Polymorphous Ventricular Tachycardia Associated With Acute Myocardial Infarction

Christopher L. Wolfe, MD, FACC; Carleton Nibley, MD; Anil Bhandari, MD, FACC; Kanu Chatterjee, MB, FRCP, FACC; and Melvin Scheinman, MD, FACC

Background. During a 2.9-year period, 11 patients developed polymorphous ventricular tachycardia 1–13 days after acute anterior (seven patients) or inferior (four patients) myocardial infarction. None of the 11 patients had sinus bradycardia (mean heart rate, 90±23 beats/min), but three had a sinus pause immediately before the onset of polymorphous ventricular tachycardia. In all 11 patients, the QT interval and corrected QT interval (QTc) were normal or minimally prolonged (QT, 385±34 msec; QTc, 442±40 msec). None had significant hypokalemia (mean serum potassium concentration, 4.3±0.5 meq/l) or a grossly abnormal serum magnesium or calcium concentration (2.1±0.4 and 8.9±0.7 mg/dl, respectively).

Methods and Results. Immediately before the onset of polymorphous ventricular tachycardia, symptoms and/or electrocardiographic changes consistent with recurrent myocardial ischemia occurred in nine of 11 patients. One patient died before drug therapy could be initiated. Lidocaine was used in 10 patients and proved to be effective in only one. Intravenous procainamide was used in six patients: one improved, and five had recurrence of polymorphous ventricular tachycardia. Bretylium was used in five patients and was ineffective in all cases. Overdrive pacing was used in four patients and failed to suppress recurrent arrhythmias in all cases. Four patients with persistent polymorphous ventricular tachycardia unresponsive to lidocaine, procainamide, or bretylium responded to intravenous amiodarone. One patient with polymorphous ventricular tachycardia that was consistently preceded by ST segment elevation responded to intravenous nitroglycerin. Two patients with persistent polymorphous ventricular tachycardia and obvious recurrent ischemia unresponsive to pharmacological intervention responded to emergency coronary revascularization. A third patient who experienced recurrent angina and polymorphous tachycardia was initially stabilized with pharmacological therapy but subsequently underwent elective revascularization and has remained stable without antiarrhythmic therapy.

Conclusions. Post–myocardial infarction polymorphous ventricular tachycardia is not consistently related to an abnormally long QT interval, sinus bradycardia, preceding sinus pauses, or electrolyte abnormalities. This arrhythmia has a variable response to class I antiarrhythmics but may be suppressed by intravenous amiodarone therapy. It is often associated with signs or symptoms of recurrent myocardial ischemia. Furthermore, coronary revascularization appears to be effective in preventing the recurrence of polymorphous ventricular tachycardia when associated with recurrent postinfarction angina. (Circulation 1991;84:1543–1551)

Polymorphous ventricular tachycardia (PVT) occurring in the course of acute myocardial infarction (MI) appears to be rare and is poorly characterized in the existing literature.1,2 The appropriate management of post-MI PVT is even less well defined. Several investigators1,2 suggest that management of PVT with acute MI is predicated on the duration of the QT interval, suggesting that class Ia antiarrhythmic agents are effective in patients with normal QT intervals, whereas these agents may aggravate the arrhythmia in cases in which repolarization is prolonged. The purpose of this report was to characterize the clinical setting, response to therapy, and prognosis of patients having PVT in the setting of acute MI.

