Case Report

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Delayed Recovery from Anaesthesia in a Patient with Optimised Hypothyroidism and Incidental Hypokalemia

Anaesthesia Section

PREETI MORE[1], VANDANA V LAHERI[2], TEJASI WAIGANKAR[3], CHARCHILL WAGH[4]

ABSTRACT

Delayed recovery/awakening/ emergence can occur under anaesthesia and is multifactorial, could be drug or non drug related. Similarly, we report a case of delayed recovery in a 68-year-old patient, for laparoscopic cholecystectomy, a known case of hypertension, bronchial asthma and hypothyroidism. Preoperatively, she was optimised for her medical disorders; however, she had delayed recovery from general anaesthesia. The delayed recovery, often, would be expected in a case of hypothyroidism, however in our patient it was found to be associated with inadvertent hypokalemia

Keywords: Bronchial asthma, Delayed emergence, Hypertension, Hypokalemia

CASE REPORT

A 68-year-old, 70 kg female patient, a known case of hypertension, bronchial asthma and hypothyroidism, was posted for laparoscopic cholecystectomy. For hypertension and bronchial asthama, since 10 y, she was receiving tablet Amlodepin 5 mg and tablet Coversyl plus (a combination of Angiotensin Converting Enzyme Inhibitor Perindropil 5mg and diuretic Indapamide 1.25mg) and Salbutamol, Ipratropium and Budesonide as Metered Dose Inhaler (MDI) three times daily during the attacks. For hypothyroidism, since four years she was on tablet L-thyroxine 25 mg once daily.

Her hemogram, renal and liver function tests, serum electrolytes (Serum potassium – 3.5 mmol/l) and blood sugar levels were within normal limits. Thyroid function tests showed T3 and T4 within normal limits, however, TSH value was slightly higher (i.e., 6.04 IU/L against the normal value of 0.3- 5.5 IU/L). X-Ray chest and electrocardiogram were normal. On examination her pulse rate was 102/min, BP was 140/84mm of Hg, and Respiratory rate 14/min, breath sounds and CVS was normal. Preoperative optimization with salbutamol nebulisation done, morning dose of anti-hypertensives and L-thyroxine were taken. Premedication with intravenous Dexamethasone 4 mg, Deriphylline (combination of theophylline and etophylline) 2ml, Glycopyrrolate 0.2 mg, Midazolam 1 mg and Ondansetron 4 mg. Routine monitoring with electrocardiogram, blood pressure, pulse oximetry and capnography with urine output was done. After adequate pre-oxygenation, induction was done with IV Propofol 100 mg and Fentanyl 80 mcg followed by intubation with IV Succinylcholine 100 mg and maintained with Nitrous oxide (65%) in oxygen and end-tidal Isoflurane (1%) using Vecuronium (total dose 7mg) for controlled mechanical ventilation. Normothermia was maintained.

Intraoperative vitals parameters maintained, surgery lasted for 2 h and 30 min. Patient received 1.5 lt of crystalloids (lactated Ringer's solution). Her urine output was 200 ml. For postoperative analgesia IV Paracetamol 700 mg and port- site infiltration with 0.5% bupivacaine was given.

Reversal with IV Neostigmine 3 mg along with Glycopyrrolate 0.4 mg was done and patient had good breathing attempts with acceptable tidal volume; but overall muscle tone, especially the "head-lift" was poor and there was very poor response to verbal commands. Breathing attempts of the patient were assisted and observed.

