QRS Alteration-induced Torsade de Pointes in a Patient With an Artificial Pacemaker and Hypokalemia

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The present patient demonstrated torsade de pointes (TdP) after a pacemaker implantation without a decrease in heart rate or pacemaker malfunction. Marked QT prolongation and TdP were closely related to alteration of the depolarization pattern (appearance of junctional rhythm). Resetting the pacemaker to a rate that inhibited spontaneous rhythm was effective in preventing TdP.
(*Jpn Circ J* 1996; **60**: 189–191)

SYNCOPE in a patient with an implanted artificial pacemaker is usually caused by ventricular standstill or torsade de pointes (TdP) associated with pacemaker malfunction! In this paper, we report a patient who demonstrated TdP without malfunction of a pacemaker which had been implanted 10 years previously. Detailed analysis of the initiating pattern of TdP in this patient revealed that the arrhythmia was closely related to alteration of the depolarizing pattern.

CASE REPORT

The 64-year-old female underwent an artificial pacemaker implantation (VVI mode; rate, 70 beats/min) on 2 September 1980 because of sinus bradycardia and symptomatic cardiac arrest after supraventricular tachycardia. Neither TdP nor QT prolongation was noted. Seven years later, the pacer-

maker was altered to DDI mode (lower rate, 70 beats/min) because of dyspnea on effort and cardiomegaly. In June 1990, when the patient was 74 years old, she complained of frequent syncopal episodes associated with exercise. Episodes of TdP were observed during Holter monitoring. TdP was initiated during junctional rhythm and was associated with marked QT prolongation and a bizarre T-wave pattern (Fig 1). Hypokalemia (K, 3.0 mmol/L) was noted, while other serum electrolyte levels were within normal ranges. She received no cardioactive drugs before or during these episodes. After her admission to the hospital, the pacemaker rate was decreased to 45 beats/min (VVI mode) to determine whether the junctional rhythm was responsible for the initiation of TdP. The junctional beats appeared spontaneously without physical or mental stress. Despite a narrow QRS and short RR interval, the junctional beats were associated with marked QT prolongation and T-wave changes (Fig 2A). Hand-grip exercise induced more frequent junctional beats with subsequent ventricular premature beats and TdP (Fig 2B). One month later, when the

Key words:

Torsade de pointes
Prolonged QT interval
Artificial pacemaker
Hypokalemia
QRS change

(Received April 25, 1995; accepted July 18, 1995)

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Fig 1. Junctional rhythm appeared with slight physical exercise. The QT intervals during junctional rhythm (75–80 beats/min) are longer than those during ventricular pacing (DDI mode, 70 beats/min), despite the narrower QRS and shorter RR interval (upper panel). Although the pacemaker is functioning normally, typical torsade de pointes develops following junctional rhythm (lower panel).

Fig 2. Marked QT interval prolongation (bizarre T wave) and torsade de pointes after junctional rhythm. (A) When the pacing rate was decreased to 45 beats/min (VVI mode), junctional rhythm (arrows) appeared without any mental or physical stress. The marked QT prolongation and bizarre T-wave developed with the junctional rhythm. The QT interval was 560 ms during ventricular pacing and 680 ms during junctional rhythm. The RR interval preceding the junctional beat was shorter than that during pacemaker rhythm. (B) Premature ventricular beats (PVBs) and torsade de pointes were induced by hand-grip exercise. All ventricular arrhythmias were associated with junctional rhythm (arrows). The occurrence of PVBs and torsade de pointes was not dependent on further RR prolongation, but rather on spontaneous junctional beats.

serum potassium level was normal, the pacing rate was again decreased to 45 beats/min. Although the QT interval was longer during junctional rhythm than during ventricular pacing, prolongation was not as prominent

and no ventricular arrhythmia was induced.

Therefore, we programmed the pacemaker mode to rate-responsive VVI and the lower pacing rate to 80 beats/min to inhibit the escape rhythm (neither DDD nor DDI

mode could be selected because atrial pacing and sensing were not possible). She has had no syncopal episodes for 28 months since this pacemaker programming.

DISCUSSION

A TdP-induced syncopal episode in a patient with a pacemaker implantation strongly suggests pacemaker malfunction¹ since TdP in acquired long QT syndrome is usually dependent on bradycardia². However, in the present patient, TdP developed without any slowing of the heart rate and without pacemaker malfunction. An alternate pattern of depolarization which was related to the junctional rhythm induced marked QT prolongation and TdP.

Long-lasting ventricular pacing induces T-wave polarity changes after a normal supraventricular activation pattern. If the altered ventricular activation sequence is long enough to produce a ventricular gradient (the inverse correlation between activation time and action potential duration), then the T-wave following activation in the opposite direction becomes concordant with the previous paced R wave (cardiac "memory")^{3,4}. Under these conditions, dispersion of repolarization may be increased since ventricular sites with shorter action potential durations are activated faster and those with longer durations are activated later. The heterogenous repolarization pattern induced by alteration of QRS might be related to QT prolongation and TdP. Therefore, we consider that maintaining a constant depolarizing pattern by increasing the pacemaker rate is an effective method of decreasing the dispersion of

repolarization and hence of preventing the recurrence of TdP.

In the present patient, hypokalemia may have been one of the predisposing factors for TdP, since QT prolongation was less marked and TdP was not induced during junctional rhythm in the normo-kalemic state.

The exercise-related TdP in this patient suggests the arrhythmogenic role of sympathetic activity, which is, however, not always effective because the junctional rhythm in the absence of mental or physical stress demonstrated an equally prolonged QT interval (Fig 2).

Our analysis of the initiating pattern of TdP in this patient provides important information for the management of pacemaker patients. When the pacemaker rate is set slightly above the patient's own heart rate, occasional spontaneous rhythm may induce TdP. Therefore, resetting the pacemaker to a rate that inhibits spontaneous rhythm is an effective therapy in preventing TdP.

REFERENCES

1. Mond HG: Testing methods for suspected malfunction. In: Mond HG, editor. *The Cardiac Pacemaker, Function and Malfunction*. New York, Grune & Stratton, 1983; 267–286
2. Stratmann HG, Kennedy HL: Torsades de pointes associated with drugs and toxins: recognition and management. *Am Heart J* 1987; **113**: 1470–1481
3. Rosenbaum MB, Blanco HH, Elizari MV, Lazzari JO, Davidenko JM: Electrotonic modulation of the T wave and cardiac memory. *Am J Cardiol* 1982; **50**: 213–222
4. Costard-Jäckle A, Goetsch B, Antz M, Franz MR: Slow and long-lasting modulation of myocardial repolarization produced by ectopic activation in isolated rabbit hearts. *Circulation* 1989; **80**: 1412–1420