ORIGINAL ARTICLE

Premature Ventricular Complexes as a Trigger for Ventricular Fibrillation

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Introduction and objectives. The mechanisms that trigger ventricular fibrillation (VF) are poorly understood. The aim of this study was to analyze the initiation of VF in electrograms stored in implantable cardioverter-defibrillators (ICDs).

Methods. We analyzed ICD electrograms from patients who had suffered at least one episode of VF.

Results. Of 250 patients with ICDs, 13 (10 male and 3 female, age 49±22 years) had at least one episode of VF. The diagnoses were Brugada syndrome (n=4), ischemic heart disease (n=3), dilated cardiomyopathy (n=2), hypertrophic cardiomyopathy (n=1), short-coupled variant of torsades de pointes (n=1), endocardial fibroelastosis (n=1) and idiopathic VF (n=1). In 7 patients, VF was the reason for ICD implantation. Overall, 31 episodes of VF were recorded, including three episodes of arrhythmic storm. In the 7 patients who had more than one episode of VF (within minutes or up to 3 years apart), all episodes started with premature ventricular complexes (PVCs) that had the same morphology and similar coupling intervals. A short-long-short cycle was observed in 2 patients. In 21 episodes, PVCs that did not trigger VF were observed during sinus rhythm. There was no significant difference between them and PVCs that did trigger VF in terms of morphology, coupling interval (409±121 ms vs. 411±123 ms) or the preceding sinus rhythm RR interval (801±233 ms vs. 793±230 ms).

Conclusions. Spontaneous VF in the form of an arrhythmic storm or an isolated episode were triggered by PVCs. On occasions, PVCs preceded VF without triggering it.

Key words: *Defibrillator. Ventricular fibrillation. Sudden death.*

Extrasistolia ventricular desencadenante de la fibrilación ventricular

Introducción y objetivos. Los mecanismos de inicio de la fibrilación ventricular (FV) son poco conocidos. El objetivo de este estudio es analizar el inicio de la FV en los electrogramas almacenados en los desfibriladores automáticos implantables (DAI).

Métodos. Hemos analizado los electrogramas de pacientes con DAI y al menos un episodio de FV.

Resultados. De una población de 250 pacientes portadores de DAI, 13 tuvieron al menos un episodio de FV, 10 varones y 3 mujeres (edad, 49 ± 22 años), diagnosticados de síndrome de Brugada (n = 4), cardiopatía isquémica (n = 3), miocardiopatía dilatada (n = 2), miocardiopatía hipertrófica (n = 1), *torsades de pointes* por extrasístole ventricular (EV) con acoplamiento corto (n = 1), fibroelastosis cardiaca (n = 1) y FV idiopática (n = 1). En 7 pacientes la FV fue el motivo del implante. Se registraron 31 episodios de FV (3 tormentas arrítmicas). En cada paciente, todos los episodios comenzaron con una EV de la misma morfología y similar intervalo de acoplamiento en los 7 pacientes con más de un episodio (minutos-3 años). Se objetivó ciclo corto-largo-corto en 2 pacientes. En 21 episodios, se registraron EV en ritmo sinusal que no desencadenaron FV. La morfología, el intervalo de acoplamiento (409 ± 121 frente a 411 ± 123 ms) y el ciclo del latido sinusal precedente (801 ± 233 frente a 793 ± 230 ms) no presentaron diferencias significativas al compararlas con las EV inductoras de FV.

Conclusiones. La FV espontánea se desencadena por EV en las tormentas arrítmicas y en episodios aislados. En ocasiones las EV preceden a la FV sin desencadenarla.

Palabras clave: *Desfibrilador. Fibrilación ventricular. Muerte súbita.*

INTRODUCTION

The clinical study of ventricular fibrillation (VF) has always been impeded by its unpredictable occurrence and by the short time periods the patient can cope with it. Consequently, VF signal recordings are complicated and difficult to obtain

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ABBREVIATIONS

ICD: implantable cardioverter-defibrillator
PVC: premature ventricular complex
VF: ventricular fibrillation

and references in the literature to spontaneous VF with electrocardiogram (ECG) records are limited.[1] Recently, the successful ablation of premature ventricular complexes (PVCs) that trigger VF episodes in arrhythmic storm has been reported.[2-7] These cases stimulate our interest in understanding the mechanism that triggers VF. Implantable cardioverter-defibrillators (ICDs) enable us to store data on arrhythmic episodes and analysis of this data means our study of VF-trigger mechanisms can progress. To study the mechanism underlying the initiation of isolated VF or VF recurrence in arrhythmic storm, we analyzed ICD-recorded data of patients experiencing spontaneous VF episodes.