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Methods

Data for this study were collected from patients admitted to the cardiac care units (CCUs) of Moffitt-Long Hospital, University of California Medical Center (UCSF) (eight patients) and the Los Angeles County–University of Southern California Medical Center (USC) (two patients) with acute MI and the development of PVT. Data were collected retrospectively for 1.2 years from UCSF and USC and prospectively for 1.7 years from UCSF. One additional patient (patient 11) was included who developed PVT after a perioperative MI during coronary artery bypass graft surgery (CABG). This patient was not included in calculating the incidence of PVT, however, because postoperative patients were not routinely screened for PVT during the study period. A total of 402 patients with acute MI were screened at UCSF for entry into the study during the 2.9-year recruitment period. Acute MI was defined as having occurred in the appropriate setting associated with classic evolutionary electrocardiographic (ECG) changes and an elevation in serum concentrations of creatine kinase (CK) (more than 128 μm/l) and CK cardiac isoenzyme fraction MB (6% or more of total). An anterior MI was defined when the electrocardiogram demonstrated ST segment elevation of 1 mm or more occurring in leads I and aVL or in at least two adjacent precordial leads. An inferior MI was defined when a similar degree of ST segment elevation occurred in at least two of the inferior leads II, III, and aVF. All such patients were admitted to and continuously monitored in the CCU. PVT was defined as rapid (200 beats/min or more) irregular VT of more than 10 beats in duration with continuous variation in QRS complex. A pattern of torsade de pointes (rotation of the QRS polarity across the baseline) was observed in several patients who were included in the general category of PVT. All patients had at least one episode of PVT. Serum electrolyte levels were reported from the day of onset of PVT. Normal values for our laboratory are as follows: K⁺, 3.5–5.0 meq/l; Ca²⁺, 8.5–10.5 mg/dl; and Mg²⁺, 1.6–2.7 mg/dl. The heart rates and QT intervals were obtained from a 12-lead sinus rhythm electrocardiogram recorded 1–4 hours before the initiation of PVT. The QT interval was measured as described by Lepeschkin. The corrected QT interval (QTc) was calculated according to Bazett’s formula. The QT interval and QTc were defined as prolonged if they exceeded 440 and 460 msec, respectively. Rhythm strips demonstrating the onset of PVT were obtained in all cases. A sinus pause was defined as an increase in the spontaneous sinus cycle length of at least 25% in the beat immediately preceding the onset of PVT. The presence of a premature ventricular depolarization occurring on the upstroke of the T wave at the initiation of PVT was defined as an “R on T” tachycardia onset. ST segment changes were noted if a new ST segment deviation of 1 mm or more occurred immediately before the onset of PVT. Angina was noted if the patient experienced chest pain before initiation of PVT that was similar in character to the pain on hospital admission. Patients receiving class Ia drugs at the onset of PVT and those with primary or secondary ventricular fibrillation were not included in the study. However, one patient (3) was included who had initially received three doses of quinidine but continued having recurrent episodes of PVT more than 72 hours after the quinidine was discontinued. As noted in Table 1, patient 3 experienced PVT that was not preceded by a sinus pause but was preceded by chest pain. Left ventricular function before the development of PVT was assessed in nine of 11 patients by contrast left ventriculography, echocardiography, or radionuclide ventriculography. The left ventricular ejection fraction was calculated by methods described previously. In cases in which left ventricular function was not assessed (patients 1 and 6, Table 1), the patient was either very unstable and died soon after admission (6) or suffered brain death during resuscitation (1). When angiographic data were available, significant coronary artery atherosclerosis was defined as a luminal diameter narrowing of 70% or more.

Antiarrhythmic therapy was initiated with lidocaine in all except one patient. Subsequent therapy was dictated by the attending physician and included the use of intravenous procainamide (10–15 mg/kg loading dose over 1 hour followed by 1.0–2.0 mg/min i.v. infusion) or bretylium (5.0 mg/kg loading dose followed by 1.0–3.0 mg/min i.v. infusion). Four patients underwent overdrive pacing at rates ranging from 95 to 120 beats/min. When conventional antiarrhythmic agents were ineffective, intravenous amiodarone was administered in a loading dose of 5.0 mg/kg infused over 45 minutes. If successful, amiodarone was continued as a continuous infusion (1,200 mg/24 hr) for a total of 3 days, followed by oral amiodarone in standard doses. Antiarrhythmic treatment was judged effective if all episodes of PVT were promptly abolished within 30–60 minutes after initiation of therapy. Treatment was judged ineffective or proarrhythmic when PVT occurred at the same or increased frequency with a given treatment. Revascularization including percutaneous transluminal coronary angioplasty (PTCA) or CABG was used when clinical events (persistent angina, worsening ECG changes) occurred in addition to PVT. Patient outcome is described. In cases in which death resulted, arrhythmic deaths resulting from intractable PVT are separated from deaths occurring from complications arising from cardiac resuscitation.

Statistical Analysis

All values are expressed as mean±SD.

Results

During the 2.9-year recruitment period, 11 patients met the criteria for inclusion into this study. The eight patients with PVT studied at UCSF represent an incidence of 2% of all patients with an acute
### Table 1. Demographic and Clinical Data

