TREATMENT OF TORSADE DE POINTES WITH INTRAVENOUS MAGNESIUM IN IDIOPATHIC LONG QT SYNDROME

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A middle aged woman with idiopathic long QT syndrome was found to have repetitive ventricular tachycardia of the "torsade de pointes" type. The arrhythmia was resistant to mexiletine and lidocaine, but was controlled by intravenous magnesium sulfate (MgSO_4). The recurrent attacks were abolished by a bolus of 2.0 g MgSO_4 , and extremely prolonged QTU interval was reduced by intravenous infusion of 5 mg/min MgSO_4 for 36h. This case shows the effectiveness of intravenous magnesium in controlling the attack of torsade de pointes in patients with idiopathic long QT syndrome.

TORSADE de pointes (TdP) is a special form of polymorphic ventricular tachycardia most frequently induced by QT-prolonging drugs^{1,2} and also observed in idiopathic long QT syndrome³. The recommended therapy for this unique arrhythmia in patients with acquired long QT syndrome is isoproterenol or cardiac pacing²⁻⁴ and that for idiopathic long QT syndrome is beta-blockers^{5,6}. Recently intravenous administration of magnesium sulfate has been proposed as a simple and effective method of treating TdP induced by quinidine-like drugs^{7,8}. We report a case of TdP with idiopathic long QT syndrome treated by magnesium sulfate.

CASE REPORT

A 44-year-old woman with active lung tuberculosis had been transported to our

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department in Tottori University Hospital on November 10, 1988 for treatment of a cardiovascular disorder after resuscitation from cardiac arrest preceded by ventricular fibrillation found in a sanatorium. An electrocardiogram on admission showed a sinus bradycardia (HR 43 beats/min) with a prolonged and prominent negative T wave ($\text{QTc}=0.78$). She had had a permanent pacemaker (VVI mode) implanted due to severe sinus bradycardia with recurrent sinus arrests (3-4s). Average heart rate was 20-30 beats/min. Her past and family histories are not clear because she had no live relatives. The electrocardiogram showed QT prolongation not only in the pacemaker rhythm at a rate of 60/min but also in the sinus and ectopic rhythm at rates of 60-70/min without any other remarkable abnormalities. She had been diagnosed with idiopathic (or congenital) long QT syndrome because of the absence of other causes of QT prolongation at that time³. She was not considered to be able to tolerate the propranolol

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Fig.1. A: Initial ECG shows pacemaker rhythm with long QTU interval. The small deflection on each T wave suggests a P wave induced by retrograde ventriculo-a trial conduction. B: Previous (a, December, 1988) and admission (b) ECGs and serial ECGs of lead II during magnesium infusion (c-e) show the decrease in amplitude of U waves and block of retrograde conduction. (c: April 28, 1h after magnesium injection, d: April 29, during magnesium infusion and 10h after bolus injection, e: May 1). C: Initiation and termination of torsade de pointes observed in 24h ambulatory recording (lead: NASA).

therapy due to its hypotensive side effect, suppression of sinus node automaticity (additional decrease of about 20 mmHg in blood pressure was observed in pacing rhythm compared to the sinus rhythm), and possibility of worsening of respiratory function

already adversely affected by recurrence of lung tuberculosis, and was given oral mexiletine 50 mg, three times daily from November 30, 1988. The administration of mexiletine did not change the QT interval (Fig. 1, Ba) and abolished the premature ventricular con-

tractions which were confirmed by Holter monitoring (no premature ventricular contractions and 66344 electrically paced beats in 87736 of the total beats during 24h monitoring).

