Sir,

Beta2 agonist & steroid remain the main stay in the initial management of acute asthma. Aminophylline may be used in cases not responding to above treatment with certain limitations. IV Magnesium sulphate may be considered in such cases.

A 65-year-old male was admitted to the intensive care unit with complaints of severe dyspnoea. The patient had history of bronchial asthma and systemic hypertension. The patient was conscious but restless, he was unable to lie down, showed signs of respiratory distress with accessory muscle use, and was unable to speak even a single word. Breath sounds were diminished throughout both lungs with diffuse, bilateral wheezing. The other physical examination was unremarkable. Vital signs were as follows: arterial pressure 140/80 mmHg, heart rate 140 bpm & irregularly irregular, SpO2 70% in air. Temperature 100 degree F, respiratory rate 40/min with prolonged expiratory phase.

Promptly O2 with reservoir mask was administered. ABG showed pH 7.14 (7.35 to 7.45), pCO2 77 mmHg (35 to 45 mmHg), pO2 44.2 mmHg (80 to 100 mmHg) HCO3 28.4 mEq/L (22 to 26 mEq/L). The patient was nebulized with salbutamol and budesonide nebulization solution & repeated 3 times at intervals of 20 minutes, injection hydrocortisone 200 mg i.v. stat and 100 mg thrice daily started. The bronchospasm persisted. In view of atrial fibrillation, intravenous aminophylline was deferred. At this point magnesium sulfate 1.25 gm in 100 ml normal saline was infused over 20 minutes and another 1.25 gm was infused over next 30 minutes as the initial infusion showed improvement in his clinical symptoms. SpO2 came up to 92%, his chest movement could be visible and the normal pattern of respiration was resumed, FEV1 value improved to 45%. Encouraged by this result IV magnesium sulfate 2.5 gm in 500 ml normal saline was infused over next 24 hours along with alternate salbutamol and ipratropium nebulization every 6 hourly. There was continuous improvement in his symptoms & SpO2 value came up and remained above 98% after two hours of magnesium sulfate infusion. After 24 hours, magnesium sulfate infusion was discontinued but alternate salbutamol and ipratropium nebulization every 6 hourly were continued. During magnesium sulfate infusion no haemodynamic changes were noticed. Biochemical parameters in the blood were within normal limits including the magnesium level 1 mmol.L–1 (N=0.74-1.03 mmol.L–1). He was observed in the ICU for another day and then shifted to ward. At that time his respiratory rate was 12-16/minute, SpO2 98-100% in room air and FEV1 was 75% of his predicted value.

Magnesium sulphate is a safe and cheap medication with some bronchodilator effect.1 Magnesium has a predominantly intracellular action, is an important co-factor in many enzymatic reactions and is linked to cellular haemostasis. In addition, magnesium has an effect on smooth muscle cells, with hypomagnesemia causing contraction and hypermagnesemia causing relaxation of smooth muscles. There is some evidence that when magnesium is administered to asthmatic patients, it produces bronchodilatation.2 Magnesium competes with calcium for entry into smooth muscle cells,3 inhibits release of calcium from the sarcoplasmic reticulum,4 inhibit histamine release from mast cells.5 Thus I.V. Magnesium sulfate can be considered for patients with acute severe asthma who do not respond to standard therapeutic medications i.e. regular anti-asthamatics.

Aswini Kumar Sahoo1, S. C. Mohapatra2

1Associate Professor, Department of Medicine, Maharajah’s Institute of Medical Sciences, Nellimarla, Vizianagaram, Andhra Pradesh
2Professor, Department of Medicine, Maharajah’s Institute of Medical Sciences, Nellimarla, Vizianagaram, Andhra Pradesh, India

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