Mode of onset of polymorphic ventricular tachycardia in acute myocardial infarction

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BACKGROUND: Polymorphic ventricular tachycardia (PVT) can occur during acute myocardial infarction (MI). In the past, studies investigated the initiation pattern of ventricular tachycardias in different patient populations; however, the mode of onset of PVT in acute MI patients has not been investigated previously.

OBJECTIVE: To retrospectively investigate the electrophysiological features of PVT with different initiation patterns in acute MI patients to assess whether there is a relationship of the initiation patterns of PVT with clinical and electrophysiological characteristics.

METHODS: Sixty-two rhythm strips defined as PVT from 53 patients (mean $\pm$ SD) age 63 ± 8 years) with acute ST elevation MI were analyzed. All patients were monitored while they were hospitalized in the coronary care unit, and the electrocardiogram strips were obtained from continuous monitoring. PVT was defined as sudden-onset tachycardia if it was not preceded by ventricular ectopic beats. PVT that was preceded by single or multiple ectopic beats was defined as nonsudden-onset tachycardia.

RESULTS: Nonsudden-onset episodes were more common than sudden-onset episodes (40 episodes [64.5%] versus 22 episodes [35.5%]). In the nonsudden-onset group, 25 episodes (62.5%) were initiated after a single ectopic beat, while 15 episodes (37.5%) were initiated after multiple complexes. The mean ($\pm$ SD) left ventricular ejection fraction of patients with nonsudden-onset PVT was decreased ($53 \pm 6\%$ versus $65 \pm 7\%$, $P < 0.01$). Nonsudden-onset tachycardias had lower coupling intervals than sudden-onset tachycardias. Similarly, the PVT cycle length was shorter in the presence of nonsudden-onset initiation. When nonsudden-onset PVT episodes were further subclassified based on the morphology of the first beat of tachycardia, 26 PVTs (65%) had a first beat of tachycardia similar to the subsequent PVT beats and 14 (35%) did not.

CONCLUSIONS: These results demonstrate that PVT is often preceded by ventricular ectopy in acute MI patients. Nonsudden-onset PVT is usually characterized by a lower coupling interval, shorter PVT cycle length and an associated lower ejection fraction.

Key Words: Acute myocardial infarction; Ventricular tachycardia

Le mode d'apparition de la tachycardie ventriculaire polymorphe en cas d'infarctus aigu du myocarde

HISTORIQUE : Une tachycardie ventriculaire polymorphe (TVP) peut se produire pendant un infarctus aigu du myocarde (IAM). Par le passé, les recherches ont porté sur le mode d'apparition des tachycardies ventriculaires au sein de différentes populations de patients, mais le mode d'apparition de la TVP chez des patients atteints d'un IAM n'a jamais fait l'objet d'études.

OBJECTIF : Faire l'examen rétrospectif des caractéristiques électrophysiologiques de la TVP selon divers modes d'apparition en cas d'IAM afin d'évaluer s'il existe un lien entre les modes d'apparition de la TVP et les caractéristiques cliniques et électrophysiologiques.

MÉTHODOLOGIE : Les chercheurs ont analysé 62 bandes de rythme définies comme un TVP chez 53 patients (âge moyen $\pm$ ÉT) de 63 ± 8 ans) ayant un IAM avec élévation du segment ST. Tous les patients ont fait l'objet d'une surveillance pendant leur hospitalisation à l'unité de soins coronariens, et ils ont obtenu les tracés électrocardiographiques de la surveillance continue. La TVP était définie comme une tachycardie à apparition subite si elle n'était pas précédée de battements ectopiques ventriculaires. Une TVP précédée d'un ou plusieurs battements ectopiques ventriculaires était définie comme une tachycardie à apparition non subite.

RÉSULTATS : Les épisodes à apparition non subite étaient plus courants que ceux à apparition subite (40 épisodes [64,5 %] par rapport à 22 [35,5 %]). Dans le groupe à apparition non subite, 25 épisodes (62,5 %) ont commencé après un seul battement ectopique, tandis que 15 (37,5 %) ont commencé après des complexes multiples. La fraction d'éjection ventriculaire gauche moyenne ($\pm$ ÉT) des patients ayant une TVP à apparition non subite était réduite (53 ± 6 % par rapport à 65 ± 7 %, $P < 0,01$). Les tachycardies à apparition non subite s'associaient à des intervalles de couplage plus courts que les tachycardies à apparition subite. De même, le cycle de TVP était plus court en cas d'apparition subite. Lorsque les épisodes de TVP à apparition non subite étaient sous-classés d'après la morphologie du premier battement de tachycardie, 26 TVP (65 %) étaient liés à un premier battement similaire aux battements subséquents de la TVP et 14 (35 %) ne l'étaient pas.

CONCLUSIONS : Ces résultats démontrent que la TVP est souvent précédée d'une ectopie ventriculaire chez les patients ayant un IAM. La TVP à apparition non subite se caractérise généralement par un intervalle de couplage plus court, un cycle de TVP plus court et une fraction d'éjection connexe plus basse.