Blood glucose level and body temperature was recorded to rule out hypoglycemia/hyperglycemia and hypothermia, the most common causes of delayed recovery, were normal. IV Hydrocortisone 100 mg was given for airway hyper reactivity to endotracheal tube as she was a known asthmatic. Also, muscle tone was poor so IV Calcium Gluconate 10ml was given slowly over 10 minutes and IV Frusemide 10 mg was given so that active metabolites of the drugs get excreted. Patient was on oxygen through endotracheal tube and assisted spontaneous ventilation

One hour after the reversal there was no change in clinical condition. Oxygenation was continued and spontaneous ventilation was assisted so as to maintain $EtCO_2$ within normal range. Arterial blood gases (ABG) and electrolytes were estimated to rule out acid/base and electrolyte disturbances. The results were: pH – 7.33; PO_2 – 417 mm Hg; PCO_2 – 48.2 mm Hg; SaO_2 – 99.9%; HCO_3 – 25.3 mmol/L; Na – 137.9 mmol/L; K – 2.98 mmol/L and Cl – 100.7 mmol/L. Since potassium was low, we started KCl infusion 10 ml (20mEq) in 500ml NS, over a period of 30 min with ECG monitoring.

Two hours later, she was responsive to verbal commands, but had poor muscle tone. ABG and electrolytes were repeated. This time PCO_2 was 44.7 mm Hg and potassium was 3.9 mmol/L. Since nothing abnormal was found, we continued oxygenation through endotracheal tube with assisted spontaneous ventilation so as to maintain EtCO2 within normal range.

Two and a half hours later, the patient showed adequate muscle tone and power. Vitals were stable. Patient was extubated and shifted to High Dependency Care Unit with supplemental oxygen and observed for two hours and finally shifted to general ward.

DISCUSSION

Delayed awakening/ emergence/ recovery of varying degrees is not uncommon after anaesthesia, and may have a number of different causes, individual or combined, which may be both drug or non - drug related [1-4].

Drug related factors like overdose, duration and type of anaesthetic given, potentiation by other drugs, prolonged neuromuscular blockade (as seen in acidosis, renal failure) is known [5]. Induction agents, inhalational anaesthetics, narcotic opioids and sedatives used for general anaesthesia can affect the recovery when their action gets prolonged (drug related factors). This can be seen when the metabolism, excretion, redistribution and/or elimination of the drugs are affected due to age, renal, hepatic, metabolic diseases or cumulative effects due to prolonged surgery. Drug interactions

with other medications like central nervous system depressants, narcotic analgesic, alcohol and residual neuromuscular blockade can also affect the recovery time.

Non- drug related causes like metabolic, neurological and respiratory failure can affect recovery from anaesthesia. Metabolic causes like hypoglycaemia, severe hyperglycaemia, hypothyroidism, hepatic and renal diseases, acid –base and electrolyte imbalances [1,4] and hypothermia can be related to delayed recovery.

Neurological complications like cerebral hypoxia, intracerebral events like haemorrhage, embolism or thrombosis is known cause of delayed recovery.

Our patient was elderly as well as minimally hypothyroid, hence, chance of delayed recovery due to drug related factors was a possibility. However, IV Midazolam 1 mg, Propofol 100 mg and Fentanyl 80 mcg given during induction and 7mg Vecuronium with 1% end-tidal Isoflurane for maintenance of anaesthesia in a surgical procedure lasting two and a half hours in a 70kg female cannot be considered as over-dosages for her. IV Furosemide 10 mg was given for excretion of active metabolites of the drugs. Also, IV Calcium Gluconate 10ml (10%) was given slowly over 10 min to improve the muscle tone.

We ruled out hypothermia as patient was adequately kept warm and temperature was 38^0 and hypoglycemia/hyperglycemia by estimation of blood sugar levels. She was on treatment for hypothyroidism and was optimised with L-thyroxine. Keeping in mind the TSH levels of our patient, there is a possibility that hypothyroidism could have been the cause of her delayed awakening. She was not suffering from renal or hepatic disease.

