Case Reports

Is There an Effect of Amiodarone on the Defibrillation Threshold?

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SUMMARY

The interaction between amiodarone and the defibrillation threshold (DFT) is still controversial. We present a case with dilated cardiomyopathy and recurrent sustained monomorphic ventricular tachycardia who received an automatic implantable cardioverter defibrillator (AICD) while under long-term amiodarone treatment. AICD implantation was performed without thoracotomy. The transvenous lead was inserted via a left subclavian vein puncture and the patch was placed on the lateral chest wall, submuscularly. At the time of implantation a 35J shock was not successful in converting ventricular fibrillation to sinus rhythm, but a 40J rescue shock was successful. After discontinuation of amiodarone, DFT measurements were repeated. Sixteen days later DFT was still higher than 34J, but 71 days later it decreased to 20J. (Jpn Heart J 34: 221-226, 1993.)

Key Words:
Amiodarone Defibrillation threshold

The issue of the defibrillation threshold (DFT) and amiodarone is still unresolved. Some studies have suggested that patients on long-term amiodarone treatment have significantly elevated DFTs.1-4) Others have not found this increase in DFT or energy requirements for successful defibrillation.5,6)

In this report, we present a case with dilated cardiomyopathy and sustained monomorphic ventricular tachycardia (VT) who received an automatic implantable cardioverter defibrillator (AICD) while under long-term amiodarone treatment.

CASE REPORT

A 27-year-old male patient was referred to our clinic for treatment of...
sustained monomorphic VT and congestive heart failure. His first complaint (palpitation) began in 1987; also his first VT episode was documented in the same year and was treated with β-blocker and quinidine. He remained well until June 1990 when he was admitted repeatedly to hospital because of VT episodes which were treated with direct current (DC) shock cardioversion. After discharge the patient experienced a gradual onset of dyspnea and edema in the lower extremities. Ten days before admission to our department, VT recurred and he was treated with DC shock. VT recurred again one day after DC shock. Lidocaine and propafenone were not effective for conversion of VT.

Physical examination revealed a hypotensive and dyspneic man with a pulse rate of 137/min. Jugular venous distention was present. The heart was enlarged and a S3 sound was heard. Auscultation of the chest revealed inspiratory rales in the lower half of the lung fields bilaterally. The liver had descended 10 cm below the right costal margin. Peripheral edema was present.

A resting 12-lead electrocardiogram showed monomorphic VT with a rate of 137 per minute (cycle length was 438 msec, Fig. 1). Chest roentgenogram revealed cardiomegaly. There were no metabolic or electrolytic (including magnesium) abnormalities. Serum digoxin level was normal. He was treated with diuretics, oxygen, digoxin and lidocaine initially. Then he underwent DC shock cardioversion to sinus rhythm, but after a few sinus beats VT recurred and persisted. Administration of quinidine, diphenylhydantoin, mexiletine and intravenous amiodarone were not effective for conversion of incessant VT to sinus rhythm. Electrophysiological studies were performed while he was receiving oral amiodarone. Ventricular tachycardia was easily terminated with three ventricular extrastimuli and sustained VT was induced with single ventricular extrastim-
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ultra from the right ventricular apex. Spontaneous VT recurrence was also present under oral amiodarone treatment. In these VT episodes, tachycardia cycle lengths were 480 msec (125 bpm) and 462 msec (130 bpm). On echocardiographic study all cardiac chambers were enlarged (left ventricular end-diastolic dimension 3.3 cm/m² [normal: <3.2 cm/m²]; right ventricular dimension 2 cm/m² [normal: 0.4–1.4 cm/m²]; left atrial dimension 2.3 cm/m² [normal: 1.2–2.2 cm/m²]) and wall motions were diffusely hypokinetic. Cardiac catheterization and coronary angiography revealed generalized hypokinesia in the ventricles and normal coronary arteries. Ejection fraction was 32%. During VT intrarterial systolic blood pressure dropped from 103 to 78 mmHg. All these findings were compatible with dilated cardiomyopathy.

We implanted an AICD with antitachycardia pacing capability (Ventak PRx 1705, CPI, St. Paul, MN, USA) under fentanyl anesthesia without thoracotomy using a transvenous lead (Endotak C0062, CPI) and submuscular patch (Endotak SQ 0063, CPI) as described elsewhere in the literature.7),8) Defibrillation threshold measurement was done using an external cardioverter defibrillator (Ventak ECD, model 2806, CPI). Ventricular fibrillation was induced with an external AC fibrillator. During implantation spontaneous clinical VT was converted to sinus rhythm with a 10J shock by the device. We could not find an acceptable DFT. Defibrillation threshold was less than 40J but it was above 35J. In spite of the high DFT we implanted the device because this patient had never experienced a ventricular fibrillation episode and his VT conversion threshold was at most 10J and probably less (we did not go down to lower values for measurement) and its rate was not high. Also, according to published reports amiodarone might be the cause of high DFT.

