Self-Terminating Polymorphous Ventricular Tachycardia Occurring at the Peak of Myocardial Ischemia

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Polymorphous ventricular tachycardia (PVT) is a unique arrhythmia that may occur during or shortly after acute myocardial ischemia. It is believed that the occurrence of PVT at the time of ischemia is due to differences in the shortening time of the myocardial potentials in the different layers of the myocardium, caused by the heterogenic blood supply at that time. We describe a case of a patient who developed two consecutive episodes of PVT, both induced by ventricular premature beats (VPBs) that occurred during the peak of myocardial ischemia as detected by the ST analyzing system while hospitalized in the intensive coronary care unit.

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A 52-year-old male was admitted to the intensive coronary care unit due to an inferior wall myocardial infarction. His potassium, calcium, and magnesium levels were all within normal limits and the corrected QT interval on his admission electrocardiogram, as calculated using the Bazett formula, was 410 milliseconds. Eight hours after the admission, the ST segment analyzer demonstrated recurrent ST elevation (Fig. 1), followed by two consecutive events of polymorphous ventricular tachycardia (PVT, Figs. 2a and 2b). Both events were initiated by ventricular premature beats with extremely short coupling interval (the "R on T" phenomenon). The cycle length during both the events was around 250 milliseconds, and while the first episode terminated spontaneously, the second episode deteriorated to ventricular fibrillation and was terminated by a direct shock. The patient was transferred to the catheterization lab, where his occluded right coronary artery was opened using a drug eluting stent. The rest of the hospitalization was uneventful and the patient was discharged a few days later without changes in his drug regimen.

DISCUSSION

Ischemic PVT is believed to be initiated by reentrant arrhythmias during the very early phases of repolarization, presumably due to the uneven shortening of the myocardial potentials that occur at the different layers of the myocardium at the time of ischemia.¹ Due to this mechanism, ischemic PVT is typically initiated by extrasystoles with very short coupling intervals and with an associated normal or minimally prolonged QT corrected (QTc) interval, as in the case of the patient described above.² Pathophysiologically and electrocardiographically, ischemic PVT differs from another, distinct, ischemia-associated arrhythmia termed infarct-related torsades de pointes. This latter arrhythmia typically takes place several days after the infarct, while inversion of the T waves and prolongation of the QTc interval occur. Halkin et al. described postinfarction torsades de pointes to be pause dependant, with the characteristic "short-long-short" cycle length sequence preceding it.³ In contrast, in nearly all prior descriptions of patients with ischemic PVT the reported QTc

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Figure 1. At 6:06 a.m., the ST analyzer showed elevation of the ST segment in leads II, III, and AVF, culminating about 8.5 minutes later.

Figure 2. At 6:15 a.m., a few ventricular premature beats with very short coupling intervals occurred, without prolongation of the QTc interval. Two of them initiated a polymorphic VT. The first episode (a, top) terminated spontaneously while the second one (b, bottom) deteriorated to VF.

interval was not prolonged,^{2,4,5} and treatment attempts with magnesium, overdrive pacing, and class I antiarrhythmics all failed in suppressing the tachycardia.^{2,4,5}

Notably, in nearly all the previously described cases of ischemic PVT (ours inclusive), revascularization was found to be highly effective in both

terminating and preventing the recurrence of the arrhythmia.

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