

Difficult airway leading to carbon dioxide narcosis in a case of fixed cervical spine

Rahul Yadav, Mihir P. Pandia, Parmod K. Bithal, Sachidanand J. Bharati, Indu Kapoor

Abstract

Inability to secure the airway of a patient after induction of anaesthesia may lead to serious consequences including permanent brain damage and even death. Hypoxia is quite common in difficult intubations especially when it is difficult to ventilate the patient. However, carbon dioxide retention severe enough to cause carbon dioxide narcosis and delayed recovery is a rare occurrence. Here, we report a case of a craniovertebral junction anomaly where inadequate ventilation after induction of anaesthesia resulted in carbon dioxide narcosis and delayed awakening. A 54-year-old, American Society of Anesthesiologists II female patient weighing 70 kg with a diagnosis of craniovertebral junction was scheduled for implant removal for dislodged occipital screw. Fiberoptic intubation was attempted after induction of anaesthesia and muscle paralysis. Even after multiple attempts, intubation could not be done and ventilation by face mask became difficult. Though oxygen saturation could be maintained with the insertion of a laryngeal mask airway (LMA), ventilation was not adequate. The patient remained unresponsive long after discontinuation of anaesthetic agent and reversal of muscle paralysis. Subsequent blood gas analysis showed severe carbon dioxide retention and respiratory acidosis. Patient was given assist control mechanical ventilation through LMA. LMA was removed after improvement in sensorium and the blood gas picture.

Key words: Carbon dioxide, delayed recovery, difficult airway, narcosis, respiratory acidosis

INTRODUCTION

Inability to secure the airway of a patient after induction of anaesthesia may lead to serious complications including permanent brain damage and even death. Inadequate ventilation during airway management causes both hypoxia and hypercarbia. Hypercarbia is known to cause alterations in the level of consciousness ranging from anxiety to coma, depending on the rapidity and severity of development.^[1-4] Here, we report a case of a craniovertebral junction anomaly where inadequate

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ventilation after induction of anaesthesia resulted in carbon dioxide narcosis and delayed awakening.

CASE REPORT

A 54-year-old, American Society of Anesthesiologists II female patient weighing 70 kg with a diagnosis of craniovertebral junction anomaly was scheduled for implant removal for dislodged occipital screw. She had undergone transoral odontoidectomy and posterior fixation with titanium polyaxial lateral mass screws for nonreducible atlanto-axial dislocation with basilar invagination, 3 years ago at our institute. During her previous surgery, fiberoptic tracheal intubation using a 7.0 mm cuffed orotracheal tube (was done) under general anaesthesia. The intraoperative and postoperative periods were uneventful.

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Except for bilateral lower limb weakness (grade 4/5), the patient had no other systemic medical illness. Magnetic resonance imaging scan of craniovertebral junction revealed occipitocervical fusion with the implant *in situ* and loose occipital screws. Preoperative investigations including electrocardiogram (ECG) and chest X-ray were normal. Her airway assessment showed a Mallampati grade 4 views with restricted neck movements. Since she refused to undergo awake fiberoptic intubation, fiberoptic intubation under general anaesthesia was planned. She was premedicated with glycopyrrolate 0.2 mg intramuscularly, 1 h before surgery. After attaching routine monitors (pulse oximeter, ECG, noninvasive blood pressure) and preoxygenation for 3 min, anaesthesia was induced with fentanyl 150 µg and propofol 150 mg. After ensuring satisfactory mask ventilation, muscle relaxation was achieved with 70 mg rocuronium. Orotracheal intubation with size 7.0 mm tube with fiberoptic laryngoscopy was attempted after 2 min of rocuronium administration. Though the glottic opening could be visualised, tracheal intubation was not successful even after repeated attempts by experienced anaesthesiologists. It was realised that because of the large size of the tongue, the oropharyngeal space was small and obstructing the passage of the tube. In between the attempts of fiberoptic laryngoscopy, bag, and mask ventilation were successful. ETCO_2 values remained between 30 mmHg and 35 mmHg during mask ventilation. After 5 attempts of fiberoptic intubation, mask ventilation started becoming difficult. In spite of the best fitting mask, jaw thrust, use of both hands for holding the mask and insertion of Guedel's airway, ventilation was inadequate with poor chest expansion. Despite unsatisfactory mask ventilation, however, the patient maintained her SpO_2 at 90–91%. During this period the patient was ventilated with 100% oxygen in sevoflurane 2–3% inspired concentration. A size 4 laryngeal mask airway (LMA) was inserted which improved lung ventilation to a certain extent and increased SpO_2 95%, but the chest expansion was not very good. We did not get a stable capnogram waveform and the ETCO_2 value fluctuated between 20 mmHg and 40 mmHg. To avoid awareness, intermittent boluses of 20 mg propofol were given. In view of a cannot intubate and difficult to ventilate situation, it was decided to defer the surgery and awaken the patient.

