Frequency and Implications of Ischemia Prior to Ventricular Tachyarrhythmia in Patients Treated With a Wearable Cardioverter Defibrillator Following Myocardial Infarction

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ABSTRACT

Background: Autopsy studies imply that recurrent myocardial infarction (MI) accounts for the majority of sudden death early after acute MI, rather than primary arrhythmia. However, diagnosis of recurrent MI by autopsy is challenging and excludes electrocardiographic data to adjudicate arrhythmic causes. We examined the frequency of ischemia prior to treated ventricular tachycardia/fibrillation (VT/VF) and outcomes in patients using the wearable cardioverter defibrillator (WCD) following acute MI.

Hypothesis: Primary arrhythmia, rather than ischemia, is a frequent contributor to sudden death following MI.

Methods: All patients treated for VT/VF over a 6-year period while wearing a WCD following acute MI with advanced left ventricular dysfunction (ejection fraction $\leq 35\%$) were included. Patients with ST-segment changes ≥ 0.1 mV before VT/VF were classified ischemic. Demographics and clinical outcomes were compared between those with ischemia-mediated vs primary arrhythmia.

Results: Among 273 patients fulfilling study criteria, 15.4% had ischemia prior to VT/VF. Clinical and WCD use characteristics did not significantly differ between ischemic and primary VT/VF groups. Termination of VT/VF by WCD treatment approximated 96% in both groups. Survival 24 hours post-treatment was 88% and 84% ($P = 0.54$) for patients with and without ischemic VT/VF, respectively. Furthermore, 30-day cumulative survival for those with and without ischemic VT/VF was 77% and 70%, respectively ($P = 0.57$).

Conclusions: Ischemia is an infrequent cause of VT/VF following MI, contradicting previous study conclusions that recurrent MI is responsible for most post-MI sudden death. Etiology of VT/VF, however, did not influence defibrillation success or survival, which was high for both groups.

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Introduction

Following myocardial infarction (MI), patients remain at substantial risk for ventricular arrhythmias and sudden cardiac death (SCD).^{1–3} Despite the widespread use of revascularization and pharmacological therapies, this risk is accentuated early after MI, especially in patients with left ventricular dysfunction.^{4–7} Indeed, in the VALsartan IN acute myocardial infarction Trial (VALIANT), the rate of SCD was 2.3% within 30 days following MI.⁶

Although early implantable cardioverter-defibrillator (ICD) implantation would be expected to improve outcome in such patients, clinical studies suggest otherwise. In the Defibrillator IN Acute Myocardial Infarction Trial (DINAMIT) and the Immediate Risk stratification Improves Survival (IRIS) trials, early ICD implantation following MI had neutral survival impact, because the reduced risk of SCD was offset by an increased risk of nonarrhythmic mortality.^{8,9} To inform these results, a substudy of the VALIANT trial reported frequency of autopsy-defined causes of sudden death such as recurrent MI or cardiac rupture—rather than primary arrhythmia—to be highest in the early post-MI period.¹⁰ Other studies have also implicated ischemia as a predictor of arrhythmic events.¹¹ Although these observations were suggested to explain lack of benefit observed in early post-MI ICD trials, diagnosis of recurrent MI by autopsy may be challenging when death occurs soon after the index MI. Furthermore, characterization of myocardial ischemia such as ruptured plaque, coronary thrombus, or even coronary vasospasm that might contribute to ventricular tachyarrhythmias (ventricular tachycardia/ventricular fibrillation [VT/VF]) may be elusive to pathologic examination. Altogether, although autopsy may suggest recurrent MI as a major cause of SCD, it is uncertain whether VT/VF triggered by ischemia is the principal cause of SCD in the immediate post-MI period. To this purpose, we examined the influence of ischemia prior to treated VT/VF and subsequent clinical outcomes in patients using the wearable cardioverter-defibrillator (WCD) following acute MI.

Methods

All patients prescribed a WCD in the United States entered into a database maintained by the manufacturer (ZOLL, Pittsburgh, PA) for regulatory, reimbursement, and administrative purposes. All patients signed consent to use their data for quality monitoring, health care operation activities, and/or research. Patients for this study were identified initially from a database that exclusively contained patients treated for sustained VT or VF while wearing the device between May 2008 and May 2014. Then, these data were adjudicated to select only those patients who were assigned an *International Classification of Diseases, Ninth Revision* diagnostic code of 410.XX (index acute MI) for prescription of the WCD.

