Efficacy of Magnesium Sulfate for Treatment of Ventricular Tachycardia in Amitriptyline Intoxication

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Abstract: In our country, tricyclic antidepressants are usually present in most of the homes. Myocardial depression and ventricular arrhythmias are the severe side effects in tricyclic antidepressant overdose. A 4-year-old boy was brought to our hospital after taking 70 mg/kg of amitriptyline. On arrival, the patient was comatose (Glasgow Coma Score was 3), had a shallow breathing pattern with bradycardia (HR <30/min), and hypotension. He was intubated and resuscitated with multiple doses of adrenaline and sodium bicarbonate. He was infused with a bolus of 20 mg/kg of normal saline for hypotension. After 30 minutes, ventricular fibrillation was detected. Lidocaine and bicarbonate were not effective in converting the rhythm to normal, therefore, synchronized cardioversion was used. After cardioversion, the rhythm converted to ventricular tachycardia. Because ventricular tachycardia did not resolve, we administered a load of less than 2 g of magnesium sulfate for 30 minutes followed by a continuous infusion of 3 mg/min. After magnesium sulfate infusion, a normal cardiac rhythm was achieved. Magnesium sulfate is a very effective treatment in intractable arrhythmias caused by high-dose amitriptyline intoxication.

Key Words: tricyclic antidepressant, magnesium sulfate, ventricular tachycardia

Because of the increasing incidence of depression in the recent years, antidepressants have become easily available drugs frequently present in most of the homes in our country. Tricyclic antidepressant (TCA) intoxications are very important because of their severe adverse effects and probable fatal outcomes. Magnesium sulfate (MgSO₄) has an effective role in the treatment of fatal cardiac arrhythmias occurring in high-dose amitriptyline intoxication.¹,² Sustained cardiac arrhythmias seen in amitriptyline intoxication can be treated with MgSO₄ as in our case.

CASE

A 4-year-old boy was brought to our hospital in a comatose state. He was found unconscious at home with a nearly empty drug case by his bedside. Gastric lavage and activated charcoal administration were performed before his admission in a local hospital. The amount of ingested amitriptyline was estimated to be 70 mg/kg. His Glasgow Coma Score was 3, had an irregular, shallow breathing pattern with bradycardia (HR <30/min), and hypotension. His capillary refill time was longer than 3 seconds. It was not possible to make toxicological screening. The patient was endotracheally intubated and resuscitated with multiple doses of adrenaline and sodium bicarbonate. He was infused with a bolus of 20 mL/kg intravenous isotonic sodium chloride solution resuscitation followed by sodium bicarbonate infusion. Dopamine and afterload dobutamine infusion of 10 µg/kg per minute were administered. Thirty minutes after his admission, he developed tonic-clonic seizures. He was given 0.1 µg/kg of midazolam, followed by a phenytoin load of 20 mg/kg and maintenance of 5 µg/kg per day in 2 intervals. To overcome his persistent seizures, continuous infusion of midazolam and Pentothal were used. Ventricular fibrillation was detected at 30 minutes after his admission. Because lidocaine administration of 1 mg/kg was not effective in converting the rhythm to normal, synchronized cardioversion was used immediately. After cardioversion, the electrocardiogram showed nodal rhythm first with ventricular bigeminy (Fig. 1), then with ventricular tachycardia (VT) (Fig. 2). To correct VT, he was given a load of 2 g of MgSO₄ in 30 minutes followed by continuous infusion of 3 mg/min. After MgSO₄ infusion, a normal cardiac rhythm was achieved (Fig. 3). Because the patient’s clinical condition did not improve, hemoperfusion was considered.³ However, hemoperfusion did not become necessary because his cardiac rhythm returned to normal, and his seizures stopped after MgSO₄ infusion. Twenty-four hours after achievement of normal cardiac rhythm, MgSO₄ infusion was progressively decreased and stopped in the third day of the follow-up. Thiopental infusion was decreased and stopped after 24 hours. Dopamine, dobutamine, and midazolam were stopped on the third day. The patient was extubated on the fifth day. His electroencephalogram and cranial magnetic resonance results were found normal. Flash visual-evoked potentials at P100 latency were increased. The patient was discharged on the 12th day.

