INDIAN HEART JOURNAL 67 (2015) 392–394

Available online at www.sciencedirect.com

ScienceDirect

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

Images in Cardiology

The classical "R-on-T" phenomenon

Fatih Oksuz, Baris Sensoy, Ekrem Sahan, Fatih Sen, Kazım Baser, Hande Cetin, Sefa Unal, Ozcan Ozeke, Serkan Topaloglu, Dursun Aras*

Turkiye Yuksek Ihtisas Training and Research Hospital, Department of Cardiology, Ankara, Turkey

ARTICLE INFO

Article history:
Received 14 July 2014
Accepted 19 February 2015
Available online 27 April 2015

Keywords:
R-on-T phenomenon
Polymorphic ventricular tachycardia
Torsade de pointes

ABSTRACT

The polymorphic ventricular tachycardia (PVT) is uncommon arrhythmia with multiple causes and has been classified according to whether they are associated with long QT interval or normal QT. Whereas "Torsade de pointes (TdP)" is an uncommon and distinctive form of PVT occurring in a setting of prolonged QT interval, which may be congenital or acquired (congenital or acquired), "PVT with normal QT" is associated with myocardial ischemia, electrolyte abnormalities (hypokalemia), mutations of the cardiac sodium channel (Brugada syndrome), and the ryanodine receptor (catecholaminergic PVT). This distinction is crucial because of the differing etiologies and management of these arrhythmias. Moreover, the PVT in the setting of acute MI generally occurs during the hyperacute phase, is related to ischemia ("ischemic PVT") and is not associated with QT prolongation. It is triggered by ventricular extrasystoles with very short coupling interval (the "R-on-T" phenomenon) and is not pause-dependent. However, recently there has been described a new PVT during the "healing phase" of MI in patients with no evidence of ongoing ischemia and following excessive QT prolongation, the electrophysiologic abnormality being a "pause-dependent infarct-related TdP" due to a LQTS in healing MI patients. Therefore, "ischemic PVT" differs from "infarct-related TdP" in terms of pathophysiology and ECG manifestations.

The "R-on-T phenomenon" was first described by Smirk in 1949 as "R waves interrupting T waves".[1,2] In 1966, Francois Dessertenne described a specific electrocardiographic form of polymorphic ventricular tachycardia (PVT) characterized by changing amplitude of the complexes with a characteristic twist around the isoelectric baseline with prolonged QT interval, which he termed "torsades de pointes" (TdP).[3] In its most typical form, sudden slowing of heart rate (i.e., pauses) invariably precede each burst of TdP, and the recurrent arrhythmia is referred to as "pause-dependent TdP".[4] Indeed, the PVT is uncommon arrhythmia with multiple causes, and has been classified according to whether they are associated with long QT interval or normal QT.[5,6] Contemporary classifications of the long QT syndrome (LQTS) refer to the congenital LQTS as "adrenergic dependent" and to the acquired LQTS as "pause dependent".[4] Whereas "TdP" is a form of PVT occurring in a setting of prolonged QT interval, which may be congenital or acquired, "PVT with normal QT" is associated with myocardial ischemia, electrolyte abnormalities (hypokalemia), mutations of the cardiac sodium channel

* *Corresponding author*. Türkiye Yüksek İhtisas Hastanesi, Kardiyoloji Klinigi, Ankara 06100, Turkey. Tel.: +90 505 383 67 73.
E-mail address: ozcanozeke@gmail.com (O. Ozeke).
http://dx.doi.org/10.1016/j.ihj.2015.02.030