<table>
<thead>
<tr>
<th>Patient</th>
<th>Sex</th>
<th>Age (years)</th>
<th>Post-MI day</th>
<th>Myocardial infarction location</th>
<th>Medication at onset of PVT</th>
<th>Heart rate (beats/min)</th>
<th>QT (msec)</th>
<th>QTc (msec)</th>
<th>R on T</th>
<th>STΔs</th>
<th>Angina</th>
<th>LVEF (%)</th>
<th>Angiography data – coronary artery stenosis</th>
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MI, myocardial infarction; K*, serum potassium concentration (meq/l); Ca2+, serum calcium concentration (mg/dl); Mg2+, serum magnesium concentration (mg/dl); Pause, slowing of heart rate on beat immediately preceding onset of polymorphous ventricular tachycardia (PVT); R on T, premature ventricular depolarization occurring on upstroke of T wave at initiation of PVT; STΔs, ST segment deviations of ≥1 mm at onset of PVT; Angina, experienced chest pain immediately before onset of PVT; LVEF, left ventricular ejection fraction by echocardiography, thalligraphy, or radioisotope ventriculography; L, lidocaine; SK, streptokinase; N, nitrates; Nif, nifedipine; Q, quinidine – PVT recurred after quinidine had been discontinued for 72 hours; UK, urokinase; t-PA, tissue-type plasminogen activator; B, B-blocker; ASA, aspirin; CAD, coronary artery disease (% coronary artery stenosis reported as % luminal diameter narrowing); LAD, left anterior descending coronary artery; RCA, right coronary artery; LCx, left circumflex artery; L main, left main coronary artery; –, absent; +, present; $, infarct-related artery.

MI admitted to the CCU at our institution. Demographic and clinical data for each patient are presented in Table 1. None of the 11 patients had significant electrolyte abnormalities that might explain their arrhythmia. Serum potassium concentration averaged 4.3±0.4 meq/l and was 3.4 meq/l or greater in all patients. Only one of the patients in whom serum levels were obtained had mild hypocalcemia (patient 6), and none had hypomagnesemia. The QT interval was normal in 10 patients and minimally prolonged in only one patient (6). Although five patients had a QTc, of more than 460 msec (Table 1), QTc prolongation occurred in association with sinus tachycardia in two patients. None of the patients had sinus bradycardia before the onset of PVT, although a pause preceded PVT in three patients (2, 8, and 9). In five patients, R on T initiating sequences were noted (1, 2, 3, 5, and 11).

PVT occurred without regard to infarct location, degree of left ventricular function, or extent of coronary artery disease (Table 1). Seven patients had anterior MIs, and four had inferior MIs. Of nine patients in whom left ventricular function was defined, left ventricular ejection fraction was normal in
two (2 and 9), mildly depressed in five (3, 5, 8, 10, and 11), and severely depressed in two (4 and 7) patients. Of seven patients in whom angiographic data were obtained, two had one-vessel, three had two-vessel, and two had three-vessel disease (Table 1).

The response to treatment is summarized in Table 2. Intravenous lidocaine was administered to 10 patients and proved effective in only one. Intravenous procainamide was administered to six patients: one improved (5), and five had repeat episodes of PVT. Procainamide appeared to exacerbate PVT in two patients (1 and 6). Intravenous bretylium was administered to five patients but was ineffective in each case. Flecainide and encainide were ineffective in the two patients tested. Intravenous magnesium (4 g) was administered to three patients (6, 7, and 11) and was ineffective in suppressing PVT in all three cases. Overdrive pacing, at rates ranging from 95 to 120 beats/min, failed to suppress PVT in all four patients in whom this was attempted (1, 4, 6, and 11). However, it should be noted that pacing was not used in the three patients with pause-dependent PVT (patients 2, 8, and 9). Intravenous amiodarone was administered in four patients (1, 3, 4, and 7), resulting in suppression of PVT within 30–60 minutes in all cases.

It should be noted that there was no difference in the response to class Ia agents based on the duration of QT interval and QTc, QT interval and QTc were similar in the patient who responded (n=1) compared with those who failed to respond to class Ia antiarrhythmics (n=5) (380 versus 382±49 msec, respectively, and 400 versus 434±44 msec, respectively). Furthermore, the patient with the shortest QT and QTc (patient 4) (QT, 320 msec; QTc, 400 msec) failed to respond to a class Ia agent.

Although PVT occurred without regard to the extent of coronary artery disease, each patient in whom angiographic data were obtained had a high degree of residual stenosis (90% or greater vessel diameter narrowing) in the infarct-related artery (Table 1). Furthermore, immediately before the initiation of PVT, symptoms of angina and/or ST segment deviation on the electrocardiogram (Figures 1–3) that were suggestive of recurrent MI were noted in nine of 11 patients (Table 1). It should be noted that four patients had received thrombolytic therapy (2, 5, 9, and 10), but subsequent angiography revealed high-grade residual stenosis or total occlusion of the infarct-related artery in each case. Intravenous nitroglycerin was given to six patients (2, 5, 6, 9, 10, and 11) as antianginal therapy but was effective in preventing recurrence of PVT in only one case (11). However, three patients (5, 9, and 10) who had recurrent angina and PVT despite intravenous nitroglycerin had resolution of both recurrent PVT and ischemia after coronary revascularization. In two of these patients (9 and 10), emergency revascularization appeared to acutely stabilize the patient, preventing the recurrence of both postinfarction angina as well as postinfarction PVT (Figures 3 and 4).

