Recurrence of cardiac arrest after 14 years without ICD interventions: a VF cluster immediately after delivery

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One day after childbirth, a 29-year-old woman had several episodes of polymorphic ventricular tachycardia and ventricular fibrillation. She was rescued by an internal cardioverter defibrillator (ICD) which she had received 14 years ago, after out-of-hospital cardiac arrest, without recurrences until now. The electrocardiogram showed a normal QT interval, and ventricular premature beats, which seemed to arise from the same site. This case report illustrates that, even after years with freedom of ICD therapy, depleted devices still have to be replaced. (*Neth Heart J* 2008;16:242-5).

Keywords: implantable cardioverter defibrillator, pregnancy, arrhythmias, sudden cardiac death

Survivors of sudden cardiac arrest without structural heart abnormalities and with no evident underlying trigger usually receive an implantable cardioverter defibrillator (ICD) for secondary prevention. This case confirms that survivors are still at risk for recurrence of these life-threatening arrhythmias, even after many years.

Case report

A 29-year-old woman was referred to our hospital due to a threatening spontaneous premature partus at 27 weeks and 4 days. It was her first pregnancy. In 1990, she had survived an out-of-hospital cardiac arrest (OHCA) with ventricular fibrillation (VF). At that time, primary electrical disease was diagnosed, while no structural heart disease was found, although cardiomyopathy was suspected. The family history revealed no cardiac diseases, unexplained deaths or other

abnormalities. She had undergone an abdominal implantation of a single chamber ICD system, with two epicardial patches. In 1992, 1995 and most recently in 1999, she had undergone elective replacements due to normal battery depletion. The actual ICD at the time of referral was a Ventak Mini III (Guidant, St. Paul, Minnesota, USA). ICD interrogation at admission showed a single zone programming with cut-off rate of 270 ms with shock-only therapy, a Holter without any ICD treatments and a first-stage battery depletion status (MOL1). Until that moment she had not received any spontaneous ICD shocks for more than 14 years.

Despite maximal tocolytic therapy with adalat and prednisolone, she gave birth to a daughter at noon the next day. The morning after delivery, she fell out of her bed and suffered face injuries. ICD interrogation showed two correctly treated episodes of VF or polymorphic ventricular tachycardia (pVT) at the moment she fell out of her bed. One unrecognised but also treated VF/pVT episode had already occurred the evening before (figure 1, first 3 episode strips, numbers 9, 11 and 5 respectively). The frequency of the arrhythmias increased and she received several more ICD shocks. She was referred to the intensive care unit (ICU) where treatment with β -blockers (metoprolol) was started. In two days she received a total of seven full-energy shocks, before the arrhythmias faded away and finally disappeared (table 1). Four days after delivery, an echocardiogram was taken, which showed a structurally normal heart with some systolic impairment, and shortening fraction of 22%. She was able to leave the hospital after five more days without any further arrhythmias. A last predischARGE interrogation of the device revealed a total of 29 nonsustained episodes and seven sustained and treated VF/pVT episodes since hospital admission. The onset of the arrhythmias (figure 1) all seemed to be initiated with a short-coupled and morphologically identical ventricular premature beat (VPB) from the same region. Twelve-lead electrocardiogram (ECG) recordings from the ICU confirmed this assumption, and showed a vertical axis with aspecific ST-T changes (figure 2). The prematurity index of the VPB, calculated from

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Figure 1. Onset of the arrhythmias.

the saved RR intervals, ranged from 44.1 to 49.4% (table 1). The battery status of the ICD reached the elective replacement indicator, and replacement was performed. We were considering ablation of the focus

of the VPB, but the patient refused, as she did all other investigations. Up to now (with a follow-up of 46 months), there have been no recurrences of the arrhythmias.

Figure 2. ECG report displaying the onset of episode 32. Sinus rhythm with cycle length of 720 ms. Vertical axis, aspecific changes in the repolarisation. A ventricular premature beat initiates a polymorphic rhythm, which degenerates into ventricular fibrillation. In this 12-lead ECG, the coupling interval seems to be shorter than in the recorded electrograms.

