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Postoperative hypocalcaemia in a young child: an unusual presentation

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Key points

The calcium ion is essential for many biological processes. Of particular relevance to the anaesthetist are the effects on the myocardium, vascular smooth muscle and blood coagulation.

Abstract

A case report of a young male child, who had postoperative hypocalcaemia of acute origin and needed Intensive care unit admission / assisted ventilation for the treatment is described. In most of the cases, it is not possible to definitely distinguish whether an observed event is usual convulsion or due to electrolyte imbalance.

Keywords: acute hypocalcaemia, ICU management, pediatric.

Introduction

Hypocalcaemia is defined as "a decrease in total plasma calcium level below 8.5 mg/dl or 2.20 mmol/lt in the presence of a normal plasma protein concentration." It presents clinically as tetany, seizures, muscle cramps, laryngospasm, bronchospasm, carpopedal spasm, irritability, confusion, dementia, and hallucinations. In this case study, the patient was hyperexcited and was in uncontrolled state, so intubation and assisted ventilation was necessary.

Case Report

A 12 yrs old male presented with restlessness, disorientation, facial twitchings and confusion. The patient had undergone rectopexy under spinal anaesthesia 6 hrs back. Preoperative and intraoperative periods had been uneventful. There was no history of thyroid surgery or neck trauma in the past. He had a history of gastritis for which he was taking tabiet cimetidine since 2 years. The patient was malnourished, his weight was less for his age and there was history of chronic diarrhea.

Examination revealed:

- an uncooperative patient, running away from bed;
- uncommon voice of laryngeal stidor;
- hyperventilation;
- facial twitchings;
- not responding to oral commands, but reaction to painful stimuli was present;
- carpopedal spasm, and he was unable to breathe because of laryngeal stidor.

We attended the call in the ward, and since the patient was having severe stidor, an intravenous propofol 1mg/kg was given and the trachea of the patient was intubated with cuffed endotracheal tube number 7.0. The patient was then shifted to the Intensive care unit on 100% oxygen using Bains circuit for further diagnosis and treatment. In the ward, the surgeons had already given intravenous diazepam to the patient, as

they were suspecting of simple convulsions. But when the patient did not settle, they sent for an ICU call. In the intensive care unit, the patient was put on assisted ventilation using synchronized intermittent mandatory ventilation mode. On examination cardiovascular and respiratory systems did not reveal any abnormality, i.e., there was no cardiomegaly, heart sounds were normal, no crepts or rhonchi and no signs of cardiac failure or pulmonary oedema. Fundus examination was normal, and the skin and hair did not show any abnormality. There was no signs of rickets on examination. Patient maintained Saturation of 98% on ventilator with 50% oxygen. Electrocardiogram showed a prolonged QT interval. In the unit X- ray chest was done which was found to be normal. Other investigations revealed a hemoglobin of 11 gm/dl, and the peripheral smear, Serum proteins, blood urea, and blood glucose were all within normal limits. Electrolytes showed a Na^+ level of 140 meq/lt, Cl^- of 110 meq/lt, Mg^{2+} of 1.5 meq/lt, Ca^{2+} of 6 mg/dl and serum albumin of 4 mg/lt. Blood gas analysis was normal, with no acidosis or alkalosis. There was no hypoxia or hypercarbia. Such patient if was not ventilated or treated immediately, can have cardiac arrhythmias, congestive cardiac failure and severe hypotension because of severe hypocalcaemia. The patient was given intravenous calcium gluconate, 10 ml 10% slowly over a period of 10 minutes under ECG monitoring. The patient showed some improvement for 1 hr and then again became uncooperative due to laryngeal stidor. It was very difficult to wean off the patient at this moment, and thus it was decided to ventilate the patient till the serum calcium becomes normal. The patient was then started on Calcium gluconate infusion which was started at the rate of 0.5 mg/kg/hr and was later on increased to 1 mg/kg/hr. Vitamin D_3 sachet (25,000 u) was also started via ryle's tube one sachet once a day. After two days of treatment, the patient became fully conscious and was gradually weaned off the ventilator. On the third day, he was put on T- piece trial with oxygen and was observed till evening and was then finally extubated. That he took a long time to wean off from the ventilator was may be due to the severity and chronicity of the illness. Series of serum electrolytes were done to keep their levels within normal limit. On the fourth day, the patient was shifted to surgical ward.