Both sustained monomorphic ventricular tachycardia (MVT) and polymorphic ventricular tachycardia (PVT) are important complications of acute myocardial infarction (MI). Death from a sustained ventricular tachycardia (VT) in the setting of an acute MI has historically been one of the most frequent causes of sudden cardiac death. When PVT occurs after an acute MI, it is typically associated with signs or symptoms of recurrent myocardial ischemia (1). In recent years, some studies (2-4) have investigated the mode of onset of VT in

different populations using various recording techniques. The use of Holter electrocardiogram (ECG) recordings and analysis of stored intracardiac electrograms from implantable cardioverter defibrillators (ICDs) enabled us to study the initiation patterns of ventricular arrhythmias, providing the opportunity to assess mechanisms of VT onset and potential future therapeutic implications (5-11).

Few studies have investigated the initiation pattern of sustained ventricular arrhythmias in acute MI patients. In one of our studies (9),

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TABLE 1
Clinical characteristics of patients

	Tachycardia onset		P
	Sudden (n=21)	Nonsudden (n=32)	
Age, years, mean $\pm$ SD	64 $\pm$ 6	62 $\pm$ 8	NS
Men	13 (62)	21 (65)	NS
Hypertension	9 (43)	11 (34)	NS
Hypercholesterolemia	14 (67)	21 (65)	NS
Smoking	17 (81)	27 (84)	NS
Diabetes mellitus	5 (24)	8 (25)	NS
Inferior MI	12 (57)	19 (59)	NS
Anterior MI	7 (33)	10 (31)	NS
LVEF, %, mean $\pm$ SD	64 $\pm$ 7	53 $\pm$ 6	<0.01
History of syncope or arrest	4 (19)	5 (16)	NS
Use of ACE inhibitor or ARB	15 (71)	22 (69)	NS
Use of beta-blocker	14 (67)	20 (63)	NS

Data presented as n (%) unless otherwise indicated. ACE Angiotensin-converting enzyme; ARB Angiotensin receptor blocker; LVEF Left ventricular ejection fraction; MI Myocardial infarction; NS Nonsignificant

the analysis of rhythm strips demonstrated that MVT is most often preceded by ventricular ectopic beats in the acute phase of MI. However, little is known about how PVT is initiated in the acute MI patient.

The aims of the present study were to investigate the electrophysiological features of PVT with different initiation patterns in acute MI patients and to assess whether there is a relationship of initiation patterns in sustained PVT with clinical and electrophysiological characteristics.

METHODS

The present retrospective study used data from 62 rhythm strips indicating PVT, from 53 patients (mean [$\pm$ SD] age 63 $\pm$ 8 years) with acute ST elevation MI between January 1, 2003, and December 31, 2008. All patients were monitored while hospitalized in the coronary care unit (CCU), and the ECG strips were obtained by printout from continuous monitoring. Patients receiving antiarrhythmic drugs (except beta-blockers) and those with a history of VT were not included in the study. Patient characteristics are presented in Table 1.

Definitions

ST elevation MI was defined as described in the consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction (12). PVT was defined as an unstable rhythm with continuously

TABLE 2
Electrophysiological parameters

	Tachycardia onset		P
	Sudden (n=22)	Nonsudden (n=40)	
CL-SR, ms	775 $\pm$ 196	930 $\pm$ 211	<0.001
CL-PVT, ms	252 $\pm$ 35	212 $\pm$ 27	<0.05
Coupling interval, ms	689 $\pm$ 198	512 $\pm$ 162	<0.001
Prematurity index	0.98 $\pm$ 0.5	0.55 $\pm$ 0.4	<0.01
Corrected QT duration, ms	432 $\pm$ 76	427 $\pm$ 68	NS

CL-PVT Mean cycle length of polymorphic ventricular tachycardia; CL-SR Mean cycle length of sinus beats obtained before tachycardia (in steady-state period); NS Nonsignificant

varying QRS complex morphology at a rate of at least 200 beats/min. PVTs were classified as sudden onset or nonsudden onset. Tachycardia that was not preceded by a ventricular ectopic beat was defined as sudden-onset PVT (Figure 1). PVT that was preceded by a single or multiple ectopic beats, including a short-long-short sequence, was defined as nonsudden-onset PVT (Figure 2). The preceding RR cycle length (measured as the mean of eight RR intervals before PVT), tachycardia cycle length, coupling interval (CI) and prematurity index (PI) of the first tachycardia beat were assessed. The CI of the beat initiating tachycardia was defined as an interval between the first beat of tachycardia and the final beat preceding tachycardia. The PI was calculated as the ratio of the CI of the beat initiating PVT to the immediately preceding cycle length.

Statistical analysis was performed using an unpaired Student's *t* test and ANOVA for comparison of continuous variables among groups, while the χ^2 test was applied for the analysis of discrete variables.