Discussion

Recurrence of arrhythmias

This case report describes late recurrence of ventricular arrhythmias in a patient with a diagnosis of primary electrical disease. For these patients, recurrence of life-threatening arrhythmias after a very long period is not unique, and already well described by Wever and colleagues.¹ Patients without structural heart disease who survived a cardiac arrest keep an elevated risk for recurrences, and ICD implantation reduces cardiac mortality.¹⁻⁵

Pregnancy

The vast majority of pregnant women with an ICD tolerate and complete their pregnancies without any serious problems.^{6,7} Problems are often related to the use of antiarrhythmic drugs.⁷ Discontinuation of antiarrhythmic therapy during pregnancy can be the cause of recurrences as well. A significantly increased risk of arrhythmias remains present during the postpartum

period for up to nine months in the long QT syndrome (LQTS), and particularly LQTS-2.⁶ Beta-blocker therapy offers a significant reduction in ventricular arrhythmias, especially in this postpartum period.⁸ However, the QT and frequency-corrected QT (QTc) intervals from this patient were normal on all available ECGs, making this syndrome unlikely. This increased vulnerability is not described in the short-coupled variant of torsades de pointes, catecholaminergic polymorphic ventricular tachycardia or short QT syndrome.⁹⁻¹¹ This patient showed relatively short coupled VPBs, initiating pVT/VF. These last-mentioned syndromes were considered in this patient, and finally not upheld as the ultimate diagnosis, as the QT interval was not shortened, and the VPB coupling was not as extremely short as described by Leenhardt and colleagues.⁹ No β -agonists were used, which could have been responsible for sinus tachycardia, QT shortening or a rise in the number of VPBs.

Probable triggers of the postpartum development of ventricular tachycardias are abrupt cardiac remodel-

Table 1. Details of episodes saved in the ICD.

Episode	Comments	Treatment	VBP interval (ms)	Preceding RR interval (ms)	Calculated PI (VPB interval/preceding RR interval)
1	Induced	6 J	NA	NA	NA
5	Spontaneous	31 J	324	727	0.446
9	Spontaneous	31 J	324	734	0.441
11	Spontaneous	31 J	338	732	0.462
15	Spontaneous	Diverted	309	664	0.465
16	Spontaneous	31 J	309	646	0.478
25	Spontaneous	31 J	324	717	0.452
32	Spontaneous	Diverted	332	715	0.464
33	Spontaneous	31 J	328	664	0.494
34	Spontaneous	31 J	326	676	0.482
37	Spontaneous	Diverted	309	670	0.461

Details of episodes 1 to 37 (gaps in episode numbers indicate episodes that did not meet initial duration). PI=prematurity index, VPB=ventricular premature beat, J=Joule, NA=not available.

ling, changes in cardiac output and contractility, together with hormonal and autonomic nervous system alterations.^{6,8} As the echo showed only minor functional changes, these triggers were considered most likely in this case.

Analysis of ICD recordings

However, the recordings of the ICD were helpful to demonstrate that only one initiating focus was involved, probably in the left ventricle. This was suggested by the 12-lead ECGs from the ICU. VPBs, no matter how frequent, are regarded to be benign in patients without structural heart disease. The question why this VPB initiated the arrhythmia and why some episodes were nonsustained is still not clarified.¹² Analysis of the prematurity index did not help in this respect, as some of the longest coupled VPBs led to sustained arrhythmias. As mentioned, ablation of the VPB trigger, leading to these VF/pVT episodes, was considered, but the patient has refused up to now.¹³

Conclusion

The period around childbirth bears an increased risk for recurrent arrhythmias. This was also the case for our patient in whom a cardiomyopathy was suspected, but not proven. The fact that all episodes of VF/pVT originated from one focus was clearly illustrated by the electrograms of this device, which intervened for the first time after 14 years of silence. This is an additional argument for the diagnosis of idiopathic VF. ICD replacement in survivors of OHCA without evident structural heart disease and many years without ICD interventions, is necessary.

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