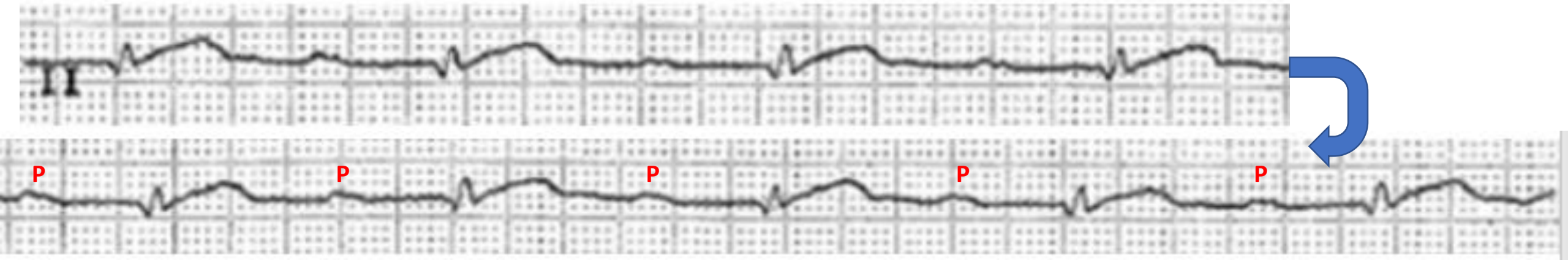
fQRS, T-wave inversion, and ST depression are independent predictors of mortality during a mean follow-up period of 34 +/- 16 months. In conclusion, fQRS on ECG is a moderately sensitive but highly specific sign for ST elevation MI and NSTEMI. fQRS is an independent predictor of mortality in patients with ACS.

Entities where fQRS is used as a non-invasive marker of events (Das 2009)

- **Coronary artery disease (Das 2010)** where it represents a conduction delay of the stimulus and is associated to an increase in mortality and arrhythmic events in these patients.
- **Non-ischemic cardiomyopathies (Das 2010). In non-ischemic dilated cardiomyopathy with narrow QRS to predict dyssynchrony (Tigen K 2009)**
- **Arrhythmogenic right ventricular cardiomyopathy/dysplasia (ARVC/D) (Peters S 2008)**
- **Cardiac sarcoidosis (Homsi M 22009)**
- **Congenital heart diseases (Moss A 2010)**
- **Brugada syndrome (Haraoka K 2010)**
- **Acquired long QT syndrome (Yuce M 2010)** The existence of fQRS plays an important role in the appearance of Torsades de Pointes (TdP) in patients with acquired long QT interval.