Patient use was calculated using data stored by the WCD monitor. Clinical data were extracted from the medical documents supplied to ZOLL for reimbursement purposes. Wearable cardioverter-defibrillator 2-lead electrocardiograms (ECGs) recorded during WCD fitting (baseline) were compared with ECG recordings before the onset of

the treated VT/VF event for ST-segment changes. For the purposes of this study, if a patient was treated multiple times during a 24-hour period, only ECGs associated with the first treatment were used for comparison with the baseline. The criteria for ischemia were any ST-segment depression of ≥ 0.1 mV at 80 ms after the J point or ST-segment elevation of ≥ 0.1 mV.¹² All ECG recordings were analyzed by a ZOLL ECG technician and further reviewed by one of the authors (R.P.). End-of-use reasons from device returns as well as calls received after treatment events were used to evaluate mortality outcomes.

Wearable Cardioverter-Defibrillator Description

Details of the WCD components and arrhythmia algorithm are described elsewhere.¹³ Briefly, the monitoring electrodes are held in place circumferentially around the chest by tension from an elastic belt to provide 2 nonstandard, orthogonal, surface ECG leads, front-back and side-side. One defibrillation electrode is placed in an apical position, and the remaining 2 defibrillation electrodes are placed posteriorly. If an arrhythmia is detected, an escalating alarm sequence starts, including vibration against the skin, audible alarms, and a voice cautioning bystanders of an impending treatment. During these alarms, patients are trained to hold a pair of response buttons that acts as a test of consciousness. If no response occurs, the monitor charges, extrudes gel from the defibrillation electrodes, delivers up to five 150-J biphasic shocks, and records the ECG.

Statistical Analysis

Data are presented as mean $\pm$ SD and as median (range) for skewed distributions. Data were analyzed using *t* tests or Mann-Whitney *U* tests for continuous variables and χ^2 tests for discrete variables. Thirty-day cumulative survival curves were generated according to Kaplan-Meier analysis. Multivariate logistic regression was used to identify factors associated with survival. For purposes of this study, all ventricular tachyarrhythmias and their corresponding shocks occurring within 24 hours of the index arrhythmia were considered as 1 event (“shock events”). Data were considered statistically significant using a 2-sided *P* value < 0.05 . All analyses were conducted using SPSS version 22 (IBM Corp., Armonk, NY) and XLSTAT version 2014.4 (Addinsoft, New York, NY).

Results

Between May 2008 and May 2014, 273 post-MI patients were treated appropriately for sustained VT or VF while wearing the WCD. Of these patients, 42 (15.4%) exhibited ST-segment changes ≥ 0.1 mV before the onset of VT/VF (Figure 1), indicating an ischemic etiology. The ST-segment changes for ischemic VT/VF patients averaged 0.3 ± 0.2 mV, and ST-segment depression was present in 86% of these patients; conversely, 14% of patients experienced ST-segment elevation.

Similar clinical characteristics were observed in patients with and without ischemic VT/VF (Table 1). Overall, approximately 80% of the patients were male, with a mean age of 64 years. In both groups, patients had advanced

Figure 1. Side-side (SS) and front-back (FB) ECG recordings from the WCD. (A) Baseline recording when the WCD is first worn; (B) recording showing ST-segment depression and QRS widening before the onset of VT. Abbreviations: ECG, electrocardiographic; VT, ventricular tachycardia; WCD, wearable cardioverter-defibrillator.

Table 1. Baseline Clinical Characteristics

	With Ischemic VT/VF	Without Ischemic VT/VF	P Value
Demographics and Clinical History			
Patients, n(%)	42 (15.4)	231 (84.3)	—
Sex, % Male	79.5	79.2	0.92
Age, y (mean $\pm$ SD)	68.2 $\pm$ 11.9	64.3 $\pm$ 11.9	0.06
LVEF, % (mean $\pm$ SD)	27.6 $\pm$ 11.5	25.5 $\pm$ 7.9	0.27
Previous MI (%)	26	22	0.52
DM %	43	44	0.87
HF	60	52	0.37
Hyperlipidemia	55	40	0.08
Hypertension	40	30	0.17
Revascularization	79	69	0.20
Medications	N = 28	N = 154	
ACEIs/ARBs	54	49	0.68
β -Blockers	79	79	0.62
Diuretics	64	51	0.21
Vasodilators	18	27	0.3
Statins	75	68	0.43

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; DM, diabetes mellitus; HF, heart failure; LVEF, left ventricular ejection fraction; MI, myocardial infarction; SD, standard deviation; VT/VF, ventricular tachycardia/ventricular fibrillation.

