Egyptian Journal of Anaesthesia (2013) **29**, 333–335

Egyptian Society of Anesthesiologists

Egyptian Journal of Anaesthesia

www.elsevier.com/locate/egja
www.sciencedirect.com

Case report

Hyperventilation causing symptomatic hypocalcaemia during labour in a parturient

Teena Bansal *, Sarla Hooda

Department of Anaesthesiology & Critical Care, Pt. B.D. Sharma University of Health Sciences, Rohtak, Haryana 124 001, India

Received 12 January 2013; revised 27 April 2013; accepted 21 May 2013
Available online 15 June 2013

KEYWORDS

Hyperventilation;
Hypocalcaemia;
Parturient

Abstract Hyperventilation often occurs under stressful conditions and has been reported before, during or after anaesthesia and surgery. Hyperventilation is characterised by multiple somatic symptoms due to hypocalcaemia induced by respiratory alkalosis because of inappropriate hyperventilation. It should be managed as an emergency. Here, we report a case of 30 year old full term parturient presenting in labour in a highly anxious state, presenting with symptoms and signs of hypocalcaemia in the form of circumoral numbness along with carpal spasm. The patient was managed successfully with reassurance and intravenous calcium gluconate.

1. Introduction

Hyperventilation syndrome often occurs under stressful conditions which cause hypocapnia and consequently results in respiratory alkalosis and a wide range of somatic symptoms. Respiratory alkalosis can induce secondary hypocalcaemia that may cause cardiac arrhythmias, conduction abnormalities and various somatic symptoms such as paraesthesia, hyperreflexia, convulsive disorders, muscle spasm and tetany [1]. Acute hypocalcaemia is an emergency that requires prompt attention and management. Here, we report a case of a parturient having excessive hyperventilation during active labour and presenting as circumoral numbness and carpal spasm along with its anaesthetic management.

2. Case report

An otherwise healthy 30 year old full term parturient weighing 50 kg and 5 feet in height, G_4P_2 presented in labour in a highly anxious state. Anaesthesia consultation was sought because of non-progression of labour. She was hyperventilating with a respiratory rate of 30/min. Pulse was 96/min and blood pressure was 130/80 mmHg. After some time, she complained of circumoral numbness and her both wrists were in extreme flexion (carpal spasm). Arterial blood gas analysis revealed pH – 7.68, $PaCO_2$ – 24 mmHg and plasma HCO_3^- – 30 mmol/L. ECG showed prolonged QT interval. Samples of blood were drawn for estimation of serum calcium, proteins and parathormone levels. An impression of severe anxiety induced alkalosis causing hypocalcaemia was made. ***No anxiolytic was given,*** the patient was reassured, and infusion of 10 ml of 10% calcium gluconate was given over 10 min. The patient improved

* Corresponding author. Address: 2/8 FM, Medical Campus, PGIMS, Rohtak, Haryana 124 001, India. Tel.: +91 9315839374.
E-mail address: aggarwalteenu@rediffmail.com (T. Bansal).

Peer review under responsibility of Egyptian Society of Anesthesiologists.

ELSEVIER Production and hosting by Elsevier

 http://dx.doi.org/10.1016/j.egja.2013.05.008

symptomatically over next 15–20 min. Due to non-progress of labour, caesarean section was planned by obstetrician. ***Foetal heart sound varied from 120–140/min***. Systemic examination revealed normal heart sounds and chest was clear bilaterally. Airway assessment was unremarkable and spine was normal. Haemoglobin, bleeding time, clotting time, urine examination and INR were normal. Standard monitors were attached. Spinal anaesthesia was planned for the procedure. It was given in L_3–L_4 interspace with 25 G Quincke babcock needle using 1.8 ml of 0.5% hyperbaric bupivacaine. Adequate sensory block was achieved up to T4. ***APGAR Score of neonate was 7 and 9 at 1 and 5 min*, respectively**. Intraoperative and postoperative period was uneventful. ***Postoperatively patient was administered diclofenac 75 mg intramuscularly 8 hourly for next 24 h***.