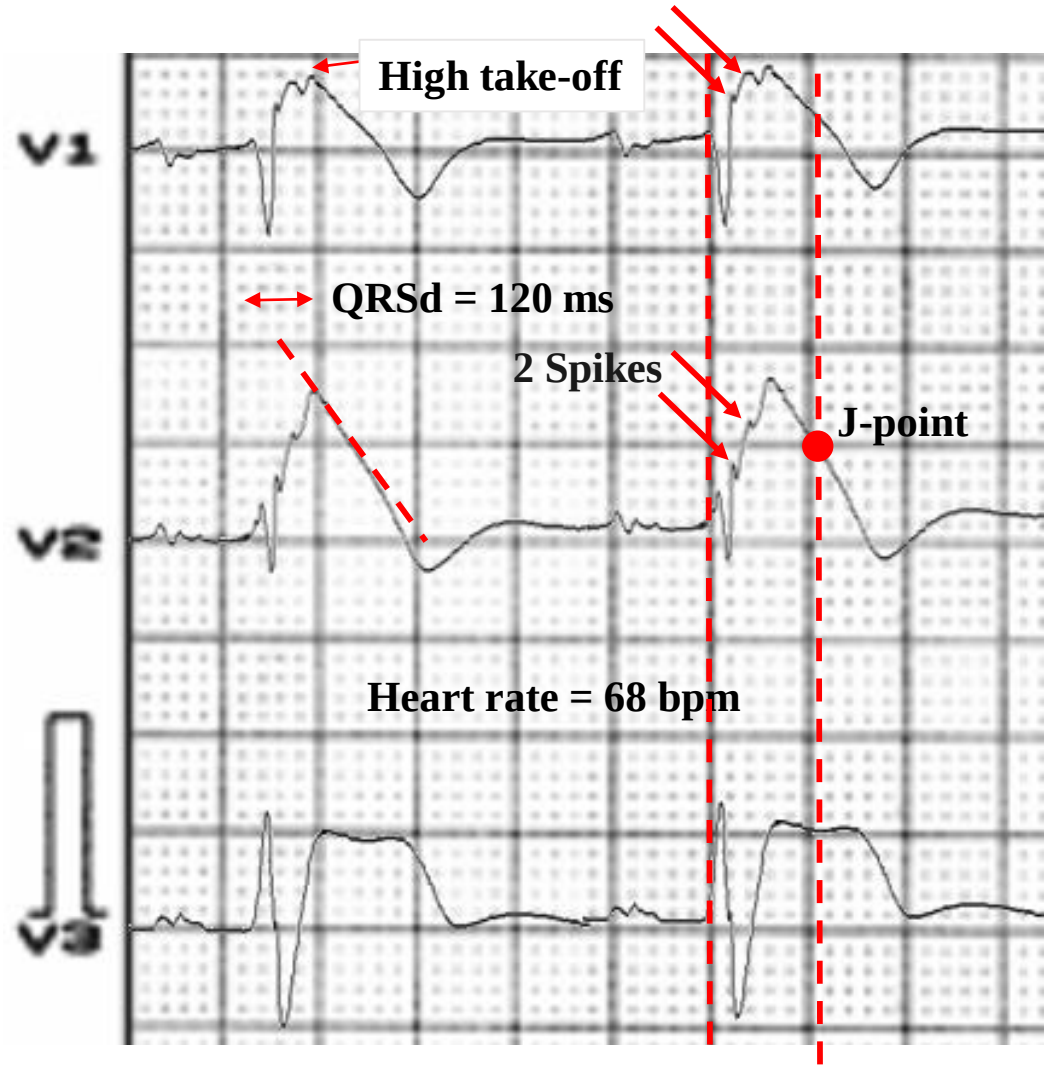


Q wave ≥ 3ms and ≥0.1 mV deep or QS complex in lead I, II, aVL, aVF, or V4 to V6 in any 2 leads of a contiguous lead grouping (I, aVL, and V6; V4 to V6; and II, III, and aVF) (Das MK 2009)



Very prolonged PR interval(440ms), sequelae Q necrose(Q > 40ms III and aVF), minimal ST segment elevation followed by biphasic “plus-minus” T-wave in III and aVF.

Fragmented QRS in Brugada Syndrome



Two spikes are observed at the upstroke of the S wave in leads V_1 and V_2.

Dotted lines show onset and termination of the QRS complex

Fragmented wide QRS complex in a 35-year-old Asian male patient with BrS. f-QRS appears to be a marker for the substrate for spontaneous VF in BrS and predicts patients at high risk of syncope. It is a conduction abnormality within the QRS complex (**Morita 2008**).

References

1. Bayés de Luna A, Wagner G, Birnbaum Y, Nikus K, Fiol M, Gorgels A, Cinca J, Clemmensen PM, Pahlm O, Sclarovsky S, Stern S, Wellens H, Zareba W; International Society for Holter and Noninvasive Electrocardiography.A new terminology for left ventricular walls and location of myocardial infarcts that present Q wave based on the standard of cardiac magnetic resonance imaging: a statement for healthcare professionals from a committee appointed by the International Society for Holter and Noninvasive Electrocardiography.Circulation. 2006 Oct 17;114(16):1755-60.
2. Das MK, Khan B, Jacob S, Kumar A, Mahenthiran J.Significance of a fragmented QRS complex versus a Q wave in patients with coronary artery disease. Circulation. 2006 May 30;113(21):2495-501.
3. Das MK, Saha C, El Masry H, Peng J, Dandamudi G, Mahenthiran J, McHenry P, Zipes DP. Fragmented QRS on a 12-lead ECG: a predictor of mortality and cardiac events in patients with coronary artery disease. Heart Rhythm 2007;4:1385–1392.
4. Das MK, Michael MA, Suradi H, Peng J, Sinha A, Shen C, Mahenthiran J, Kovacs RJ.Usefulness of fragmented QRS on a 12-lead electrocardiogram in acute coronary syndrome for predicting mortality. Am J Cardiol. 2009 Dec 15;104(12):1631-7.
5. Haraoka K, Morita H, Saito Y, et al. Fragmented QRS is associated with torsades de pointes in patients with acquired long QT syndrome. Heart Rhythm. 2010 Dec;7:1808-1814.
6. Homsi M, Alsayed L, Safadi B, et al. Fragmented QRS complexes on 12-lead ECG: a marker of cardiac sarcoidosis as detected by gadolinium cardiac magnetic resonance imaging. Ann Noninvasive Electrocardiol. 2009 Oct;14:319-326
7. Kwok CS, Rashid M, Beynon R, Barker D, Patwala A, Morley-Davies A, Satchithananda D, Nolan J, Myint PK, Buchan I, Loke YK, Mamas MA.Prolonged PR interval, first-degree heart block and adverse cardiovascular outcomes: a systematic review and meta-analysis.Heart. 2016 May;102(9):672-80.
8. Miyamoto A1, Hayashi H, Makiyama T, Yoshino T, Mizusawa Y, Sugimoto Y, Ito M, Xue JQ, Murakami Y, Horie M.Risk determinants in individuals with a spontaneous type 1 Brugada ECG.Circ J. 2011;75(4):844-51.
9. Morita H, Kusano KF, Miura D, Nagase S, Nakamura K, Morita ST, Ohe T, Zipes DP, Wu J. Fragmented QRS as a marker of conduction abnormality and a predictor of prognosis of Brugada syndrome. Circulation. 2008 Oct 21;118(17):1697-704.
10. Moss AJ. Fragmented QRS: the new high-risk kid on the block in acquired long QT syndrome. Heart Rhythm.2010 Dec;7:1815-18166.
11. Peters S, Trümmel M, Koehler B. QRS fragmentation in standard ECG as a diagnostic marker of arrhythmogenic right ventricular dysplasia-cardiomyopathy. Heart Rhythm. 2008 Oct;5:1417-1421.

12. Tigen K, Karaahmet T, Gurel E, et al. The utility of fragmented QRS complexes to predict significant intraventricular dyssynchrony in nonischemic dilated cardiomyopathy patients with a narrow QRS interval. Can J Cardiol. 2009 Sep;25:517-522.
13. Yokokawa M, Noda T, Okamura H, Satomi K, Suyama K, Kurita T, Aihara N, Kamakura S, Shimizu W. Comparison of long-term follow-up of electrocardiographic features in Brugada syndrome between the SCN5A-positive probands and the SCN5A-negative probands. Am J Cardiol. 2007 Aug 15;100(4):649-55.
14. Yuce M, Davutoglu V, Ozbala B, et al. Fragmented QRS is predictive of myocardial dysfunction, pulmonary hypertension and severity in mitral stenosis. Tohoku J Exp Med. 2010; 220: 279-283