Amiodarone is a potent antiarrhythmic drug effective in refractory ventricular tachyarrhythmias, and it is reported that amiodarone can be administered without dose adjustment even in patients with renal dysfunction. Although electrolyte disorders are frequently observed in patients with end-stage renal disease, the interaction between electrolyte disorders and the antiarrhythmic effects of amiodarone has not been established. Here, we describe a patient with myocardial infarction and end-stage renal disease, in whom ventricular tachycardia (VT), suppressed by amiodarone, recurred only during hyperkalemia.

Case Report

The patient, a 58-year-old man undergoing continuous ambulatory peritoneal dialysis for 5 years, was admitted to the hospital because of acute myocardial infarction and cardiogenic shock. Percutaneous transluminal coronary angioplasty and the deployment of an intracoronary stent resulted in successful recanalization of the occluded left anterior descending artery, and the patient’s hemodynamic status improved. On day 3 after admission to hospital, the patient developed sustained monomorphic VT (Fig 1) and electrical cardioversion was frequently required. After repeated termination of VT by ventricular pacing and unsuccessful conventional antiarrhythmic drugs, amiodarone was administered at a loading dose of 600 mg/day for a week followed by a maintenance dose of 200 mg/day. VT subsided gradually over the following days; subsequently, only a few isolated ventricular ectopic beats were recorded in the electrocardiographic monitoring.

This patient had previously suffered recurrent episodes of peritonitis associated with peritoneal dialysis. Unexpectedly, he had developed peritonitis on this hospitalization. Because this episode of peritonitis, caused by Pseudomonas aeruginosa, was resistant to vigorous antimicrobial therapy, the peritoneal catheter was removed on day 12 and hemodialysis was instituted. Two days later another VT occurred, resulting in hemodynamic deterioration. This VT showed identical morphology to the preceding VT, but the heart rate was slower (Fig 2). Intra-aortic balloon counterpulsation was required to achieve hemodynamic stabilization. Antiarrhythmic drugs, including procainamide, propranolol, lidocaine and magnesium, were ineffective in suppressing this VT. As in the first attack, ventricular pacing reproducibly entrained and finally terminated this VT. Laboratory tests revealed an increased serum potassium concentration of 7.1 mmol/L. Because no other remediable abnormalities were found, the patient underwent hemodialysis immediately for the treatment of hyperkalemia. VT subsided gradually with alleviation of hyperkalemia during hemodialysis, and did not recur after correction of hyperkalemia. Several months later, the patient died suddenly because poor dietary compliance resulted in an increase in his potassium concentration. This case suggests that hyperkalemia may reverse the potent antiarrhythmic effects of amiodarone. (Jpn Circ J 1999; 63: 323–325)

Key Words: Amiodarone; Myocardial infarction; Renal failure; Ventricular tachycardia
Amiodarone has some unique properties: (i) it has been proven to be effective in the treatment of life-threatening cardiac arrhythmias;1 (ii) there is no need for dosage adjustment in patients with renal dysfunction (in contrast to many conventional antiarrhythmic drugs);2,3 (iii) it has weak negative inotropic effects;5 and (iv) proarrhythmic effects are rare compared with other antiarrhythmic drugs. These characteristics prompted us to administer amio-

**Discussion**

Amiodarone has some unique properties: (i) it has been proven to be effective in the treatment of life-threatening cardiac arrhythmias;1 (ii) there is no need for dosage adjustment in patients with renal dysfunction (in contrast to many conventional antiarrhythmic drugs);2,3 (iii) it has weak negative inotropic effects;5 and (iv) proarrhythmic effects are rare compared with other antiarrhythmic drugs. These characteristics prompted us to administer amio-
Amiodarone and Hyperkalemia

It has been reported that hypokalemia due to concomitant diuretic therapy in a patient treated with amiodarone induced torsades de pointes. Hyperkalemia has not previously been associated with exacerbation or recurrence of tachyarrhythmias in patients treated with this agent. An experimental study showed that an increase in extracellular potassium concentration attenuated the efficacy of amiodarone, with the result that a higher effective concentration was required.

Based on the clinical course, on the absence of other contributory factors to VT recurrence and on the experimental report, we believe that hyperkalemia probably reversed the potent antiarrhythmic effects of amiodarone, which were confirmed in the electrophysiologic study.

In summary, this case suggests an association between hyperkalemia and VT recurrence and sudden death during effective amiodarone therapy. Further investigations are required to determine whether and how electrolyte disorders affect the actions of antiarrhythmic drugs.

Fig 3. Electrophysiologic study. This electrocardiogram shows that triple extrastimuli delivered at the right ventricular apex induced, at most, non-sustained polymorphic ventricular tachycardia.

**References**