Ionised serum calcium level was low (0.7 mmol/L), while serum proteins were 7.2 gm/dl, and parathormone levels were 20 pg/ml In the recovery room, repeat arterial blood gas analysis was done which revealed pH – 7.36, $PaCO_2$ – 36 mmHg and HCO_3^- – 23 mol/L. Subsequent calcium concentrations were 1.3 mmol/L, well within normal range. Postoperatively USG of parathyroid was done which was normal. An endocrinologist was consulted who did not suspect any pathological cause for the episode of hypocalcaemia. The final clinical impression was that the patient was having hypocalcaemia due to respiratory alkalosis due to hyperventilation caused by severe anxiety because of outcome of pregnancy per se, labour pains and non-progression of labour.

3. Discussion

Respiratory alkalosis secondary to hyperventilation is probably the most common cause of acute ionised hypocalcaemia, and this appears to be the most likely cause in our case. Binding between calcium and protein is enhanced when serum pH increases, resulting in decreased ionised calcium. Ionised calcium should be measured whenever true hypocalcaemia is suspected [2]. Normal range of total serum calcium is 8.0–10.2 mg% or 2.2–2.5 m mol/L. About half of total calcium is ionised calcium (normal 4.0–4.6 mg% or 1.0–1.5 m mol/L), which is physiologically active. Ionised calcium may be roughly estimated by the following formula:

$$\text{Corrected calcium(m mol/L)} = \text{Serum calcium(m mol/L)} + 0.8[4 - \text{Serum albumin(gm/dl)}].$$

Other causes of hypocalcaemia are chronic kidney disease, hypoparathyroidism, acute pancreatitis, hypomagnesemia, sepsis, hyperphosphatemia and medications (anticonvulsants and agents used to treat hypercalcemia) which were excluded in our case by history, examination and investigations.

In the operating theatre, when severe hyperventilation is suspected as a cause of respiratory alkalosis induced hypocalcaemia, it is possible to raise serum CO_2 level by the use of rebreathing of exhaled CO_2. The use of semiopen systems with appropriate flow or circle with absorber excluded and a low flow of gases can achieve rise in serum CO_2 [3]. The resulting decrease in pH leads to a normalisation of ionised calcium level. In the present case, we did not use these measures and resorted to intravenous calcium gluconate because the patient was having non-progression of labour, and we wanted to buy time.

Hyperventilation has been described in different settings of anaesthesia. Kumar et al. reported hypocalcaemia due to hyperventilation in a 30 year old male patient posted for appendicectomy 30 min after insertion of spinal anaesthesia and attributed it to respiratory alkalosis secondary to hyperventilation [2]. In another report, Moon et al. reported hypocalcaemia and hypokalemia due to hyperventilation during spinal anaesthesia at 90 min of surgery in a 51 year old male posted for osteotomy and internal fixation for hallux valgus [1].

Cralg et al. reported hyperventilation induced transient spastic quadraparesis in a full term parturient in labour. They concluded that awareness of this differential diagnosis and prevention of extreme hyperventilation is important in the management of obstetric patients especially those without prenatal education [4]. There is also a report of tetany induced by hyperventilation in a dental patient [5]. Han et al. reported carpal spasm due to hyperventilation syndrome in a patient after spinal anaesthesia and attributed it secondary to the environment and anxiety of the operating room [6].

Mizuno et al. described hyperventilation syndrome before induction of and after awakening from general anaesthesia in a 53 year old woman with no central nervous system and psychiatric disease who was scheduled for left total hip arthroplasty under general and epidural anaesthesia. These authors purposed that her hyperventilation syndrome was induced by anxiety and stress of operation before induction and again after awakening from general anaesthesia [7].