RESULTS

In total, 43 patients experienced one episode of PVT, while 10 patients experienced at least two episodes of PVT. Nonsudden-onset episodes were more common than sudden-onset episodes (40 episodes [64.5%] versus 22 episodes [35.5%]). In the nonsudden-onset group, 25 episodes (62.5%) were initiated with a single ectopic beat, while 15 episodes (37.5%) were initiated with multiple complexes. The left ventricular ejection fraction of patients with nonsudden-onset PVT was lower (53 $\pm$ 6% versus 65 $\pm$ 7%, *P*<0.01) (Table 1). Nonsudden-onset tachycardias had a lower CI and PI, and a slower heart rate preceding VT than sudden-onset tachycardias. Similarly, the PVT cycle length was shorter in the nonsudden-onset cases (Table 2). When nonsudden-onset PVT episodes were further subclassified with respect to the morphology of the first tachycardic beat, 26 PVTs (65%) had a

Figure 1) Sudden-onset initiation of polymorphic ventricular tachycardia. Tachycardia was not preceded by ectopic beats

Figure 2) Short-long-short sequence and nonsudden-onset initiation of polymorphic ventricular tachycardia. Tachycardia was preceded by ectopic beats

first beat similar to and 14 (35%) had a first beat not similar to subsequent PVT beats.

DISCUSSION

Sustained VTs in the peri-infarction period occur in approximately 2% to 3% of patients with an ST elevation MI (5,6), and may develop due to transient arrhythmogenic phenomena in ischemic and infarcting tissue such as the following: abnormal automaticity, triggered activity, and re-entrant circuits created by heterogeneous conduction and repolarization (13-18).

Analysis of the PVT initiation patterns in our study demonstrated that the majority (65%) of PVT episodes detected in the CCU in patients with acute MI were characterized by nonsudden onset. Distinctive electrophysiological features of these PVTs with nonsudden onset include shorter CI and PI, faster tachycardia rate (shorter cycle length) and slower sinus rate preceding the PVT episode. With regard to clinical characteristics, patients with nonsudden-onset PVT had significantly lower left ventricular ejection fractions.

The finding that the majority of PVTs are characterized by nonsudden onset (65% of all PVT episodes) is in agreement with previous reports (19-23) on patterns of spontaneous VT onset studied by Holter monitoring or analysis of ICD electrograms. Two main patterns of VT onset have been reported: nonsudden-onset tachycardia – initiated by late-coupled ventricular ectopic beats with a short-long-short cycle – and sudden-onset tachycardia (19-22). The frequency of so-called nonsudden-onset VT has been reported to be 56% to 85% (19-22). Ventricular fibrillation episodes were initiated by either ventricular premature beats or a short-long-short cycle (23).

Most studies described patterns of monomorphic VT onset in patients with coronary artery disease, Chagas disease and ischemic or nonischemic cardiomyopathy with an ICD (2-4,19-22). Our study complements the results of previous reports by characterizing patterns of PVT initiation early after acute MI.

Clinical and electrophysiological features, such as left ventricular dysfunction and faster heart rate of PVT, in patients with acute MI resemble the characteristics of VTs reported in other patient populations, regardless of VT morphology (3-4,19-22). Patients with nonsudden-onset PVT had lower ejection fractions, which can be attributed to the fact that left ventricular dysfunction may serve as a substrate for arrhythmia.

Our findings of a shorter PI of the first beat that initiates the arrhythmia and slower heart rate preceding PVTs of nonsudden

onset are in accordance with a previous study (22) on characteristics of PVT initiation in patients who underwent ICD implantation. Taylor et al (20) demonstrated that PVTs, in comparison with MVTs, had shorter PIs and were preceded by bradycardia, postulating that the triggered mechanism may be responsible for a shorter PI in PVTs. Although we do not know the exact mechanism of differences in PI and preceding heart rate in our patient population, we speculate that, shortly after an MI, ongoing ischemia or reperfusion could induce PVT due to triggered activity with delayed depolarizations (13,14). At the same time, a slow heart rate preceding a nonsudden PVT episode might reflect the bradycardia-dependent re-entry mechanism in the setting of ischemia and infarction (15).

It should be noted that in the present study, we analyzed ECG strips obtained by printout from a continuous monitoring system for patients hospitalized in the CCU. Thus, analysis of sustained tachycardia initiation patterns using rhythm strips provides information that is as sufficient as Holter ECG recordings or pacemaker/ICD-stored electrograms.

Limitations

In the present study, rhythm strips were obtained either before or after reperfusion. In some patients, reperfusion strategies were not successful. Because our aim was simply to analyze the initiation pattern of PVT in acute MI using rhythm strips, we did not investigate the initiation pattern of PVT in patients undergoing PCI or in patients separately receiving thrombolytic therapy.

Data from patients who experienced episodes of both sudden- and nonsudden-onset tachycardia during their stay in the CCU were not included in the study because it would have skewed the statistical analysis.

CONCLUSIONS

The analysis of rhythm strips demonstrated that PVT is most often preceded by ventricular ectopic beats in the acute phase of MI, as previously shown for MVT. PVT with nonsudden onset is usually characterized by a lower CI and PI, a shorter PVT cycle length, a slower heart rate preceding arrhythmia and an associated lower ejection fraction. Our findings support previous investigations on the types of VT onset in different patient populations. However, additional studies are necessary for a better understanding of the mechanisms in those patients.

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