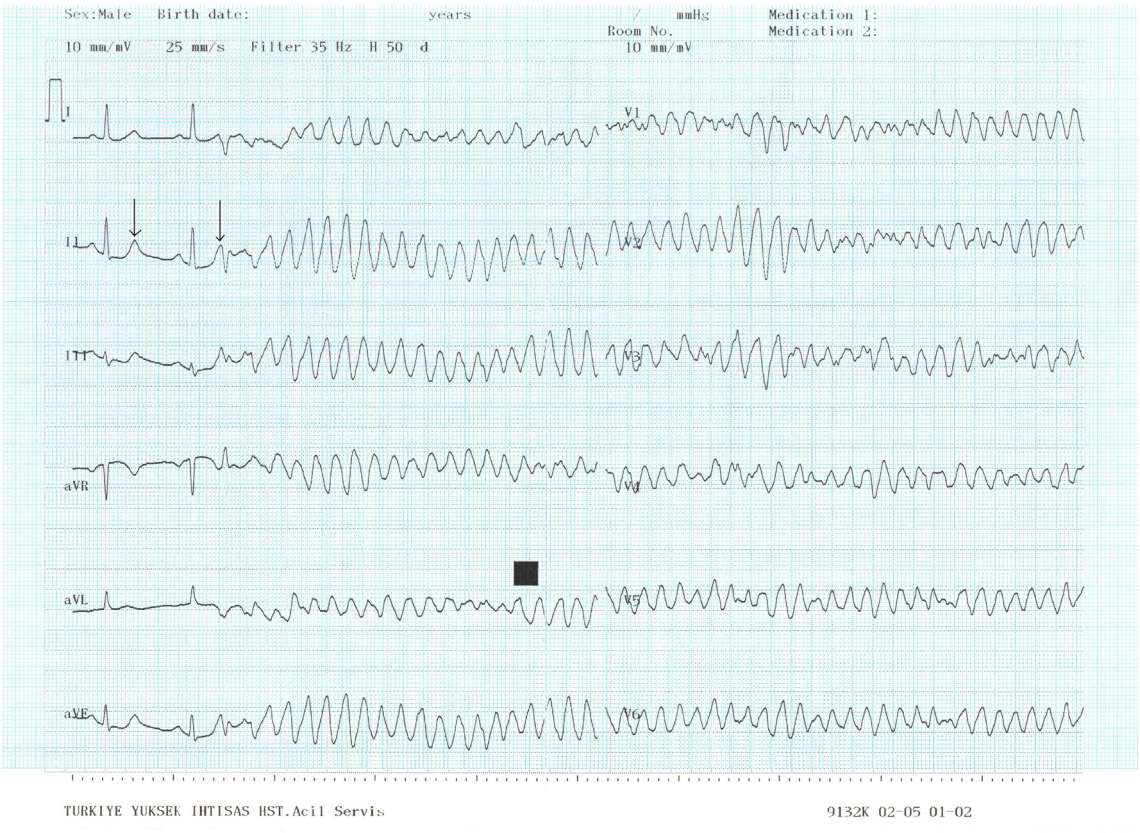

Fig. 1 – 12 lead ECG taken at emergency department showing the initiation of polymorphic ventricular tachycardia (PVT) by R-on-T phenomenon (arrows).