DISCUSSION

Tricyclic antidepressant overdosage has toxic effects over cardiovascular, autonomous nervous, and central nervous systems, and may result in cardiac conduction delays, dysrhythmia, hypotension, altered mental status, and seizures.³ Serious and lethal toxic effects have been reported with TCA intoxications of 10 to 30 mg/kg and 50 mg/kg, respectively.²,⁴ Children are more susceptible to TCA overdose, and fatal cases with ingestion of only one capsule or tablet have been reported.⁵ Tricyclic antidepressant intoxication may result in cardiac conduction delays, dysrhythmia, and hypotension. Conduction delays may become apparent on electrocardiogram as prolonged PR, QT, and QRS intervals. Sinus and supraventricular tachycardia, premature ventricular complexes, idioventricular tachycardia, VT, or fibrillation may be seen.²⁴ The most serious causes of death in TCA intoxication are persistent hypotension...
caused by myocardial depression, ventricular tachycardia, and ventricular fibrillation. In TCA intoxication, sodium bicarbonate treatment is suggested as the first agent. Sodium bicarbonate is known to decrease ventricular arrhythmia frequency, prevent QRS prolongation, and correct hypotension. This effect can be accentuated by increasing plasma sodium concentration and alkalinization. Increasing plasma sodium concentration may prevent cardiac arrhythmias by decreasing sodium channel inhibition caused by TCA intoxication. It has been reported that alkalinization decreases heart rate, shortens QRS interval, and corrects ventricular arrhythmias.

Lidocaine and phenytoin have also been used to treat dysrhythmias occurring in amitriptyline intoxication.

Magnesium sulfate is efficient in treating sustained ventricular arrhythmias, especially ventricular and torsade de pointes tachycardia and is known to prevent sudden deaths. Magnesium plays an important role in maintaining intracellular potassium levels activating sodium-potassium adenosine triphosphatase pumps. Intracellular potassium levels may also be low in presence of normal serum levels. Low intracellular potassium levels have been shown to decrease membrane threshold potentials and increase cellular excitability. Magnesium infusion is reported to

**FIGURE 1.** Nodal rhythm with bigeminy ventricular complexes.

**FIGURE 2.** Ventricular tachycardia.
increase intracellular potassium levels more significantly than potassium infusion. Antiarrhythmic effect of magnesium is based on this mechanism.\textsuperscript{2,6,7} Our patient had ingested amitriptyline more than the lethal dose. In the emergency unit, he was comatose, had bradycardia, and needed resuscitation with adrenaline and sodium bicarbonate after cardiopulmonary arrest. Sodium bicarbonate infusion was then started. Phenytoin was loaded 20 mg/kg both as an anticonvulsant and arrhythmic agent. Lidocaine bolus was given to treat ventricular fibrillation, and cardioversion was applied to correct the cardiac rhythm. Fibrillation stopped after cardioversion, but his cardiac rhythm did not return normal. Because his arrhythmia was intractable despite sodium bicarbonate, phenytoin, and lidocaine infusion, we started MgSO\textsubscript{4} infusion as reported by Citak et al.\textsuperscript{2} Considering the possible side effects, MgSO\textsubscript{4} was given 2 g as an infusion for 30 minutes, instead of bolus, and continued as 3 mg/min.\textsuperscript{2} Hypotension that developed after 30 minutes of infusion improved by decreasing the infusion rate, and no other side effects were seen. His ventricular arrhythmia stopped, and cardiac rhythm returned to normal after 30 minutes of MgSO\textsubscript{4} infusion, and p waves were apparent. Cardiac rhythm was completely normal in the 12th hour of MgSO\textsubscript{4} infusion. Infusion rate was gradually decreased and stopped on the third day. His arrhythmia did not repeat.

Our case is the second reported case after that of Citak et al.,\textsuperscript{2} being important as magnesium is effective in treating arrhythmia in high-dose amitriptyline intoxication. Magnesium sulfate is a very effective treatment in intractable arrhythmias caused by high-dose amitriptyline intoxication.

REFERENCES