Muscle relaxant was not repeated after the initial intubating dose. By then 90 min had elapsed since the administration of rocuronium and the patient was showing sign of the return of spontaneous respiratory efforts. The residual effect of muscle relaxant was reversed with administration of neostigmine and glycopyrrolate. Patient's spontaneous respiratory efforts returned to normal. However, she was not responding to verbal commands. The patient remained unresponsive

even though last bolus of propofol was administered 30 min ago, and the monitor showed zero end-tidal expired tension of sevoflurane. When the patient started to open eyes to a painful stimulus, her pupils were normal in size and she was reacting to light. Train of four monitoring revealed all four twitches of equal height. To antagonize the unlikely persistent action of fentanyl, naloxone 1 mg was given, without any benefit. At this stage, an arterial blood gas analysis (ABG) showed severe respiratory acidosis with hypercarbia (pH 6.923, PaCO_2 154 mmHg, PaO_2 101 mmHg). Repeat ABG done after 30 min, had improvement in respiratory acidosis (pH 7.042, PaCO_2 101 mmHg, PaO_2 208 mmHg). Patient was shifted to ICU with LMA *in situ* and kept on assist control mechanical ventilation. Significant improvement in sensorium and blood gas picture (pH 7.32, PaCO_2 50 mmHg, PaO_2 251 mmHg) was seen after 30 min of assist-control ventilation. LMA was removed over the next 15 min when the patient became fully awake.

She underwent the surgical procedure 5 days later and on this occasion awake fiberoptic intubation was done using a 7.0 mm cuffed endotracheal tube (oral). The patient was counseled and she gave her consent for awake fiberoptic aided intubation. The intra operative and subsequent postoperative course remained uneventful and the patient was discharged home on the 4th postoperative day.

DISCUSSION

Delayed recovery from anaesthesia in our patient occurred due to carbon dioxide narcosis resulting from an inadequate mask ventilation during the airway management. The anaesthetic agents had been stopped long before the attempt was made to awaken the patient from anaesthesia. There was no electrolyte abnormality and patient had no other systemic illness, which could have contributed in delayed awakening. Although the patient's temperature was on the lower side (35°C), the hypothermia was only mild which would not interfere with recovery from anaesthesia. The patient had received only one bolus of 150 mcg fentanyl at the time of induction, which was unlikely cause of delay in recovery. Moreover, naloxone administration did not speed up recovery. Inadequate reversal of residual muscle paralysis was also ruled out with the presence of all four twitches of equal height on the TOF monitor. The patient developed CO_2 narcosis because of inadequate mask ventilation compounded by periods of apnoea while attempting fiberoptic laryngoscopy. It is recommended to administer suxamethonium in the presence of anticipated difficult airway. However, the presence of preexisting muscle weakness deterred us from using suxamethonium. Though we ensured the ease

of mask ventilation before giving rocuronium, ventilation became difficult progressively because of the pharyngeal trauma resulting from airway insertion and repeated fiberoptic laryngoscopy. Pooling of oral secretions and blood also aggravated the difficulty in ventilation. Even though oxygenation was maintained with ventilation using LMA, ventilation was not adequate. Distention of stomach due to gastric insufflations during the difficult mask ventilation and subsequent tenting of the diaphragm also hampered the excursion of chest wall further reducing washout of carbon dioxide. The oxygen saturation of the patient was maintained satisfactorily in spite of poor chest excursions. We assume that the absence of any pulmonary pathology and ventilation with 100% oxygen prevented hypoxaemia during the phase of inadequate ventilation.

Retention of carbon dioxide due to inadequate ventilation is commonly seen in the perioperative setting, but severe hypercarbia resulting from difficult intubation causing carbon dioxide narcosis and stuporous or comatose condition is a rare event in the perioperative settings. Primary carbon dioxide narcosis severe enough to alter consciousness level occurs when the PaCO₂ level exceeds 100 mmHg.^[5] Moderate elevation of PaCO₂ commonly causes anxiety, agitation, and signs of increased sympathetic nervous system activity.^[5]

Yoshida *et al.* have reported a case of occurrence of sudden EEG depression and low bispectral index (BIS) value due to hypercarbia in a patient who was undergoing laparoscopic surgery.^[6] In case of inadvertent injection of a muscle relaxant before induction of anaesthesia, a 45-year-female patient developed carbon dioxide narcosis and had a BIS value between 35 and 40. The PaCO₂ of the patient was 130 mmHg.^[7]

Our case emphasises that maintenance of oxygen saturation during difficult intubation and difficult mask ventilation may give a false assurance of adequate ventilation, more so if 100% oxygen is being

administered. Oxygen saturation of the patient may not fall in the initial stage if the residual functional capacity of the patient is good and decreased consumption of oxygen under general anaesthesia. ABG analysis should be checked to rule out severe hypercarbia during prolonged periods of difficult mask ventilation, especially if the patient is unresponsive to commands after ruling out other causes. Hypercarbia and carbon dioxide narcosis should always be suspected as a possible cause of delayed awakening after prolonged difficult mask ventilation.

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Conflicts of interest

There are no conflicts of interest.

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