PVT was ultimately controlled in nine patients (Table 2). Current follow-up for patients surviving hospitalization is given in Table 2. It should be noted that of the four patients who initially responded to intravenous amiodarone, two subsequently died of sequelae from prolonged hypotension sustained during the resuscitative effort, one was continued on oral amiodarone, and one underwent coronary revascularization and was discharged without the need for further antiarrhythmic therapy. Of the three patients who underwent coronary revascularization, all three were discharged from the hospital without the need for antiarrhythmic agents. Four patients died during hospitalization. Of these deaths, one (6) resulted from intractable PVT, and one (2) occurred soon after the onset of PVT, before therapy could be initiated. Two patients (1 and 7) were initially stu-
lized with intravenous amiodarone but later died from central nervous system damage that was incurred during resuscitative efforts. Each of these patients had been treated with two antiarrhythmic agents before the administration of amiodarone, potentially delaying what ultimately proved to be effective therapy.

**Discussion**

The present study reports three important findings concerning PVT after acute MI. First, although the majority of cases of post-MI PVT occurred in patients with a normal or mildly prolonged QT interval and QTc, the arrhythmia generally failed to respond to class I antiarrhythmics. Second, intravenous amiodarone appeared to be effective in suppressing post-MI PVT in the four patients who received this agent. Third, in the majority of patients, post-MI PVT was accompanied by symptoms or ECG evidence of recurrent ischemia. Although intravenous nitroglycerin was effective in suppressing recurrent episodes of PVT in only one of six patients who received this agent, coronary revascularization appeared to be effective in three of these patients.

The clinical features and therapy of PVT occurring with acute MI have not been well delineated. Zilcher and coinvestigators reported nine patients who developed PVT after acute MI, noting that the QT interval was normal in six of nine cases. However, the individual ECG characteristics of these patients and the relation between the QT interval and response to therapy were not reported. Morady et al. reported four patients who suffered PVT after acute MI. However, they did not report the ECG characteristics of these patients. Soffer et al. reported one patient who had PVT with acute MI. This patient was noted to have a normal QT interval and responded to procainamide therapy. On the basis of this case, the authors suggest that PVT occurring in the absence of QT prolongation can often be treated with success using conventional antiarrhythmics, including class I agents. In contrast, Nguyen and coworkers reported three patients who developed PVT after acute MI, noting that all three had normal QT intervals and that all three failed conventional class Ia antiarrhythmics.

In our series of 11 patients, PVT after acute MI generally failed to respond to class I antiarrhythmics regardless of QT interval duration. Our data showed that lidocaine is rarely effective in suppressing PVT with acute MI. This observation is similar to data collected for PVT occurring with MI without infarction. Furthermore, we found that class Ia agents were generally ineffective or proarrhythmic in patients with post-MI PVT, despite the fact that our patients had normal or minimally prolonged QT intervals. These data were consistent with observations of Nguyen et al. and in contrast to suggestions by Soffer et al.
We found that intravenous magnesium (three patients) and overdrive pacing (four patients) were not effective in suppressing recurrence of PVT (Table 2). This is in contrast to the reported success of these interventions in suppressing PVT associated with a prolonged QT interval.13,14 As noted earlier, pacing was not used in patients who exhibited pause-dependent PVT.

In our experience, intravenous amiodarone was useful in patients with PVT induced by acute MI. In the present study, four patients who failed therapy with conventional antiarrhythmics, including lidocaine, procainamide, and bretylium, had effective suppression of PVT with intravenous amiodarone. The onset of action was rapid, and no toxic effects were observed. Previous reports have confirmed the efficacy of intravenous amiodarone for refractory ventricular tachyarrhythmias, including VT, after acute MI.9,10,15 Although amiodarone may exert proarrhythmic effects in the form of PVT, the incidence of this is extremely low.10,16 The safety and efficacy of oral amiodarone with drug-mediated torsade de pointes have been well documented17,18 despite amiodarone-induced QT prolongation. The infrequent nature of PVT induced by amiodarone relative to class I antiarrhythmic agents has been attributed to the concomitant blockade of Na+ and Ca2+ channels.19

Other investigations have reported PVT associated with other acute and chronic ischemic syndromes, including variant and unstable angina,11,12 chronic stable angina,20 and silent exercise-induced ischemia.21 Sawaya and Rubeiz11 and Puddu et al12 reported two cases of variant or unstable angina associated with PVT. Tchou et al20 described 16 patients who developed PVT in association with severe coronary artery disease, 14 of whom had suffered prior MIs. One of these patients with severe coronary stenosis developed exercise-induced PVT. Silent MI with exercise-induced PVT was reported in four patients by Pedersen et al.21 They identified six other patients with similar exercise-induced PVT in the literature.

