Torsade de pointes and Sudden Death in a Patient with Amyloidosis-Associated Nephropathy

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Dear Sir

A variety of cardiac arrhythmias can be induced by hypokalemia [1-3], and the relationship between hypokalemia and ventricular arrhythmias may account for the possible increase in sudden cardiac death observed in patients with preexisting cardiac disease (left ventricular hypertrophy and acute myocardial infarction) who are treated with diuretics [4]. We here report a fatal torsade de pointes which suddenly developed in a patient with amyloidosis-associated nephropathy.

A 45-year-old male was transferred to Kangnam St. Mary's Hospital due to edema on the whole body and dyspnea which had developed 1 month ago. He had been diagnosed as having amyloidosis-associated nephrotic syndrome by renal biopsy 2 years ago and took diuretics. On admission, physical examination revealed a decreased breathing sound, a distended abdomen and 3+ pitting edema of the extremities. Laboratory findings were as follows: sodium 142 mEq/l, potassium 4.1 mEq/l, magnesium 2.4 mg/100 ml, BUN 3.6 mmol/l (12.8 mg/dl), creatinine 97.3 μ mol/l (1.1 mg/dl), total protein 5.4 g/100 ml, albumin 3.2 g/100 ml, total cholesterol 188 mg/100 ml, triglyceride 64 mg/100 ml. Urine revealed 3+ protein. Chest PA film revealed cardiomegaly and pleural effusion, and echocardiography showed hypertrophy of the left ventricle and interventricular septum with poor wall motion, suggesting amyloidosis-associated infiltrative cardio-myopathy (fig. 1). The patient was treated with intravenous furosemide with successful control of edema. On the 5th hospital day, he suddenly fainted and torsade de pointes was registered on electrocardiographic monitoring (fig. 2). Laboratory findings revealed hy-

Fig. 1. The echocardiography shows a markedly thickened left ventricular wall and interventricular septum and decreased wall motion.

Fig. 2. The electrocardiogram shows the long Q-T interval and torsade de pointes.

hypokalemia (3.3 mEq/l) and hypomagnesemia (1.8 mg/100 ml). In spite of intravenous infusion of potassium, lidocaine, amiodarone and magnesium, ventricular tachycardia persisted and he expired on the 6th hospital day due to intractable ventricular fibrillation. In conclusion, a slight progression of hypokalemia and hypomagnesemia can cause fatal torsades de pointes in selected cases such as amyloidosis-associated cardiomyopathy. Therefore, careful monitoring of serum potassium, magnesium and echocardiography must be considered during diuretic therapy.

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