Discussion

The normal total serum calcium is 8.2 to 10.2 mg/dl. Free calcium or the ionized form of calcium, is the physiologically active component of calcium, and it measures 4.8-7 mg/dl. The laboratory tests, indicate the total serum calcium levels and not the ionized calcium levels. So, hypocalcaemia may be wrongly diagnosed if there is a decrease serum albumin, although the ionized calcium is unchanged. The correction factor that increases total calcium by 0.75 gm/dl is: for each 1 gm/dl decrease in albumin <3.5 gm/dl. Total Serum calcium level does not always reflect the ionized ca level. Ion specific electrodes can however directly measure ionic calcium. [1,4]

Severe hypocalcaemia with life threatening symptoms should be treated along with supportive treatment, i.e., oxygen, ventilatory support, and intensive monitoring. Calcium regulation is critically needed for normal cell function, neural transmission, membrane stability, bone structure, blood coagulation and intracellular signaling. So, depending on the cause, unrecognized or poorly treated hypocalcaemic emergencies lead to morbidity or death. [1]

Family history of hypocalcemia should always be excluded. Laboratory studies should include measurements of free serum calcium, phosphate, magesium, creatinine and Parathyroid Hormone. Hypocalcaemia may occur with - low Parathyroid Hormone level in conditions like parathyroid agenesis, destruction, reduced function of the parathyroid glands and in high Parathyroid Hormone level in conditions like vitamin D deficiency, Parathyroid Hormone resistance syndromes, ca lcium chelators, and pancreatitis.[2,3] Almost always, changes in serum

calcium or phosphate in an Intensive care unit setting , serve as markers of the severity of illness.

In a hypocalcaemic patient, past history should be explored for pancreatitis, renaldiseases, liverdiseases, Gastrointestinal disorders, hyperthyroidism, sepsis, burns, massive transfusion and hyperparathyroidism.[1, 2, 4]

Points to ponder

Always evaluate dietary calcium intakes. Cimetidine decreases the gastric pH and thereby slows down calcium absorption. Calcium is bound to serum proteins, 50% chiefly to albumin. An additional 5-10% is in combination with bicarbonate, and the rest is in free form. Check ionized calcium and repeat serum electrolytes regularly. Intravenous replacement of calcium is recommended in acute symptomatic cases, in doses of 90-100 mg of elemental Calcium (10 ml diluted in 50 ml of 5% dextrose / Normal saline over 5-10 minutes). This dosage raises the ionized Calcium level to 0.5-1.5 mmol and lasts for 1-2 hrs. This is then followed by 10 ml of calcium gluconate diluted in 100-200 ml of 5% dextrose/ Normal saline, to be given over 2-3 hrs.

Calcium infusion should be started at 0.5 mg/kg/hr and increased to 2 mg/kg/hr. [1,2,3,4]

Main pitfalls that are to be considered are:

- do not fail to consider hypoalbuminemia as the cause of hypocalcaemia;
- don't consider hypocalcaemia as laboratory error. [1, 5] Calcium regulation is maintained by Parathyroid Hormone, Vitamin D and calcitonin through complex feedback loops. They act primarily at bone, Gastrointestinal and renal sites. Vitamin D directly targets Gastrointestinal absorption of calcium. 90% of calcium is found to be in bones. Moderate to severe hypocalcaemia is always symptomatic. The approach to treatment depends upon the severity of the hypocalcaemia, the rapidity with which it develops, and the accompanying complications especially seizures and laryngospasm. [3, 4]

Acute hypocalcaemia presents with hyper excitability, tetany, seizures, bronchospasm, dementia, delirium, psychosis, hallucinations, stridor, Congestive cardiac failure and bradycardia. Electrocardiogram might show a prolonged QT interval.

Calcium correction to be done is Calcium [mg/dl] = total Ca + 0.8 [4.4 - S. albumin], where 4.4 represents normal range of serum albumin.

Treatment is started with calcium gluconate intravenously as 10 ml of 10% solution over a period of 10 minutes, followed by infusion which is made by adding 10 ampoules of 10% calcium gluconate in 1 litre of 5% dextrose and infused over 24 hrs under ECG monitoring. Hypomagnesaemia if present should always be treated. [3 ,4]

1 gm of elemental calcium/day should always be given for 10 days followed by vitamin D_2 or D_3, i.e., 25,000-50,000 Units daily and then 2-3 times / week for several months. Calcium 1gm per day is needed for 6-12 weeks. The main goal is to bring Serum calcium into the low normal range to avoid hypercalciuria. [3 ,4]

Serum calcium should be monitored weekly and then every 1-3 months till the level becomes normal. Also dietary phosphate restriction is needed in case of renal failure. [6]

Haltermen J.S. published a case of a breast fed infant who presented with profound stidor, and was found to be hypocalcaemic on investegation. [7]

Seerat Z et al. found a teenage boy to be hypocalcaemic, who had presented with generalized tonic fits but did not have a history of rickets.[8]

Conclusions

Emphasize on the importance of SERUM electrolyte levels, which should be done preoperatively especially in GASTRO intestinal cases, even if the patients are young and also in patients with altered mental state, Neuro-Muscular irritability, and laryngospasm. [9]

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