Hypocalcaemia Leading to Difficult Airway in Sepsis

Sarika Katiyar, S. Srinivasan, Rajesh Kumar Jain

Hypocalcaemia is frequently encountered in patients who are critically ill. Presentation varies widely from asymptomatic to life threatening situations. We report a case of difficult laryngoscopy in a severely hypocalcaemia septic patient.

Case History
A young female patient of average build presented with intestinal obstruction in the emergency department. She was operated on the same day in emergency. Jejunostomy was done along with resection of the gangrenous bowel on the same day. She was extubated on 7th postoperative day and was in ICU on non invasive ventilation for a total of 15 days due to septicemia. In the ICU she received multiple transfusions and was on total parenteral nutrition. Later on she was shifted to ward for further management. Her second surgery after 4 weeks for jejunostomy closure was uneventful. After 4 days of closure, patient developed anastamotic leak and was taken in emergency for reexploration.

Routine investigations including blood count, blood urea, serum creatinine, serum sodium, and serum potassium, chest x-ray were within normal limits. Serum calcium (total) was reported to be 4.1mg/dl. She had symptoms of hypocalcaemia including masseter spasm, chvostek's sign, trousseau's sign. No ECG changes were present.

30 ml of calcium gluconate (270mg) was given over 30-45 minutes. Her heart rate was 110/min and BP-110/70mm of Hg, CVP-8mm of Hg. Urine output was adequate >100ml/hr. Her chest was clear but she was tachypnoeic. In the OT we attached all the monitors, started two IV drips of ringer lactate and colloid along with calcium gluconate infusion 2mgkg⁻¹hr⁻¹. After preoxygenation general anaesthesia was induced with inj. fentanyl 75mcg, midazolam1mg, thiopentone 250mg, and then atracurium 25mg was used for intubation. After 3 minutes of mask ventilation, laryngoscopy was tried, but it failed, due to inability to open her mouth because of masseter spasm. Laryngoscopy was tried again by senior consultant but failed. After giving more relaxant and adequately ventilating the patient on mask second time, laryngoscopy (rather by pushing the blade in between jaws) was done by senior consultant while assistant was trying to forcibly open her mouth.

Intubation was accomplished with ETT 7.5 mm using a stylete with great difficulty. ETT was secured along with an airway in the mouth and anaesthesia was maintained on atracurium 0.1mg/kg along with O2+N2O in 1:1 ratio and isofluorane. Her intraoperative vitals were stable. During surgery of 2hrs we had sent a sample of serum calcium and others electrolytes including magnesium, sodium, potassium and phosphate. Jejunostomy was done and patient shifted to ICU for further care and management. In the ICU after 6hrs, her serum calcium was 6.4 mg/l while calcium infusion was going on and patient was mechanically ventilated. On 3rd postoperative day when all other hemodynamic parameters were normal and all electrolyte imbalances were corrected, patient was given a trial of weaning. She tolerated it well and she was extubated and shifted to ward the on 4th postoperative day.

DISCUSSION
This whole sequence forced us to search for the cause of difficult laryngoscopy in this case, inspite of adequate relaxant use. The cause could have been severe hypocalcaemia associated with sepsis. Hypocalcaemia is not an unusual finding in patients hospitalized for critical illnesses and has also been described during post surgical procedures. Numerous factors have been suggested for hypocalcaemia in these situations such as changes in albumin affinity for calcium, chelation by citrate from blood transfusions, or resistance to PTH or vitamin D action.

Hallmark of hypocalcaemia is neuromuscular irritability with symptoms ranging from paresthesia to tetany and seizures. Hypocalcaemia may augment the neuromuscular blockade caused by non-depolarizing muscle relaxants. This patient was in severe hypocalcaemia and was symptomatic prior to emergency surgery. We should have corrected hypocalcaemia prior to induction. Hypocalcaemia increases resting membrane potential. This increases the nerve excitability leading to masseter spasm. This action of calcium is at the neural level whereas relaxant acts distal to it i.e. on neuromuscular junction. Thus resistance

Drs. Sarika Katiyar, Asst. Professor, S. Srinivasan, Asst. Professor, Rajnish Kumar Jain, Prof and Head, Department of Anesthesiology and Critical Care, Bhopal Memorial Hospital and Research Centre, Bhopal-462038, India
Correspondence: Dr. Sarika Katiyar, E-mail: katiyarsarika@yahoo.com
to the effect of relaxant was seen in our case.