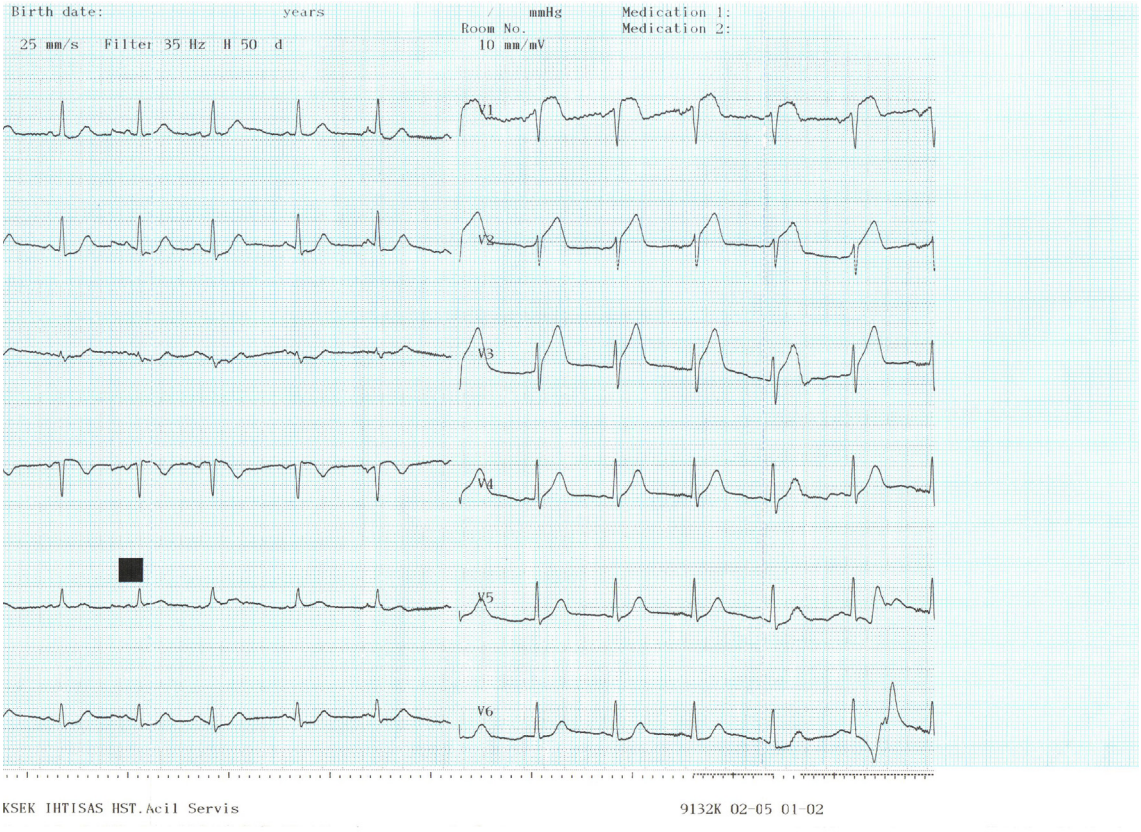

Fig. 2 – 12 lead ECG taken immediately after successful electrical defibrillation suggesting hyperacute anterior myocardial infarction.

(Brugada syndrome), and the ryanodine receptor (catecholaminergic PVT).[6] This distinction is crucial because of the differing etiologies and management of these arrhythmias. A 59-year-old male patient presented to the emergency department approximately 1 h after experiencing a severe crushing retrosternal chest pain with radiation to his left arm. No metabolic abnormalities such as hyponatremia, hypocalcemia, hypomagnesemia, or hypermagnesemia were noted. The most striking feature of the patient's ECG was its newly detected PVT, which was consistent with, but not diagnostic of, TdP initiated by "R-on-T" phenomenon (Fig. 1, arrows). ECG taken immediately after successful electrical defibrillation revealed ST elevation in V1–3 suggesting hyperacute anterior myocardial infarction (MI) (Fig. 2). Emergency coronary angiography showed a total occlusion of the middle part of the left anterior descending artery, which was successfully revascularized with a bare metal stent.

PVT in the setting of acute MI generally occurs during the hyperacute phase, is related to ischemia, and is not associated with QT prolongation.[7] They are triggered by ventricular extrasystoles with very short coupling interval (the "R-on-T" phenomenon) and is not pause-dependent.[8] Therefore, "ischemic PVT" differs from "infarct-related TdP" in terms of pathophysiology and ECG manifestations.[8] Although some investigators have used the term "TdP" to describe PVT during MI,[9,10] the available illustrations suggest that the majority of the patients had "ischemic PVT" with short QT interval. Recently, Halkin et al described a new PVT during the "healing phase" of MI in patients with no evidence of ongoing ischemia and following excessive QT prolongation, therefore, named it as "pause-dependent/infarct related TdP" due to a LQTS in healing MI patients. Although our patient had PVT that fulfilled the morphologic criterion for TdP, it was associated with normal QTc (393 ms), thereby precluding the diagnosis of TdP, and we preferred to use the term "ischemic PVT" instead of TdP.

Conflicts of interest

The authors have none to declare.

REFERENCES

1. Smirk FH. R waves interrupting t waves. *Br Heart J*. 1949;11:23–36.
2. Smirk FH, Palmer DG. A myocardial syndrome. With particular reference to the occurrence of sudden death and of premature systoles interrupting antecedent t waves. *Am J Cardiol*. 1960;6:620–629.
3. Dessertenne F. Ventricular tachycardia with 2 variable opposing foci. *Archives des maladies du coeur et des vaisseaux*. 1966;59:263–272.
4. Viskin S, Alla SR, Barron HV, et al. Mode of onset of torsade de pointes in congenital long qt syndrome. *J Am Coll Cardiol*. 1996;28:1262–1268.
5. Passman R, Kadish A. Polymorphic ventricular tachycardia, long q-t syndrome, and torsades de pointes. *Med Clin North Am*. 2001;85:321–341.
6. Lim W, Chiamvimonvat N, Amsterdam EA. Electrocardiogram with a twist. *Crit Pathw Cardiol*. 2012;11:218–219.
7. Halkin A, Roth A, Lurie I, Fish R, Belhassen B, Viskin S. Pause-dependent torsade de pointes following acute myocardial infarction: a variant of the acquired long qt syndrome. *J Am Coll Cardiol*. 2001;38:1168–1174.
8. Wolfe CL, Nibley C, Bhandari A, Chatterjee K, Scheinman M. Polymorphous ventricular tachycardia associated with acute myocardial infarction. *Circulation*. 1991;84:1543–1551.
9. Zilcher H, Glogar D, Kaindl F. Torsades de pointes: occurrence in myocardial ischaemia as a separate entity. Multiform ventricular tachycardia or not? *Eur Heart J*. 1980;1:63–71.
10. Vidal J, Hernandez Guevara JL, Cardenas M. Helicoid ventricular tachycardia, torsade de pointes, in acute myocardial infarction. *Archivos del Instituto de Cardiologia de Mexico*. 1983;53:237–245.