

# Acute hyperventilation leading to hypocalcaemia during spinal anaesthesia

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## Summary

The most common cause of hypocalcaemia under general anaesthesia is acute mechanical hyperventilation, but hypocalcaemia during spinal anaesthesia has not been reported. This case report describes the development of hypocalcaemia due to hyperventilation in a patient undergoing appendicectomy under spinal anaesthesia. The diagnostic features of hypocalcaemia and its treatment are explained in detail.

**Key Words:** Hypocalcaemia, Spinal anaesthesia.

## Introduction

Hypocalcaemia during anaesthesia is a rare entity. Acute onset hypocalcaemia has variable presentations, many of which can be life threatening. The signs and symptoms of hypocalcaemia are characteristic<sup>1</sup>, thus can be treated in emergency conditions without waiting for the result of ionized serum calcium measurement. We present a case in which the patient developed hypocalcaemia under spinal anaesthesia.

## Case history

A 30 year old male (weight – 65 kg, height – 170 cm) with familial neurofibromatosis, presented for appendicectomy. As the patient was anxious and refused general anaesthesia, spinal anaesthesia was planned. Preoperative investigations were within normal limits.

Under standard monitoring, spinal anaesthesia was performed in the right lateral position in L2-3 space using a 26G Quincke needle. 3.5ml of 0.5% heavy bupivacaine was injected in the subarachnoid space. The desired level of block to T6 was obtained and surgery was allowed to commence. Pethidine 30mg IV was given to control shivering and midazolam 2mg IV was used to sedate the patient. Thirty minutes after the insertion of the spinal, midway through the surgery, the patient became restless, complaining of perioral numbness and of inability to breathe. On examination, the level of sensory block had increased to T4. Chest wall movement was adequate, oxygen saturation was 100% and systolic BP was 100mmHg. The patient was reassured and 1 mg midazolam was given intravenously for further sedation. However, the patient became more restless, dyspnoea and tachypnoea persisted and his speech became slurred. On auscultation,

his chest was clear. Trousseau's sign<sup>2</sup> was noticed on the left hand, where the sphygmomanometer cuff was placed and inflated for measurement. A blood sample for the measurement of ionized calcium and blood gases was drawn. An infusion of 20 ml of 10 percent calcium gluconate in 100ml of 5% dextrose was given over 20 minutes. The Trousseau sign disappeared five minutes after calcium administration and patient improved symptomatically. After 20 minutes, a second infusion of calcium gluconate was begun and surgery was completed without further complication.

The patient was transferred to the recovery room in a fully conscious and haemodynamically stable state while receiving the calcium gluconate infusion. The intraoperative ECG did not show a prolonged QT interval. The intraoperative ionized calcium level was low (0.6mmol/L) and blood gases showed a respiratory alkalosis (PaO<sub>2</sub> =160mmHg, PaCO<sub>2</sub> =22mmHg (2.9kPa, pH =7.57 and HCO<sub>3</sub> =18mmol/l). Subsequent calcium concentrations were within the normal range. An endocrinologist was consulted, who did not detect a pathological cause for the episode of hypocalcaemia.

## Discussion

The most common cause of decreased total serum calcium is hypoalbuminaemia.<sup>1</sup> This is artifactual as ionized calcium remains normal. The ionized calcium may be roughly estimated by the following formula. Corrected calcium (mmol/L) = Serum calcium (mmol/L) + 0.8 [4-Serum albumin (gm/dl)]. Ionized calcium should be measured whenever true hypocalcaemia is suspected. Binding between calcium and protein is enhanced when serum pH increases, resulting in decreased ionized calcium. An increase from a pH of 7.4 to 7.6 may decrease ionized calcium by 0.25 mmol/L and precipitate tetany.

Respiratory alkalosis secondary to hyperventilation is probably the most common cause of acute ionized hypocalcaemia<sup>1</sup>, and this appears to be the case in the operating room.<sup>3</sup> However, it has not been reported during spinal anaesthesia. Many patients complain of difficulty in breathing following spinal anaesthesia probably because of loss of proprioceptive sensation from intercostal and abdominal muscles.

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In addition, high intercostal paralysis is accompanied by a feeling of suffocation<sup>4</sup>, leading to tachypnoea, hyperventilation and consequent respiratory alkalosis and hypocalcaemia. This is the most likely mechanism in this anxious patient, in whom the level of sensory blockade extended to T4. The manifestations of hypocalcaemia were the presence of Trousseau sign, irritability, and circumoral paraesthesia. Trousseau sign presents as carpal spasm secondary to ulnar and median nerve ischemia in response to the inflation of the sphygmomanometer cuff to 20mm Hg above systolic blood pressure.<sup>2</sup> We did not observe obvious QT interval prolongation although this lacks sensitivity for the diagnosis of hypocalcaemia.<sup>6</sup>

Emergency treatment of hypocalcaemia is elemental calcium, 100 to 300mg (10 to 30 ml of 10% calcium gluconate) diluted in 150 ml of 5% dextrose over 10 minutes. This usually increases the ionized calcium concentration by 0.5 to 1.5 mmol/L.

Although in the operating theatre, it is possible to raise serum CO<sub>2</sub> level by the use of rebreathing of exhaled carbon oxide. The use of semi open systems (Mapleson A to F) with appropriate flow or circle with absorber excluded and a low flow of gases would have achieved it.<sup>8</sup> The resulting decrease in pH leads to a normalization of ionized calcium level. However, we were not able to use it because of the difficulty encountered in providing a tight mask fitting in a restless patient.

In conclusion, a high level of sensory and motor blockade under spinal anaesthesia can produce such respiratory discomfort (hyperventilation) in anxious patients. This hyperventilation can result in respiratory alkalosis that increases calcium binding to albumin causing hypocalcaemia. Acute hypocalcaemia is an emergency that requires prompt attention. If symptoms of neuromuscular irritability are

present and carpopedal spasm is elicited on physical examination, treatment with intravenous calcium is indicated until the signs and symptoms of hypocalcaemia subside.<sup>9</sup> Acute onset hypocalcaemia should be suspected and treated accordingly in patients undergoing spinal anaesthesia without waiting for the calcium level.

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