CASE REPORT MAGNESIUM SUPPLEMENTATION IN CYSTIC FIBROSIS; SAVING AN INFANT

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Hypo-magnesaemia is an under diagnosed condition in patients of cystic fibrosis. Here a case is reported of an imminent complete respiratory failure, which was avoided by the timely administration of Magnesium Sulphate. The case report is followed by a brief recent literature search about hypo magnesaemia and perioperative management of cystic fibrosis.

Keywords: Perioperative, hypo magnesaemia, cystic fibrosis, bronchospasm

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INTRODUCTION

Cystic fibrosis (CF) is a common, autosomal recessive disease affecting 1 in 2500 live births in Europe.¹ localized to the chromosome 7q31.¹ The prevalence in Pakistani population is unknown, however, studies from neighbouring countries have estimated the prevalence at 1 in 10,000 live births.¹ Ninety % of the morbidity and mortality in patients with CF is due to the involvement of the pulmonary system.^{1–3}

Hypomagnesaemia is a known entity found in patients with cystic fibrosis.^{1,4} This finding appears to be independent of age and other parameters of renal function (creatinine and GFR).⁴

Severe deficiency may present as muscle weakness, cramps and tetany Magnesium is relevant in patients with cystic fibrosis because of its role primarily in the respiratory system.⁵ Experience with lung transplants in cystic fibrosis patients has the highlighted role of prolonged and frequent aminoglycoside therapy as a key factor in causing hypomagnesaemia.

The combination of cystic fibrosis, hypomagnesaemia, meconium ileus leading to intestinal obstruction and aminoglycoside therapy has been reported.⁴

Magnesium is believed to function as a major calcium antagonist leading to smooth muscle relaxation. It acts a major co-factor for enzymes involved in an electron transport chain. Other roles include inhibitory action on smooth muscle contraction, histamine release from mast cells, acetylcholine from nerve terminals and also sedative effects on the brain.^{4,5} Therefore it can be assumed that it leads to ATP production, which fits the clinical efficacy, which was observed when using Magnesium in status asthmaticus etc.

CASE REPORT

A 4-month baby boy weighing 3 Kg. diagnosed with Cystic Fibrosis, Delta (F508) presented with symptoms of projectile vomiting, abdominal distension and sepsis. He had been treated repeatedly with Gentamicin. Physical examination revealed dehydration, tachycardia (HR: 150/min), tachypnea (respiratory rate >40/min). Chest auscultation displayed scattered rhonchi. SpO² was 89–92% on room air. Abdomen was distended with sluggish bowel sounds. Abdominal X-ray showed air fluid levels. Barium Meal revealed axial mal-rotation of the gut. Urgent exploratory laparotomy and Ladd's procedure (revision of mal rotated stomach) was planned.

Anaesthetic plan was rapid sequence induction using conventional anaesthetic drugs and Atracurium. At the end of surgery, the train of four (TOF) showed all 4 twitches of equal amplitude and patient was appropriately reversed. Patient was still found to have laboured breathing using accessory muscles, and had paradoxical abdomino-thoracic movement. Auscultation showed minimal to no air entry in the lower lung zones. Severe bronchospasm were suspected. Repeated Salbutamol nebulizer did not make any improvement. Sp0₂ remained low despite Fi02 of 100%.

Background of Cystic Fibrosis, prolonged aminoglycoside therapy and muscle weakness in the presence of bronchospasm prompted a decision to consider Magnesium Sulphate (MgSO4). Therefore, 0.6 meq. (0.2 meq./kg) of MgSO4 was administered intravenously over 10 minutes. A remarkable improvement was observed. The infant started using his diaphragm; the paradoxical abdomen-thoracic movement changed to impressive symmetrical chest expansion with good air entry bilaterally. Oxygen requirements decreased from 90–21% within 30 minutes, SpO₂ improved to 100%. The patient was extubated successfully in the operating theatre.

DISCUSSION

Cystic fibrosis (CF) is an autosomal recessive disease marked by recurrent childhood upper respiratory tract infections (URTI's). Ninety (90)% of the morbidity and mortality is due to the pulmonary system^{1,2} pathology. Patients suffer bronchial hyper reactivity^{1,2}and inflammatory component with asthma² or asthma-like symptoms^{1–3}. Anaesthetic implication of this disease are identical to hyper reactive airways.^{3,5}

Hypomagnesaemia is a known entity found in patients with cystic fibrosis.^{5–7} It appears to be independent of age and renal function.^{5,6} Severe deficiency may present as muscle weakness, cramps and tetany. Magnesium is relevant in cystic fibrosis because of its role primarily in the respiratory system.^{6–8}

The constellation of cystic fibrosis, hypomagnesaemia, meconium ileus, intestinal obstruction and aminoglycoside therapy is reported.^{2,5–7} It leads credence to our hypothesis that while hypomagnesaemia played a role in this child's crisis, magnesium supplementation/therapy played the critical role in relieving his resistant bronchospasm.

Magnesium is the second most common intracellular cation^{7,8} in the human body. Magnesium is believed to function as a calcium antagonist leading to smooth muscle relaxation. It is a co-factor in electron transport chain leading to ATP production. Magnesium therapy, leads to the reduction in hospitalization^{3,5} and severity markers of asthma^{1–3}. This fits the clinical efficacy, which was observed when using Magnesium in our patient.

CONCLUSION

Timely diagnosis of severe bronchospasm, its critical differentiation from muscle paralysis and prompt

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administration of intravenous magnesium sulphate saved this infant in a resource poor environment. It highlights the role of magnesium and its relevance to cystic fibrosis.

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