METHODS

We analyzed the VF records (near-field and far-field) of 250 patients with ICD implants received at a tertiary hospital between 1997 and 2008. Of these, 13 presented ≥1 VF episode. The ICDs analyzed belonged to the MINI and PRIZM series (Guidant, St. Paul, Minnesota, USA) and the GEM series (Medtronic, Minneapolis, Minnesota, USA). All ICDs were programmed to record and store the signal prior to the declared VF episode. We defined as VF those episodes with sinusoidal electrograms, with changeable morphology, cycle and width in which clearly-defined QRS complexes could not be discerned, and with true or pseudo-bipolar records with a characteristically fragmented signal. All episodes required electric discharge to finalize them, and we rejected those that ended of their own accord. We considered these were examples of polymorphous ventricular tachycardia and excluded them from the study. We defined the short-long-short cycle as the appearance of a (short) premature beat in the baseline rhythm, followed by a pause (with coupling interval >1.2-fold the preceding interval) and premature depolarization with (short) short coupling that triggers VF.

We analyzed morphology in the near-field and far-field channel, coupling interval, preceding sinus beat cycle, and the presence of a short-long-short sequence preceding premature beats that did (fibrillatory PVC) and did not (non-fibrillatory PVC) trigger VF. We used the paired Student *t* test to compare the coupling interval and the preceding sinus beat cycle length of both premature beats.

RESULTS

We recorded 31 episodes of spontaneous VF in 13 patients (10 men and 3 women; age, 49 [22] years). Patient diagnoses were: 4, Brugada syndrome; 3, ischemic heart disease; 1, hypertrophic cardiomyopathy; 2, dilated cardiomyopathy; 1, short-coupled variant of *torsades de pointes*; 1, endocardial fibroelastosis; and 1 idiopathic VF. Implantation was indicated for VF in 7 patients: 2 had ischemic heart disease; 2, Brugada syndrome; 1, hypertrophic cardiomyopathy; 1, endocardial fibroelastosis; and 1, short-coupled variant of *torsades de pointes*. We recorded a mean of 2.5 episodes/patient (1-12). Of 31 VF episodes, 14 were isolated and the rest were related with arrhythmic storm. Three patients presented arrhythmic storm. All episodes analyzed began with PVC, which was of the same morphology and had a similar coupling interval in the 7 patients with >1 episode (time between episodes ranging from minutes to 3 years) (Figures 1 and 2). The premature beat coupling interval range was 409 (121) ms and the preceding sinus beat cycle was 801 (233) ms. We found long-short cycle occurrence was related with VF onset in only 2 cases (9%). In 21 episodes, VF was preceded by PVC in sinus rhythm that did not trigger VF. The morphology, coupling interval (411 [123] ms) and preceding sinus beat cycle (793 [230] ms) presented no significant differences with respect to the PVC that triggered VF.

DISCUSSION

The PVC that precedes VF initiation has been known for some time and instances of primary VF in acute myocardial infarction have been described.[8] More recently, this has been believed to be the mechanism that triggers VF episodes in arrhythmic storm, and VF recurrence has been avoided by using ablation to eliminate it.[2-7]

In the pioneering studies of causes of ambulatory sudden cardiac death,[1] VF preceded by PVC only accounted for 8.28% and the vast majority of VF episodes (83.4%) were triggered by ventricular tachycardia. The magnitude of the difference between these results and those we obtained may be explained by the use of different methods of recording data. Bayés et al[1] reported on data collection by Holter monitoring; our study involved ICD recordings. We assume that in our patients possible episodes of ventricular tachycardia that might have degenerated into VF were successfully treated by ICD anti-tachycardia therapy, which has proven effective in a high percentage of ventricular tachycardia.[9,10]