Data are presented as n (%), % alone, or mean $\pm$ SD.

left ventricular dysfunction characterized by a mean left ventricular ejection fraction (LVEF) of 27.6% and 25.5% in patients with and without ischemic VT/VF, respectively. More than half the patients in each group had a history of heart failure. Revascularization was performed in 79% of the patients with ischemic VT/VF and in 69% of the patients without ischemic VT/VF ($P=0.20$). In addition to the index MI for which the WCD was prescribed, $\geq 20\%$ of the patients in both groups had a history of previous MI. Established risk factors for cardiovascular disease such as diabetes, hyperlipidemia, and hypertension were also prevalent. Medication information was available for 67% of the patients (Table 1). Patients in both groups showed similar treatment with β -blockers, statins, diuretics, and angiotensin-converting enzyme inhibitors and/or angiotensin II receptor blockers.

For all patients, the median time from index MI to the WCD prescription was 4 days (range, 0–38 days), with no significant difference between groups ($P=0.79$). Daily use for each patient was calculated by dividing the total hours worn by the number of days worn minus one, to adjust for partial use on the first and last day of wear. There was no statistically significant difference ($P=0.85$) between

Figure 2. Time by months from the index MI to time of shock events. Abbreviations: MI, myocardial infarction; VT/VF, ventricular tachycardia/ventricular fibrillation.

the median daily use of 23.7 hours (range, 16.7–24 hours, $N=39$) in patients with ischemic VT/VF and the median daily use of 23.6 hours (range, 1.2–23.9 hours, $N=205$) in patients without ischemia VT/VF.

For patients with ischemic VT/VF, median time from the index MI to first shock delivery was 18.5 days (range, 5–126 days), which did not statistically differ compared with 16 days for patients without ischemic VT/VF (range, 2–226 days; $P=0.37$ for comparison). Approximately 70% of patients in both groups were treated within 1 month of the index MI (Figure 2), and $>90\%$ of the patients in both groups were treated within 3 months of the index MI.

Forty-two ischemic VT/VF patients had 47 shock events, during which they received 106 appropriate shocks. Of these shocks, 103 successfully terminated VT/VF and the remaining 3 were unsuccessful, yielding a shock conversion success rate of 97%. For those patients with unsuccessful shocks, 2 spontaneously converted to a slower rhythm and the third received additional resuscitation from bystanders. All 3 patients survived ≥ 24 hours post-treatment. For the group with ischemic VT/VF, 56% of the shocks were delivered when the rhythm prior to treatment was sustained VT, and the remaining shocks treated a VF rhythm. The average number of shocks per patient in this group was 2.5 ± 3.5 (range, 1–19), with 47% of these patients treated by ≥ 2 shocks. There were 37/42 patients who survived ≥ 24 hours post-treatment, yielding a survival rate of 88%.

A total of 231 patients without ischemic VT/VF had 243 shock events, during which they received 490 appropriate shocks. Of these shocks, 472 successfully terminated VT/VF, 8 were unsuccessful, 4 led to asystole, and the treatment outcomes were unknown in 6. Thus, the shock conversion success rate in this group was 96%, which did not significantly differ compared with the ischemic VT/VF group ($P=0.67$). For the 8 patients with unsuccessful shocks, 5 received advanced resuscitation from emergency responders, 2 had their device disconnected, and 1 spontaneously converted to a slower rhythm. Five of these 8 patients survived ≥ 24 hours post-treatment. For the group without ischemia VT/VF, 52% of the shocks were delivered when the underlying rhythm was a sustained VT, which was also not significantly different from the ischemic

Figure 3. Thirty-day cumulative survival for the 2 groups from the start of WCD use. Abbreviations: VT/VF, ventricular tachycardia/ventricular fibrillation; WCD, wearable cardioverter-defibrillator.

