A 77-year-old woman was emergently admitted for the evaluation of ventricular tachycardia (VT) which developed following an intravenous administration of procainamide (400 mg) for atrial fibrillation, complicated with loss of consciousness, and successfully treated by automated external defibrillator (AED). The surface ECG stored in the AED showed apparent monomorphic VT terminated by DC shock (Picture 1), followed by spontaneous occurrence of polymorphic VT (Picture 2). Detailed inspection of the ECG during the monomorphic VT clarified a subtle variation of QRS morphology and RR interval (Picture 1). On admission, the 12-lead ECG revealed prominent QT prolongation and premature ventricular contraction following augmentation of U wave triggered by ventricular pause (Picture 3A), consistent with bradycardia-dependent repolarization abnormality. Five days after hospitalization, the QT interval was normalized (Picture 3B). Coronary angiography demonstrated no significant stenosis. Left ventriculography showed depressed global systolic function with an ejection fraction of 49.5%. During an electrophysiologic study, neither VT nor ventricular fibrillation was induced. These findings suggest an arrhythmic diagnosis of torsades de pointes (TdP) complicated by the administration of procainamide.

The QRS morphology of VT is generally suggestive of
the etiology, the mechanism and the therapeutic strategy of the VT. Although the QRS morphology of TdP is commonly polymorphic, this sample of TdP apparently mimicking monomorphic VT is an enlightening discovery, which we should consider on the differential diagnosis of monomorphic VT (1).

Reference