In contrast to the onset of monomorphic ventricular tachycardia, which has been studied extensively in ICD recipients,[11] few studies have

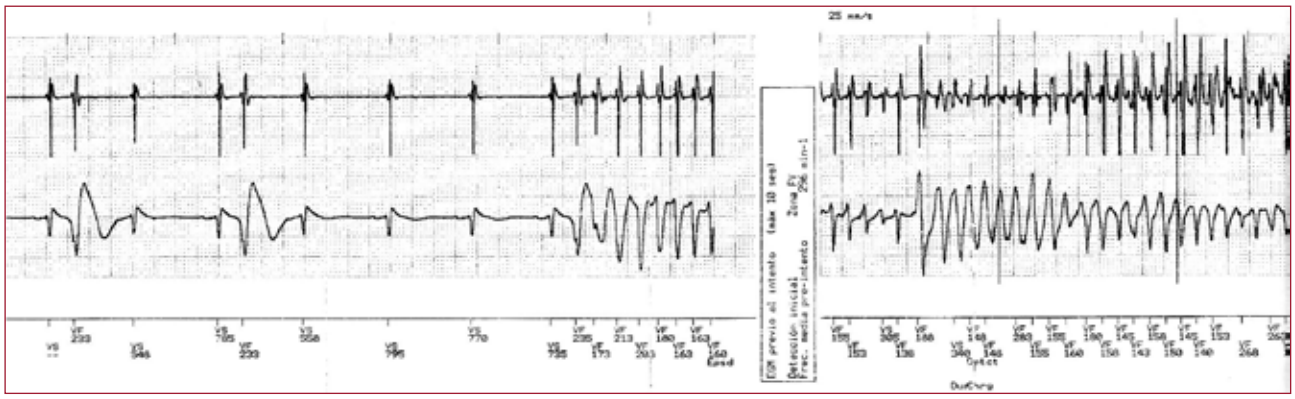

Figure 1. Onset of an episode of ventricular fibrillation in a patient with short-coupled variant of *torsades de pointes*.

