Kiran et al. Sri Lankan Journal of Anaesthesiology: 24(1):**35-37**(2016)
DOI: 10.4038/slja.v24i1.8094

Difficult ventilation following administration of low dose fentanyl

***S Kiran*[1*], *V Aggarwal*[2], *N Gupta*[3]**
Senior Professor[1], *Resident*[2], *Department of Anaesthesia and Critical Care, Post Graduate Institute of Medical Sciences, Rohtak, India. Resident*[3], *Department of Anaesthesia, Sriramachandra Medical College, Porur, Chennai, India.*

***Corresponding author*: drshashi64@rediffmail.com**

Low dose midazolam and fentanyl are commonly used to provide conscious sedation during regional anaesthesia. Inability to ventilate because of chest wall rigidity following administration of high dose fentanyl has been described. We report a case where midazolam and fentanyl were used to provide sedation and supplementary analgesia during spinal anaesthesia. The patient became unresponsive and apnoeic and could not be ventilated. Anaesthetic circuit was applied, airway was maintained using face mask 100% oxygen was commenced, and CPAP was initiated. After few seconds, she could be ventilated and her spontaneous respiratory efforts returned. Glottic closure secondary to fentanyl administration was thought to be a plausible explanation.

Keywords: Fentanyl; low dose; upper airway obstruction; inability to ventilate

Introduction:

High dose intravenous fentanyl administration has been associated with chest wall rigidity resulting in ineffective ventilation.[1] Inability to ventilate the patient during conscious sedation after intravenous administration of benzodiazepines and fentanyl has been reported.[2] We describe a case of postpartum tubal ligation who developed apnoea intra operatively under spinal anesthesia following administration of intravenous midazolam and fentanyl and could not be ventilated.

Written consent of the patient was obtained for publication of this case report.

Case Report

A 25yr old female weighing 70kgwas scheduled to undergo tubal ligation under spinal anaesthesia. She had a full term normal vaginal delivery one week before and was in good health. Her preoperative haematological investigations were normal. Under aseptic precautions spinal anaesthesia was administrated in sitting position using 2.5ml of 0.5% hyperbaric bupivacaine. A sensory analgesia up to T_6 level was achieved and surgery commenced. After about 15 minutes, midway through the surgical procedure the patient complained of stretching sensation in the abdomen which was very distressing to her. Her vitals were normal except for her heart rate which was 100 beats per minute. Midazolam 1mg i.v. was then administrated, followed by 50mcg of fentanyl. Her heart rate settled to 75 beats per minute but still she was uncomfortable. After few minutes another 50mcg of fentanyl was given intravenously. She immediately became quiet. Oxygen via facemask was already being administrated to her. Within seconds oxygen saturation fell from 100% to 85%. She became unresponsive. Her pulse rate was 80 beats per minute and blood pressure was 110/70 mmHg. Oxygen mask was immediately replaced with 100% O_2 via Bain's circuit. It was noticed that she was apnoeic. Her chest was not moving and bilateral breath sounds were absent. Continuous positive airway pressure ventilation was commenced but she could not be ventilated. She started further desaturating. Firm pressure was applied in laryngospasm notch. Within seconds she became responsive and we were able to ventilate her. She started breathing normally shortly after. The whole episode took around one minute starting from administration of midazolam and fentanyl. Her lowest O_2 saturation recorded was 80% for a transient period. She remained haemodynamically stable during this period. The remaining intraoperative period remained uneventful. The surgical procedure was completed in 45mins. She was observed in PACU for 4 hours and was discharged home the following day. She did not

have any recall about the event that happened intraoperatively.

Discussion

A combination of midazolam and fentanyl is commonly used for conscious sedation in patients undergoing surgery under regional anaesthesia. In literature there are case series reports, that high dose of fentanyl used at induction of anaesthesia cause severe problems with maintenance of ventilation by bag and mask. In all these studies there was inability to ventilate and airway patency was compromised, following high dose of fentanyl until muscle relaxation was provided.[3,4,5] The dose of fentanyl in these ranged from 15-19mcg/kg. It was also reported that if 50mcg/kg fentanyl is given over 60 seconds there is 100% incidence of rigidity.[4]

In our case we used low dose of fentanyl along with midazolam, therefore chest wall rigidity was not expected. We did not have the opportunity to assess rigidity in other parts of body such as upper limbs as patient was rapidly desaturating and it was deemed prudent to ventilate her as the first priority. There have been two case reports in literature describing chest wall rigidity with a single dose of fentanyl. In one patient fentanyl was administered over 12 minutes,[6]in other case speed of injection was not described but naloxone administration was required to reverse the effect of fentanyl,[2] whereas in our case naloxone was not required. May be it was not a chest wall rigidity; but an upper airway obstruction because of glottis closure secondary to fentanyl administration.

Scamman investigated whether the cause of inability to maintain bag and mask ventilation after fentanyl administration was above tracheal level, specifically at glottis.[7] High dose fentanyl was administrated in patients who had normal upper airway and comparison regarding pulmonary compliance was done with patients who were already tracheostomised. He found that there was very small decrease in pulmonary compliance following fentanyl administration in tracheostomised patients which was insufficient to prevent manual ventilation. On the basis of this data, the author concluded that it is unlikely that rigidity of chest wall is responsible for inability to ventilate. He indicated that fentanyl causes glottic rigidity resulting in its partial or complete closure with subsequent airway obstruction. In an experimental study it was found that benzodiazepines may decrease genioglossus activity increasing upper airway resistance.[8]In another experimental study it was found that midazolam depresses tone of pharyngeal muscles.[9] This is also evident as we see in our clinical practice that patients start snoring after administration of benzodiazepines. The effect may be exaggerated by the use of concomitant administration of narcotics.

Same explanation can be offered for our case, although we used only small dose of fentanyl. Since our patient did not require naloxone as in one report; this also indicates that rather than chest wall rigidity, glottic closure is more prudent explanation, more so because when pressure was applied in laryangospasm notch, glottis opened up and we were able to ventilate the patient. However in literature there is anecdotal evidence regarding efficacy of this manoever.[10]

In summary our case report highlights that every clinician should be aware of total upper airway obstruction which can occur with small dose of fentanyl commonly used along with midazolam for conscious sedation during regional anaesthesia.

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