The patient was admitted to the Tottori University Hospital again on April 28, 1989 because of recurrent syncopal attacks during a visit to the University Hospital to detach the Holter monitoring unit as a regular follow up examination. Her blood pressure was 90/50 mmHg, and her pulse rate was 60/min and regular. No heart murmurs were audible. A chest X-ray film showed active tuberculous lung lesions. Her leukocyte count was 14600/mm³. The serum potassium and magnesium levels were 4.5, and 2.3 mEq/l, respectively. Other routine laboratory studies were all within normal limits. An electrocardiogram on admission (Fig. 1, A and Bb) revealed a ventricular pacing rhythm with a prominent late diastolic U wave resulting in an extremely prolonged QTU interval and downward deflection which was supposed to be a negative P wave due to retrograde conduction (Fig. 1, A). A few hours after admission, episodes of TdP appeared (Fig. 1, C). Most of the episodes subsided spontaneously, but some caused Adams-Stokes attacks. Lidocaine, 100 mg, was given intravenously, but was ineffective in controlling TdP. Therefore 2.0g of magnesium sulfate (MgSO₄) 25% was injected intravenously over 5 min. Then the TdP disappeared completely. Thereafter, 5 mg/min of MgSO₄ was given intravenously for 36h and the pacing rate was increased from 60 to 75/min to support the magnesium effect on the reduction of QT interval. No further episodes of TdP were observed. During magnesium infusion over 36h, QT_c interval decreased from 0.75 to 0.65s with a reduction of U wave amplitude (Fig. 1, B, c-e) and retrograde ventriculo-atrial conduction was incompletely suppressed. After cessation of magnesium infusion, the patient was treated with propranolol (10 mg, three times a day). QT_c interval continued to decrease to 0.54s (May 1) and 0.50s (May 20), and retrograde ventriculo-atrial conduction showed no further change. The patient was discharged from the University Hospital with medication of propranolol, 20 mg three times a day for the prevention of recurrence

of TdP without any attacks of asthma. She was considered to be able to tolerate the additional decrease in blood pressure induced by propranolol.

DISCUSSION

TdP is a serious ventricular tachyarrhythmia usually induced by QT-prolonging drugs such as quinidine and disopyramide^{1,2}. The long QT syndrome is another cause of this type of arrhythmia³. Its characteristic electrocardiographic features are marked QT prolongation, short runs of VT, or even ventricular flutter with a twisting QRS axis and polymorphic appearance. Lidocaine is sometimes ineffective and temporary pacing is advocated as a safe and effective treatment for TdP. In recent years the use of MgSO₄ has been reported as an effective therapy for TdP induced by QT prolonging drugs and hypokalemia^{7,8}.

Although our patient had a permanent pacemaker (VVI mode) implanted, the electrocardiogram showed QT prolongation not only in the pacemaker rhythm at a rate of 60/min but also in the sinus and ectopic rhythm at rates of 60–70/min detected in few days during her first admission. She had been diagnosed with idiopathic or congenital long QT syndrome because of the absence of other causes of QT prolongation at that time³. Propranolol (10 mg 3 times a day) was prescribed to prevent premature ventricular contractions and TdP attack, but due to the severe hypotensive side effect and reasons described above we considered it intolerable. At that time, she had recovered from the catecholamine-dependent state to maintain her blood pressure after resuscitation. Her cardiac function recovered and her circulation could be maintained 6 months after cardiac arrest, because the reduction in blood pressure induced by a larger dose of propranolol (20 mg, 3 times a day) was tolerated better than in the first trial.

The cause of TdP attack at this time is not clear. She was taking anti-tuberculosis drugs (isoniazid and rifampicin), which have not been reported to cause QT prolongation, and mexiletine which has been reported to induce TdP in rare cases⁹. However, the administration of mexiletine did not change the already prolonged QT interval during

her first admission. This is in contrast to other class Ia drugs which usually prolong the QT interval and sometimes cause TdP. The influence of the inflammation of active lung tuberculosis to sympathetic nervous tone can not be excluded as a cause of TdP.

A previous electrocardiogram (December, 1988, Fig. 1, Ba) showed a mildly prolonged QT interval with a small positive U wave and negative P wave which was suggested the presence of retrograde ventriculo-atrial conduction. Before (Fig. 1, Bb) and during magnesium infusion (Bc-e) suppression of the prominent U wave was observed. An enhanced U wave seems to be a marker of susceptibility to TdP^{10,11}. The suppression of after-depolarization of the cardiac cell membrane⁵ may be responsible for the effectiveness of intravenous MgSO₄ in patients with TdP. A prolongation of the interval of Q and negative P waves and the absence of a negative P wave after some QRS complexes (Fig. 1, Bc-e) may be ascribed to the calcium-antagonistic effect of magnesium ions¹² in the retrograde ventriculo-atrial conduction.

Banal et al¹³ recommended intravenous magnesium not only in cases of drug-induced TdP, but also in congenital long QT syndrome and TdP. It seems reasonable to use magnesium as a simple and effective treatment to abolish TdP in congenital (or idiopathic) long QT syndrome.

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