VT/VF group ($P=0.47$). There was a nonsignificant trend ($P=0.06$) toward fewer patients without ischemic VT/VF (33%) being treated by ≥ 2 shocks. The average number of shocks per patient was 2.1 ± 3.3 (range, 1–36). The 24-hour post-treatment survival rate of 84% (195 of 231 patients without ischemic VT/VF) was not significantly different from the 24-hour survival in the ischemic VT/VF group ($P=0.54$).

Reasons for WCD end of use were used for survival analysis. In the ischemic VT/VF group, end-of-use reasons included ICD therapy (25 patients, 59.5%), death (12 patients, 28.5%), and other (5 patients, 12%). Among patients who died, the median time from start of WCD use to death was 12.5 days (range, 3–120 days). Likewise, in the group without ischemic VT/VF, 135 patients (58.5%) received an ICD, 59 patients (25.5%) died, and remaining 37 (16%) discontinued use for other reasons ($\chi^2=0.52$, $P=0.77$). In this group, the median time to death from the start of WCD use was 17 days (range, 1–195 days). Because the risk of SCD was the highest during the first month of WCD use, survival was evaluated for the first 30 days since the start of WCD use (Figure 3). The actuarial survival analysis of those with and without ischemic VT/VF showed that in the 30-day interval following the start of WCD use, the cumulative survival was 77% and 70%, respectively ($P=0.57$). Multivariate regression showed that a history of revascularization was associated with better survival after treatment (odds ratio: 2.72, 95% confidence interval: 1.42–5.24, $P<0.01$). Additionally, electrical storm, defined as ≥ 3 WCD shocks over a 24-hour time period, was associated with lower survival (odds ratio: 0.32, 95% confidence interval: 0.12–0.90, $P=0.03$). Presence of ischemia prior to onset of an arrhythmia and other additional variables such as age, sex, LVEF, days from MI to treatment, and cardiovascular risk factors were not associated with divergent survival (Figure 4).

Discussion

Among patients following MI, ischemia is an infrequent cause of ventricular arrhythmia events. This study shows that 15% of subacute MI patients with left ventricular dysfunction displayed ST-segment changes, indicative of

ischemia, before the onset of a VT/VF event. The WCD was very successful treating VT/VF, irrespective of whether or not evident ischemia played a role in the onset. The risk of VT/VF was highest in the first month from the index MI in patients both with and without ischemic VT/VF, with the median time until first treatment of approximately 2 weeks. In both groups, $\sim 70\%$ of the patients received a treatment within the first month of their index MI and $>90\%$ in the first 3 months of their index MI. Similar clinical characteristics and survival at 24 hours post-treatment and during the 30-day interval following WCD application were observed in both groups.

Worldwide, tens of thousands of WCDs are used for temporary (ie, weeks to months) SCD prevention across all indications, ranging from ICD explantation to high-risk primary prevention patients awaiting qualification criteria for an ICD. As current guidelines recommend ICD implantation waiting periods of 40 days after an MI if no revascularization is performed or 90 days when revascularization is performed,^{14,15} the WCD is frequently used to protect subacute MI patients with left ventricular dysfunction (LVEF $\leq 35\%$). The risk of SCD during these ICD waiting periods is not insignificant, and observational studies have demonstrated WCD success in treating potentially fatal arrhythmias.^{13,16–18} In the largest post-MI dataset, Epstein and colleagues reported that 1.6% of 8543 patients wearing the WCD received appropriate treatment for life-threatening ventricular arrhythmias and that inappropriate shocks occurred at a rate of 0.006 per patient month of use.¹⁷ Additionally, cost-effectiveness models have shown WCD use after MI for high-risk patients to be economically attractive.¹⁹

To place the data from the current study in perspective, our results are compared with the autopsy substudy from VALIANT.¹⁰ This subgroup analysis revealed that of 105 autopsied subjects who experienced SCD, 28 (26%) had died due to recurrent MI, 13 (12%) due to cardiac rupture, and 3 (2.8%) due to the index MI. The remaining 54 (51%) patients were presumed to have had an arrhythmic cause of death, as there was no specific autopsy evidence of another acute pathological process. Given the absence of ECG recordings at the time of death, the actual number of