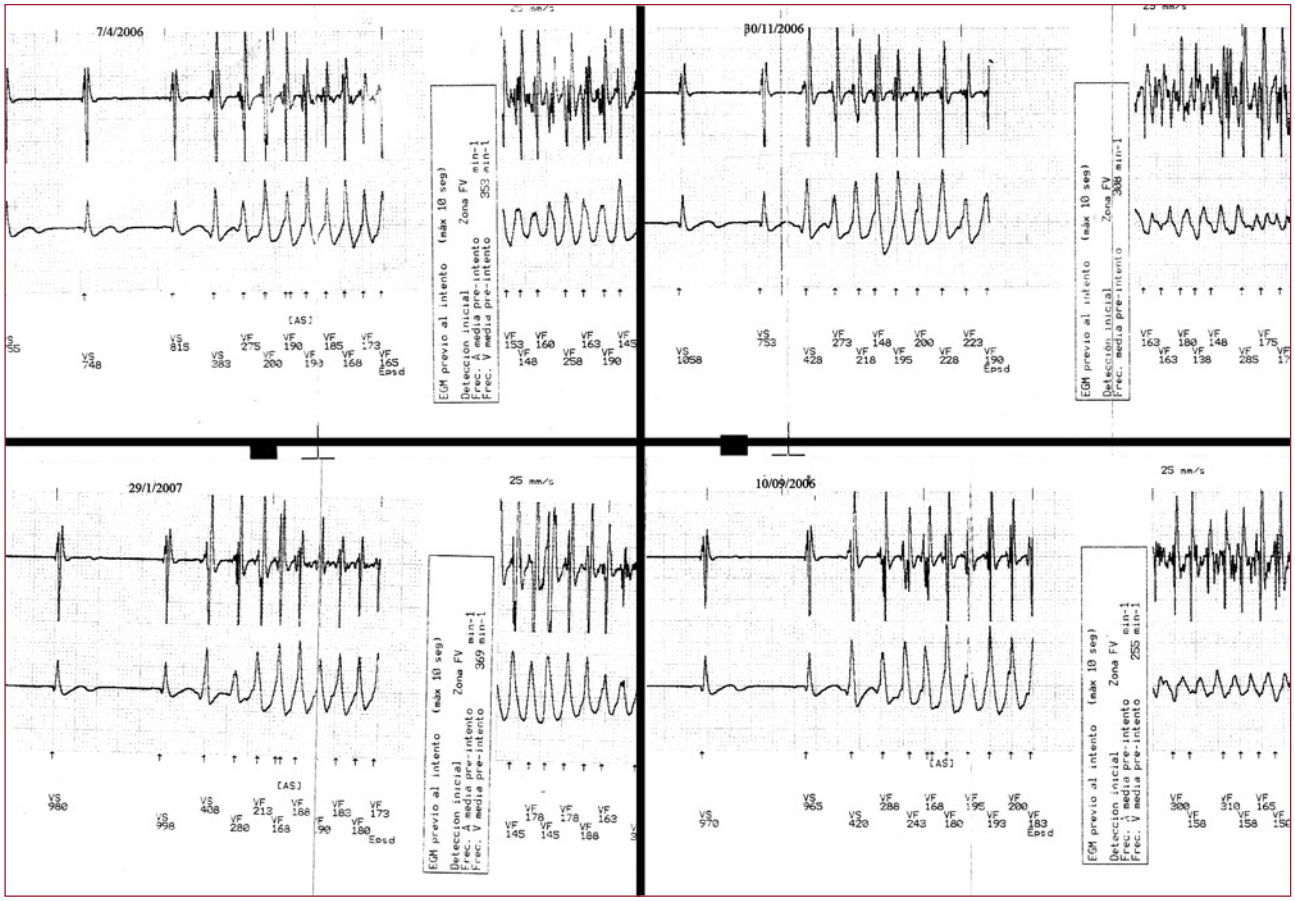

Figure 2. Onset of four episodes of ventricular fibrillation on different days corresponding to a patient with Brugada syndrome. Note the similar morphology of the premature ventricular complexes that trigger ventricular fibrillation.

analyzed VF initiation.[12,13] Among those that have, research in MADIT (Multicenter Automatic Defibrillator Implantation Trial) II-type patients, a study of primary prevention in patients with ischemic heart disease and ventricular dysfunction, stands out.[12] This study found that PVCs triggered 77% of VF episodes, in contrast to a short-long-short sequence found in 23% of patients (14 episodes out of 60).

In patients with Brugada syndrome, Kakishita et al[14] described PVC-triggered VF and included a figure that showed three episodes of VF, on three different dates, in which PVC morphology was markedly similar. We found the same consistency in VF-inducing PVC characteristics in our patients.

Although the pre-VF time recorded is brief, during non-VF inducing PVCs with similar morphologic and electric characteristics in terms of the coupling

interval, we have found we can distinguish between fibrillatory and non-fibrillatory premature beats that coincide in a short period of time. The registry of ambulatory sudden cardiac death also describes a significant increase in the number of PVCs in the hour before VF.[1]

We do not know why some premature beats trigger VF and others, with no obvious electrophysiological differences, do not. The fact that the PVC that triggers VF may at different times in the natural history of the patient have the same morphology, indicates that in each patient the premature beats that induce VF originate in the same way. This could be of interest in targeted VF ablation procedures, if we assume that it could be induced.

Limitations

The most important limitation of this study is the small number of patients included, which is partly due to the low incidence of spontaneous VF episodes.

Similarly, as only two channels are available (near-field and far-field) to analyze the episodes, difficulties in differentiation could arise in ventricular tachycardia fibrillation episodes with very fast cycles. The low number of long-short sequence initiated episodes means comparison of the two onset types' characteristics (coupling, preceding QT interval, T wave width and prior heart rate) is impossible.

Furthermore, the morphologic discrimination capability of the ICDs' stored memory channels prevents us from discounting the possibility that VF-inducing PVCs may differ slightly in VF recurrence episodes.

CONCLUSIONS

Spontaneous VF is triggered by PVCs both in cases of arrhythmic storm and in isolated episodes. Premature beats often precede VF but do not induce it. All this indicates that the VF-trigger mechanism has specific characteristics in each patient.

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