Acute hypocalcaemia is an emergency that requires prompt attention as patients may present with tetany, seizures, cardiac arrhythmias or laryngeal spasm. The hallmark of acute hypocalcaemia is neuromuscular irritability. Patients often complain of numbness and tingling in their fingertips, toes and the perioral region and may present as carpopedal spasm. Clinically neuromuscular irritability can be demonstrated by eliciting chvostek's sign or trousseaus' sign. Trousseaus' sign (carpal spasm) is elicited by inflation of a blood pressure cuff to 20 mmHg above the patient's systolic blood pressure for 3–5 min. Carpal spasm presents as flexion of the wrist in metacarpal phalangeal joints, extension of the interphalangeal joints and abduction of the thumb. 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 without waiting for serum calcium levels [8]. Calcium gluconate is the preferred intravenous calcium salt as calcium chloride often causes local irritation. Calcium gluconate contains 90 mg of elemental calcium per 10 ml ampule, and usually, 1–2 ampules (180 mg of elemental calcium) diluted in 50–100 ml of 5% dextrose is infused over 10 min. This can be repeated until the patient's symptoms have improved [9].

Anaesthesia in the presence of hypocalcaemia requires special consideration. During anaesthesia, several factors may alter the serum ionised calcium level and thus potentiating the adverse effects of hypocalcaemia in susceptible patients like patients with malnutrition and low albumin, abnormal acid base status and electrolytes. The drugs used during the perioperative period and transfusion of large volumes of citrated blood also cause hypocalcaemia. The anaesthetist should aim to prevent further changes in the plasma calcium concentration and to recognise and treat adverse effects of hypocalcaemia [10].

Anaesthetic management should aim at maintaining calcium homoeostasis in the perioperative period. Hypocalcaemia impairs coagulation and therefore coagulation tests should be ordered prior to regional anaesthesia especially if hypocalcaemia is suspected. Serial ionised calcium levels should be monitored intraoperatively in patients with history of hypocalcaemia. Alkalosis should be avoided to prevent further decrease in calcium. ECG should be monitored for QT interval or any conduction disturbance [11]. If general anaesthesia is planned, potentiation of the negative inotropic effects of barbiturates and volatile anaesthetics should be expected. Responses to neuromuscular blocking agents are inconsistent and require close monitoring with a nerve stimulator. Even extubation can be complicated due to laryngospasm.

Further, a high level of sensory and motor blockade under spinal anaesthesia can produce hyperventilation in anxious patients which can result in respiratory alkalosis causing hypocalcaemia. If symptoms of neuromuscular irritability including carpopedal spasm are present, patient should be treated with intravenous calcium without waiting for serum calcium levels.

References

[1] Moon HS, Lee SK, Chung JH, In CB. Hypocalcemia and hypokalemia due to hyperventilation syndrome in spinal anaesthesia – a case report. Kor J Anesth 2011;61:519–23.

[2] Kumar N, Podder S, Sinha T, Puri GD. Acute hyperventilation leading to hypocalcemia during spinal anaesthesia. South Afr J Anaesth Analg 2004;10:29–30.

[3] Brockwell RC, Andrews JJ. Inhaled anaesthetic delivery systems. In: Miller RD, editor. Anesthesia. New york: Churchill Livingstone; 2000. p. 191–4.

[4] Cralg BA, Panni MK. Hyperventilation-induced transient spastic quadriparesis. Br J Anaesth 2004;93:474–5.

[5] Geffner I, Murgatroyd J. Tetany a case induced by hysterical hyperventilation. Br Dent J 1980;148:264.

[6] Han MA, Ha KH. Carpal spasm due to hyperventilation syndrome after spinal anaesthesia. Intraven Anesth 2005;9:149–52.

[7] Mizuno J, Morita S, Itou Y, Honda M, Momoeda K, Hanaoka K. Hyperventilation syndrome before induction of and after awakening from general anaesthesia. Masui 2009;58:768–71.

[8] Bringhurst FR, Demay MB, Kronenberg HM. Hormones and disorders of mineral metabolism. In: Wilson JD, Foster DW, Kronenberg HM, Larsen PR, editors. Williams textbook of endocrinology. Philadelphia: WB Saunders; 1998. p. 1155–209.

[9] Cooper MS. Diagnosis and management of hypocalcemia. BMJ 2008;336:1298–302.

[10] Aguilera M, Vaughan RS. Calcium and anaesthetist. Anaesthesia 2000;55:779–90.

[11] Katiyar S, Srinivasan S, Jain RK. Hypocalcemia leading to difficult airway in sepsis. J Anaesth Clin Pharmacol 2011;27:123–4.