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Hypomagnesemic Hypocalcemia as a Cause of Persistent Upper Airway Obstruction in Acute Bacterial Tracheitis

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Bacterial tracheitis is not an uncommon cause of acute airway obstruction in infancy, characterized by moderate to high grade fever, marked inspiratory stridor, respiratory distress and polymorphonuclear leucocytosis(1). We present here an infant with acute bacterial trancheitis in whom intractable hypocalcemia secondary to an underlying hypomagnesemia lead to persistent laryngospasm and airway obstruction. It is known that 10-15% of patients in an ICU setting develop hypocalcemia(2). While cardiovascular manifestations are the common mode of presentation, laryngospasm has also been reported(2). This may lead to persistent upper airway obstruction despite resolution of underlying disease. Therefore, if not looked for, hypocalcemia may lead to diagnostic and management problems.

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Case Report

A, 2-month-old exclusively breast fed male infant, was admitted to Intensive Care Unit with history of moderate grade fever, stridor and respiratory distress of a few hours duration. The antenatal and early neonatal period had been uneventful.

The baby weighed 4.9 kg which was appropriate for age. On examination, the child was febrile, toxic and had mild cyanosis with severe respiratory distress, a respiratory rate of 68/min, inspiratory stridor and suprasternal, intercostal and subcostal recessions. Direct laryngoscopy revealed thick pus exuding from the laryngeal inlet. The epiglottis was normal and the rest of the systemic examination was non-contributory. A diagnosis of acute bacterial tracheitis was made. Investigations revealed a hemoglobin of 11.8 g/dl, total leucocyte count of 13,500/cumm with polymorphonuclear leucocytosis. The CSF examination was normal and blood and CSF cultures were sterile. Pus from trachea revealed Gram positive cocci, but culture was sterile. Lateral *X*-ray neck showed subepiglottic narrowing while *X*-ray chest was normal with a normal thymic shadow.

Initial blood gases showed metabolic acidosis with respiratory compensation (pH = 7.27; PO_2 = 100 mm Hg, pCO_2 = 28 mm Hg; HCO_3 = 12.5 mEq/L).

Child was managed with intravenous fluids and parenteral antibiotics (cloxacillin and gentamicin). The airway was secured with endotracheal intubation and respiratory care in the form of oxygenation, humidification and frequent suctioning of the tube. As the child had one episode of seizure, he was also given a loading dose of phenytoin (20 mg/kg) followed by maintenance doses. The child responded to this treatment and was extubated on the 6th day after admission. However, following extubation mild inspiratory stridor persisted. This was initially attributed to post extubation laryngeal edema and intravenous dexamethasone was started. About 48 hours later he developed high grade fever, worsening of inspiratory stridor and recurrent apneic spells. Direct laryngoscopy did not reveal any significant edema or purulent discharge but the child had to be reintubated and started on intermittent positive pressure ventilation. Next day he developed multifocal seizures despite maintenance doses of phenytoin (8 mg/kg). Phenobarbitone (3 mg/kg) was added but the seizures persisted. Investigations revealed a normal sodium and potassium, with arterial blood gases showing respiratory alkalosis (pH = 7.53; pCO_2 = 27 mm Hg; pO_2 = 70 mm Hg; HCO_3 = 22.5 mEq/L). Blood culture showed growth of *Acinetobacter loeffli*. Repeat CSF examination and cranial ultrasound were normal. Serum calcium estimation revealed hypocalcemia (serum calcium 4.0 mg/dl) with normal serum phosphorus and alkaline phosphates.

In view of the septicemia, parenteral cefotaxime was added, the child was given two bolus doses of 10% calcium gluconate (2 ml/kg) followed by maintenance calcium supplementation in doses of 90 mg/kg/day. However, seizures and inspiratory stridor persisted for the next 2 days. Repeat serum calcium also showed persistent hypocalcemia (serum calcium 5.2 mg/dl). A presumptive diagnosis of hypomagnesemic hypocalcemia was made and 2 doses of intramuscular magnesium sulphate (0.8 mg/kg/day) were given. The features of acute upper airway obstruction showed a dramatic improvement. Serum calcium levels also normalized over the next few days. The child was discharged after 20 days of hospital stay and repeat serum calcium on follow-up was normal (8.4 mg/dl).

Discussion

Hypocalcemia presenting as repeated seizures and apneic spells is a well known clinical entity in infants(3). There is a much higher prevalence of hypocalcemia in acutely ill patients than is commonly appreciated(4). When it develops in a critically ill child with features of upper airway obstruction, it can lead to exaggeration and prolongation of symptoms thereby resulting in a diagnostic and therapeutic dilemma. Hypocalcemia presenting as laryngospasm has been described particularly following neck surgery in adults. We are however, not aware of any report of a similar presentation in the pediatric age group.

In the index case, persistence of upper airway obstruction was due to intractable hypocalcemia, most probably secondary to hypomagnesemia which the child developed during the course of this illness. The absence of significant post laryngeal edema on direct laryngoscopy and the occurrence of multifocal seizures despite maintenance

doses of phenytoin and phenobarbitone were clues to the underlying metabolic etiology of the stridor. This was confirmed by the demonstration of hypocalcemia.

A number of causes are responsible for hypocalcemia in an acutely ill patient. Those which could be operative in our patient include sepsis, where an increase in free fatty acid levels causes increased calcium binding to protein leading to a resultant fall in calcium level(2,4). Respiratory alkalosis and the use of anti-convulsants like phenytoin and phenobarbitone are other factors which interfere with vitamin D metabolism(2) and were contributory in this case.

The most probable causes of the resistant hypocalcemia here, however, appear to be an underlying hypomagnesemia as evidenced by the persistence of stridor, seizure and hypocalcemia despite 3 days of calcium supplements and the prompt clinical and biochemical response following magnesium supplementation. Normal plasma phosphate is another pointer to hypomagnesemia as an underlying cause of hypocalcemia(5). We were handicapped by the non-availability of serum magnesium levels at our centre. Further, it is known that there is a very poor correlation between serum magnesium levels and clinical features of hypomagnesemia(3). Serum magnesium abnormalities are common in such critically ill patients(6,7). Hypocalcemia is a frequent manifestation of magnesium depletion(4). The most common causes of hypomagnesemia include protein energy malnutrition, sepsis, diuretic use and aminoglycoside administration(6). The etiology of hypomagnesemia in our patient was therefore probably multifactorial with hospital acquired sepsis, aminoglycoside therapy and decreased intake due to prolonged intravenous administration being important contributory factors.

In conclusion, metabolic factors like hypocalcemia and hypomagnesemia should always be kept in mind in the management of acutely ill patients in Intensive Care Units. Early diagnosis and appropriate therapy can lead to considerable decrease in morbidity and long term complications.

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