Pilsicainide-Induced Polymorphic Ventricular Tachycardia

Yoshiaki Kaneko, Tadashi Nakajima, Toshimitsu Kato and Masahiko Kurabayashi

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An 82-year-old woman with a previous history of inferior myocardial infarction was admitted to our hospital with the complaint of syncope. She had received hemodialysis for chronic renal failure and an oral administration of pilsicainide (50 mg daily) for prevention of paroxysmal atrial fibrillation. The ECG showed repetitive polymorphic ventricular tachycardia (VT) initiating from the same premature ventricular contractions with a constant, short-coupling interval, probably originating from the inferior wall, resembling Purkinje-related polymorphic VT (1, 2). (Picture 1, 2). The width of QRS complexes of 160 msec (Picture 3B) was prolonged compared with that of 110 msec before the administration of pilsicainide (Picture 3A). The serum level of pilsicainide of 1.89 μg/mL was elevated above the upper limit of the therapeutic range between 0.2 and 0.9 μg/mL. The day after the discontinuation of pilsicainide, the VT disappeared. Scar- or Purkinje-related reentry (3) provoked by slowing of myocardial conductivity due to an excess of pilsicainide may be responsible for the proarrhythmia as the mechanism.

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References