Figure 4. Forest plot of ORs for survival as function of clinical, demographic, and WCD factors, with OR for each factor (circle) and its corresponding 95% CI (whiskers). Vertical dotted line represents the no-effect line. Abbreviations: CI, confidence interval; HF, heart failure; Hx, history; LVEF, left ventricular ejection fraction; MI, myocardial infarction; OR, odds ratio; Revasc, revascularization; WCD, wearable cardioverter-defibrillator.

arrhythmias at the time of death remains unknown. It seems likely that a number of the deaths classified as recurrent MI during autopsy may actually be from fatal arrhythmias induced from acute ischemia caused by plaque rupture, thrombosis, or coronary vasospasm. In the present study, only 15% of patients showed signs of recurrent ischemia prior to the onset of a life-threatening VT/VF. Of note, despite the antecedent ischemia, VT/VF remains treatable by defibrillation and survival is high.

Additional results from the VALIANT autopsy study suggest that recurrent MI/myocardial rupture was highest in the early post-MI period, whereas the risk of SCD by presumed arrhythmic causes became predominant only after a few months (20% presumed arrhythmic deaths in the first month, rising to 75% after 3 months). The present study shows instead that 73% of treated patients had a life-threatening arrhythmic event within the first month of their index MI, and >95% of treated patients had a treated VT/VF event within 3 months, similar to what Epstein and colleagues first reported among early post-MI patients using a WCD.¹⁷ They presented the risk of SCD as highest in first month of WCD use, and 1.6% of the post-MI WCD patient population was appropriately treated.

Ambulatory ECG monitoring studies report the incidence of ischemic ST-segment changes before fatal arrhythmias to occur between 12.6% and 52% of cases.^{20,21} Results of the present study show that 15% of the post-MI patients presented with ischemic ST changes before the onset of life-threatening VT/VF, consistent with the lower range of monitored VT/VF reports. Although a higher rate of ischemia preceding VT/VF in patients with recent MI

might be expected, the lower incidence of ST changes in WCD patients in this study may be partly explained by the high rates of revascularization, as >70% of the patients underwent coronary revascularization. Secondly, though ischemia is one factor in initiating arrhythmias, the onset of VT/VF not related to an ischemic event is generally viewed as a multifactorial, probabilistic process involving a complex interplay of myocardial scar formation, electrical heterogeneity, progressive heart failure, and genetic factors,^{22,23} all of which converge in the postinfarction period.

Interestingly, results from the present study show that ischemia prior to the onset of VT/VF was not associated with poor survival. However, electrical storm (recurrent VT/VF) did negatively affect survival. Presence of electrical storm may indicate a deteriorating myocardial substrate conducive to triggering and maintaining repeat arrhythmic events.²⁴ Higher mortality rates associated with electrical storm have also been observed in ICD studies such as the Antiarrhythmic Versus Implantable Defibrillators (AVID) trial²⁵ and the Multicenter Automatic Defibrillator Implantation Trial II (MADIT-II).²⁶

Study Limitations

The study is retrospective in nature and only includes VT/VF occurring during device use, and we did not evaluate the influence of ischemia on SCD due to other arrhythmic events. Epstein and colleagues in their aforementioned study reported bradycardia deaths in 0.4% of their patients.¹⁷ If the primary cause of bradycardia death in this patient group is recurrent MI and cardiac rupture, this

bias could skew SCD etiology closer to VALIANT's autopsy results.

Ischemia was evaluated using a 2-lead orthogonal ECG system, rather than the standard 12-lead ECG, which may have led to fewer patients being identified with ischemic VT/VF. In addition, ischemia may have been the trigger of VT/VF even without visible ST-segment changes; on other hand, ST-segment changes may occur without true ischemia.

Because the current analyses were derived from the manufacturer's database, patient medical and demographic information was limited to that required from medical necessity and reimbursement processing requirements.

We also did not include data from patients who died while not wearing their WCD, as no ECG information was available for analysis. Data were not collected after the device was returned, resulting in incomplete records of deaths (sudden or nonsudden) in the subacute MI period.

Conclusion

In early post-MI patients who were treated by the WCD, ischemic ST-segment changes before the onset on ventricular tachyarrhythmia were present in only 15% of the patients. Ischemia is an infrequent cause of VT/VF following MI, contradicting previous study conclusions that recurrent MI is responsible for most post-MI sudden death. Etiology of VT/VF, however, did not influence defibrillation success or survival, and defibrillation success and survival were high for both groups.

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