Simultaneously this patient was in ICU for fifteen days after first surgery. She was on ventilator around seven days with relaxant use. Patient had developed resistance to nondepolarizing muscle relaxant. Chronic competitive antagonism of the neuromuscular junction induces up regulation of the postjunctional receptors. The new receptors differ from the normal receptors both in location and properties. These are extrajunctional and in severe disease may be found over the entire muscle membrane. Their half-life is much shorter, less than 24 hrs compared with 2 weeks for mature receptor. This also exaggerated the resistance to neuromuscular relaxant.

Anaesthetic management in such cases should be directed at maintaining calcium levels and minimizing the consequences of hypocalcaemia. Calcium, phosphate and magnesium should preferably be checked before any emergency surgery in a septic patient and corrected if abnormal. The cornerstone of therapy for confirmed, symptomatic ionized hypocalcaemia is calcium administration. In patients who have severe hypocalcaemia or hypocalcaemic symptoms, calcium should be administered intravenously. In emergency situation in an average sized adult, the rule of 10's advises infusion of 10 ml of calcium gluconate over 10 minutes followed by a continuous infusion of elemental calcium, 0.3-2mgkg−1hr−1 (i.e. 3-16 mlhr−1 of 10% calcium gluconate for a 70 kg adult.) Calcium salts should be diluted in 50-100ml distilled water, should not be mixed with bicarbonate (to prevent precipitation) and must be given cautiously to digitalized patients because calcium increases the toxicity of digoxin. Once the ionized calcium is stable in the range of 4-5 mgdl−1, oral calcium supplements can be substituted for parenteral therapy. It should be remembered that respiratory alkalosis can acutely exacerbate hypocalcaemia. ECG should be monitored for QT interval or any block. Hypocalcaemia may impair coagulation and should be monitored prior to regional anaesthesia. Even extubation can be complicated due to laryngospasm. Thus anaesthetic management should aim at maintaining calcium homeostasis in the perioperative period.

REFERENCES
1. Desai TK, Carlson RW, Geheb MA. Prevalence and clinical implications of hypocalcaemia in acutely ill patients in a medical intensive care setting. Am J Med. 1988; 84: 209-214.
2. Zaloga GP, Chernow B, Cook D, Snyder R, Clapper M, O’Brian JT. Assessment of calcium homeostasis in critically ill surgical patient: the diagnostic pitfalls of the Mclean-Hastings normogram. Ann Surg. 1985; 202: 587-594.
3. Chernow B, Zaloga G, Mc Fadden E, et al. Hypocalcaemia in critically ill patients. Crit Care Med.1982; 10: 848-851.
4. Zaloga GP, Chernow B. Stress induced changes in calcium metabolism. Semin Respir Med. 1985; 7: 52-68.
5. Drop LJ, Javer MB. Low plasma ionized calcium and response to calcium therapy in critically ill man. Anesthesiology 1975; 43: 300-306.
6. Sibbald WJ, Sardesai V, Wilson RF. Hypocalcaemia and nephrogenous c-AMP production in critically ill patients. J Trauma. 1977; 17: 766-784.
7. Hauser C Kamrath R, Sparks J, Shoemaker W. 1983 Calcium homeostasis in patients with pancreatitis. Surgery. 1983; 94: 830-835.
8. Fogh-Anderson N, Frederiksen PS, Andersen EA, Thode J. Relation between ionized calcium and pH in infants with acute acid base disturbances. Clin Chim Acta. 1983; 130: 357-361.
9. Thode J, Fogh-Andersen N, Wimberley PD, Moller Sorensen A, Siggard Andersen O. Relation between pH and ionized calcium in vitro and in vivo in man. Scand J Clin Lab Invest. 1983; 43 (Suppl 165): 79-82.
10. Kancir CB, Hyltoft Peterson P, Wandrup J. Plasma ionized calcium during pediatric anesthesia: effects of pH and succinylcholine. Can J Anaesth. 1987; 34: 391-394.
11. Hinkle JE, Cooperman LH. Serum ionized calcium changes following citrated blood transfusions in anesthetized man. Br J Anaesth 1971; 43: 1108-1121.
12. Denlinger JK, Nahrwold ML, Gibbs PS, Lecky JH. Hypocalcaemia during rapid blood transfusion in anesthetized man. Br J Anaesth. 1976; 48: 995-1000.
13. Zaloga GP, Chernow B. The multifactorial basis for hypocalcaemia during sepsis. Ann Intern Med. 1987; 107: 36-41.
14. Donald S Prough, J. Sean Funston, Christer H. Svensen, Scott W Wolf. Fluid electrolyte and acid base physiology. In: Barasch .P. editor. Clinical Anesthesia. 6th edition. 2009: 316-318.