After implantation the device was programmed to monitor only mode for a week. We also stopped the amiodarone treatment 8 days after the implantation. During this period of time the patient was under continuous rhythm monitoring in the coronary care unit. Twenty-four days after implantation, an electrophysiologic study was performed using the device and an external stimulator. Induced clinical VT episodes were easily terminated by the device (burst pacing). Also, VT conversion to sinus rhythm was achieved with a 1J shock. Ventricular fibrillation induction for DFT measurement was difficult. DFT was between 34–40J. Two months after the implantation one spontaneous VT episode was converted by the antitachycardia pacing function of the device on the first attempt. The patient was not aware of this episode. The last DFT measurement was performed 71 days after cessation of amiodarone treatment. During this measurement, a bipolar catheter was inserted percutaneously and placed with its tip at the right ventricular apex, and ventricular fibrillation was induced with an AC fibrillator using this catheter. DFT was reduced to 20J. The measurement was
repeated only twice.

**DISCUSSION**

Defibrillation threshold is an important issue in AICD implantation. Some antiarrhythmic drugs have an effect on DFTs (e.g. encainide, lidocaine, amiodarone, bretylium, sotalol). The most significant DFT interaction has occurred in patients receiving amiodarone. Fogoros reported a patient with high DFT when taking amiodarone. Three months after discontinuation of amiodarone, DFTs were markedly reduced (10J). Manolis et al found that a patient under amiodarone treatment had a DFT greater than 40J, despite the use of two large patches. Amiodarone was also found to be an independent predictor of high DFTs by Troup et al at the time of initial AICD implantation (p<0.05). Kelly et al found the defibrillation threshold to be significantly higher in patients who had received amiodarone within 1 month of implantation as compared with the threshold in patients who had not. In their 5 patients who received amiodarone within 1 month before surgery, adequate DFT was not achieved despite various lead positions and configurations. One of these patients died postoperatively (DFT>40J). In 3 of these patients DFT was <15J after 6 to 10 weeks without amiodarone therapy. In the fourth patient there was no change from the initial threshold of >40J and he did not receive a device.

However, there are some conflicting results about the effect of amiodarone on DFT. Fain et al evaluated the effect of intravenous and chronic oral administration of amiodarone on DFTs in dogs. They found that the energy required for successful defibrillation was decreased by acute intravenous amiodarone while chronic oral administration had no significant effect on DFT. While reevaluating DFTs during generator replacement in 23 patients, Guarnieri et al found striking increases in DFTs in patients taking amiodarone (from 10.9±4.3J at implantation to 20.0±4.7J at replacement). They found no significant differences in DFTs between the patients who received amiodarone and those who did not receive amiodarone at initial implantation. This discrepancy was explained by the fact that their patients were studied in the early loading phase of amiodarone treatment.

Our patient was under amiodarone treatment for 78 days until AICD implantation. Amiodarone was given at a dose of 800 mg/day at the beginning of therapy. Approximately 1 month after the beginning of amiodarone treatment, we tried to decrease the amiodarone dose. For this purpose we administered 600 mg/day for 4 days and 400 mg/day subsequently for 4 days. But because of the VT recurrences we had to then increase the amiodarone dose to 800 mg/day. During the 10 days before the implantation the doses of amiodar-
one were as follows; 800 mg/day for 2 days and 600 mg/day for 8 days. Eight days after implantation, amiodarone was discontinued and 16 days later (24 days after implantation) DFT was measured. DFT was still higher than 34J. Seventy-one days after discontinuation of amiodarone, DFT was reduced to 20J. There was no major factor that might change DFT in this patient. Chest x-ray showed a slight decrement in the heart size, but electrode position in the heart was not changed. On echocardiographic study the heart chambers were slightly decreased in diameter (left ventricular end-diastolic dimension from 3.3 to 3.0 cm/m²; right ventricular dimension from 2.0 to 1.7 cm/m²; left atrial dimension from 2.3 to 2.0 cm/m²). It is difficult to explain the improvement in DFT with these small changes in heart size.

Contrary to previous reports, Huang et al reported that amiodarone had no significant effect on DFT. In their study 28 patients were taking amiodarone up to the time of surgery, the mean DFT in this group was 12.0±4.4J (range 5 to 20). In 32 patients who were not taking amiodarone mean DFT was 12.3±5.5J (range 5 to 30).6)

Grubb et al reported that in 6 patients who were not on amiodarone treatment, an adequate DFT could not be found at the time of initial epicardial electrode implantation. After 10–15 days, DFT values decreased to adequate levels (mean 18J). The authors suggested that this finding may have been due to better electrode contact with the myocardium or the absence of recent cardioplegia and cardiopulmonary bypass.11) As we have discussed previously we could not find any decrease in DFT 24 days after implantation of AICD. Thus, we feel that amiodarone was the cause of the high DFT in our patient and 71 days were enough to correct its effect.

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REFERENCES