Respiratory factors as seen in patients with chronic obstructive pulmonary disease who do not breathe effectively during or after anaesthesia may become hypercarbic (raised CO_2) to a level that may produce sedation or even unconsciousness, leading to prolonged recovery [5]

Our patient was a known case of Bronchial Asthama, controlled with antiasthamatics and intraoperatively respiration was mechanically controlled with $EtCO_2$ monitoring to prevent hypercarbia which could prolong the recovery time. Hence, ABG and $EtCO_2$ monitoring is required. A 0.1-unit decrease in arterial Ph, as produced by a 10 mmHg increase of the $PaCO_2$, can increase plasma K concentrations by about 0.5mEq/L [4]. Because of the effect of hypokalemia on skeletal muscles there is a possibility of prolonged action of muscle relaxants, which can be a cause of delayed recovery.

We estimated her blood gases and electrolytes to rule out acid-base and electrolyte imbalance. Blood gases were within normal limits, however, serum potassium was low, 2.98 mmol/L. We gave KCL 10 ml (20mEq) in 500ml NS infusion over a period of 30 minutes and repeat serum potassium levels increased to 3.9 mmol/L.

Hypokalemia [1,4] is known to occur with surgical stress and β2--adrenergic agonists [6]. Our patient was receiving β2-agonists. In addition, we gave calcium gluconate, which could reduce serum potassium level. Factors like hyperventilation, respiratory alkalosis, diuretics, administration of calcium, sodium bicarbonate, glucose and/ or Insulin without K supplementation will reduce serum K levels [4, 5].

Potassium levels should be measured frequently if repletion is ongoing or changes due to drug administration or ventilation are expected. Alkalosis also depresses ventilation leading to delayed recovery from anaesthesia.

Treatment for acute hypokalemia is 20 mEq of K administered over 30-40 min and repeated as needed under ECG monitoring. In our patient, we started KCl infusion of 10 ml(20 mEq)in 500 ml NS over a period of 30 min with strict ECG monitoring after which K value increased to 3.9mmol/L from 2.98mmol/L.

An unusual cause of delayed awakening has been reported after acute phenytoin administration due to alteration of the pharmacodynamics and pharmacokinetics of rocuronium in chronically ill patients [7].

A case of subclinical hypothyroidism being one of the probable causes of hypothermia and delayed recovery after exclusion of other causes [8] has been reported.

Hence, the probable cause of delayed recovery in our elderly patient was due to hypothyroidism as well as associated incidental hypokalemia. Hypokalemia, though not present preoperatively, could have been due to peri-operative administration of calcium gluconate and β2-agonists.

CONCLUSION

The causes of delayed recovery are multifactorial. Drug or non drug related factors like metabolic, neurological and respiratory failure could affect recovery following anaesthesia. Common causes are electrolyte imbalances, hypothermia, hypoglycaemia, hyperglycaemia and also on the type of anaesthesia received, duration of surgical procedure and therefore the anaesthesia time, individual patient's response to the anaesthetic drugs, patients age and general condition and associated co-morbidities. In our patient, the probable cause of delayed recovery was incidental hypokalemia, probably as a result of β2-agonists therapy which returned to normal values after replacement with KCL infusion. Other factors like old age, associated with co-mordid conditions like hypothyroidism, hypertension, bronchial asthama could be the contributing factors for delayed recovery from general anaesthesia.

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PARTICULARS OF CONTRIBUTORS:
1. Associate Professor, Department of Anaesthesiology, ESI PGI-MSR, MGM Hospital, Parel, Mumbai-12, Maharashtra, India.
2. Professor, Department of Anaesthesiology, ESI PGI-MSR, MGM Hospital, Parel, Mumbai-12, Maharashtra, India.
3. Senior Resident, Department of Anaesthesiology, ESI PGI-MSR, MGM Hospital, Parel, Mumbai-12, Maharashtra, India.
4. Junior Resident, Department of Anaesthesiology, ESI PGI-MSR, MGM Hospital, Parel, Mumbai-12, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Preeti More,
Dr. S.S.Rao Road, ESI-PGIMSR, MGM Hospital, Parel, Mumbai-12, Maharashtra, India.
E-mai: preetipreety@hotmail.com

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