As noted in Table 1, the majority of our patients had symptoms of ECG changes suggestive of MI immediately before the onset of PVT. The fact that intravenous nitroglycerin failed to suppress recurrence of the arrhythmia in five of six patients who received this agent suggests that coronary spasm was not the etiology of ischemia in these patients; two patients (9 and 10) had evidence of persistent thrombus in the infarct-related artery on coronary angiography. However, it should be noted that in one patient (11), treatment with intravenous nitroglycerin resulted in resolution of both ST segment elevation and PVT, suggesting that coronary spasm may have triggered PVT in that patient.

Revascularization for patients experiencing PVT secondary to acute MI was potentially curative. In the present report, two patients (9 and 10) had resolution of PVT after acute revascularization. Both of these patients had received thrombolytic therapy, but each underwent emergency cardiac catheterization when refractory PVT coincided with symptoms and ECG evidence of recurrent ischemia (Figure 3). Coronary angiography revealed either total or subtotal occlusion of the infarct-related artery in each case (Figure 4), and revascularization resulted in resolution of both recurrent ischemia and PVT. Thus, in
the context of thrombolytic therapy for acute MI, PVT may represent a "reocclusion arrhythmia" signifying the need for more aggressive attempts at revascularization.

Although there are no other reports concerning the efficacy of revascularization in controlling PVT after acute MI, others have reported on the efficacy of CABG in PVT associated with other acute ischemic syndromes.11,20,21 Pedersen et al21 presented data for four patients with PVT and silent MI, three of whom underwent CABG after failure to control the arrhythmia with Ca²⁺ antagonists and/or β-blocking agents. In two patients, surgery resulted in 1- and 2-year event-free survival periods. The third surgical patient died suddenly 10 days after surgery. They reviewed further the literature on PVT associated with silent ischemia and its response to revascularization. In five reviewed studies, a total of six patients with coronary artery disease and exercise-induced PVT had cessation of PVT after revascularization. Sawaya and Rubeiz11 reported two cases of PVT associated with high-grade coronary artery disease and unstable angina. After β-blockers and class I antiarrhythmics failed to suppress recurrent PVT, both patients underwent CABG with impressive 4- and 6-year event-free survival intervals after surgery. Finally, Tchou et al20 recently described 16 patients with severe coronary artery disease who developed PVT. One patient, who had severe narrowing of the right coronary artery, developed PVT during exercise that was abolished after successful angioplasty of that vessel.

The mechanism of PVT is thought to be due to early afterdepolarizations and triggered activity. Early afterdepolarizations may be detected by monophasic action potential recordings and have been documented to occur during reperfusion.22 PVT occurring in the presence of a normal QT interval may be explained by focal afterdepolarizations. It is believed that a visible effect on the QT interval may not develop unless a suitably large segment of myocardium is involved in the generation of afterdepolarizations.23,24

The major limitation of the present study is that not all patients were treated in a uniform fashion. For example, despite the fact that 10 of 11 patients had a normal QT interval on an electrocardiogram before the onset of PVT, only six received a class Ia agent. However, as noted above, procaainamide was not particularly useful in suppressing PVT and appeared to exacerbate the arrhythmia in two patients. Finally, intravenous amiodarone and coronary revascularization, the two forms of therapy that appeared most useful in suppressing PVT, were administered to only four and three patients, respectively, with no
one patient receiving each intervention. However, it should be noted that both of these interventions suppressed subsequent PVT in all cases. Clearly, however, further research is necessary using standardized protocols to evaluate the relative effectiveness of each of these interventions in suppressing post-MI PVT.

In summary, PVT rarely occurs in the setting of acute MI and in large part appears to be unrelated to electrolyte abnormalities, prolonged QT interval, or preceding pauses. We found that this arrhythmia usually occurs in conjunction with recurrent ischemia and was associated with a poor prognosis, especially when delay in appropriate therapy resulted in death or brain death due to prolonged resuscitation. Our preliminary findings suggest that acute stabilization may be achieved by intravenous amiodarone and/or emergency coronary arteriography and prompt revascularization.

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References
recurrent symptomatic ventricular tachycardia. *Am J Cardiol* 1983;51:156–159


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