Case Report

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Postoperative chest wall rigidity and myoclonus following low dose intravenous fentanyl: a case report

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ABSTRACT

Opioid induced muscle rigidity and myoclonus is a frequently described complication which occurs more commonly with large doses and rapid administration of the drugs and is observed at the time of induction. In the post-operative period this complication is reported in patients undergoing cardiac surgery, where large dosages of fentanyl or analogs are employed. Rigidity and myoclonus in the postoperative period with analgesic doses of opioids is extremely rare. We report a case where a life threatening chest wall rigidity and myoclonus occurred in a patient where fentanyl was used as an intraoperative analgesic in a relatively low dose. Though the mechanism of occurrence of this phenomenon remains unclear but the fact that the rigidity was terminated with naloxone confirms that it was fentanyl induced.

Key words: Chest wall rigidity, fentanyl, myoclonus, naloxone, post-operative

INTRODUCTION

Fentanyl, a synthetic lipophilic opioid is a commonly used analgesic during surgery owing to its properties of providing intense analgesia, attenuation of the sympathetic response and shorter duration of action. Chest wall rigidity is a well-known complication associated with opioid administration and has been frequently reported in literature[1–3]. Occurrence of myoclonic movements has also been reported with the use of opioids[4,5]. However, both these complications are reported to be associated with large doses and rapid administration of fentanyl, usually at the time of induction[2,3,6]. There are reports of chest wall rigidity occurring in postoperative period, but, in cases where high doses of fentanyl were used for induction- especially in patients undergoing cardiovascular surgeries[7]. We are reporting a rare case- where a patient who was given a low dose of fentanyl at the time of induction developed myoclonus and rigidity of chest after reversal of anaesthesia and extubation. The complication was effectively managed with intravenous naloxone.

CASE REPORT

A 56-year-old female patient underwent elective laparoscopic cholecystectomy under general anaesthesia. She was a known hypertensive for past 6 years, on treatment with tablet Telmisartan 40 mg OD. She gave a history of an uneventful gynaecological surgery under spinal anesthesia 10 years back. After initiation of intravenous and monitoring lines, patient was given injection midazolam 2 mg, injection glycopyrrolate 0.2 mg and injection ondensetron 4mg intravenously. Induction of anesthesia was done with propofol 2.5 mg/kg after pre-oxygenation for three minutes. Just before induction, injection fentanyl

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100 mcg was injected and no muscle rigidity was noticed at that time as bag mask ventilation was easily done. Injection succinylcholine 2 mg/kg was given to facilitate intubation. Maintenance of anesthesia was done with N_2O:O_2 (60:40) and isoflurane (0.5-1 MAC). Injection atracurium was used for surgical relaxation. Surgery lasted for 90minutes; no additional dose of fentanyl was given during surgery. Intravenous paracetamol 1000 mg was given over a period of 30 minutes and a diclofenac suppository of 50 mg was given for postoperative pain. After completion of surgery, isoflurane and N_2O were stopped and patient was taken on 100% oxygen. The patient was normothermic, normocarbic and hemodynamically stable at that time. Muscle relaxation was reversed with neostigmine 2.5 mg, and glycopyrrolate 0.5 mg, when the capnograph showed return of spontaneous respiratory efforts and patient opened her eyes on verbal commands. Upper airway was suctioned and cough reflex was found to be good and trachea was extubated. After extubation, patient opened her eyes and made purposeful movements on verbal commands. After a few minutes,the patient had a brief episode of myoclonic movement of upper and lower limbs and she became unresponsive, her blood pressure increased significantly (from 130/80 mm Hg to 193/110 mmHg). As the ventilation got depressed, bag mask ventilation was tried but it became difficult even with four handed technique. Due to rapid rise in end tidal carbon dioxide and de-saturation of the patient, it was decided to re-intubate the trachea and start mechanical ventilation. Suspecting an inadequate reversal of the relaxant, neuromuscular monitor was attached; and though it showed a normal trainof-four twitch and sustained tetanus on 50 Hz stimulation-an additional dose of neostigmine 1 mg and glycopyrrolate 0.2 mg was given. An arterial blood gas analysis was done which showed respiratory acidosis. Mechanical ventilation with 100% oxygen improved the saturation and brought the end tidal carbon-dioxide to normal. After 5-7 minutes, the patient opened her eyes and moved her limbs purposefully but was incapable of breathing spontaneously. She started following verbal instructions, but was not able to generate effective ventilation despite great efforts. Suspecting it to be chest rigidity, injection naloxone- 4 mcg was administered intravenously slowly and within 2 minutes the patient's ventilation improved.She was able to generate adequate tidal volume and extubation could be done. The blood pressure also gradually returned to normal. Patient was shifted to post anesthesia care unit and had uneventful recovery and discharge. On enquiring in the postoperative period- patient recalled that though she was able to comprehend what was asked to her, she was not able to breathe and was feeling the tightness of chest.

DISCUSSION

Opioid induced chest rigidity-also known as wooden chest syndrome is a known but uncommon adverse effect and its true incidence is unknown[2,3]. Myoclonus resulting from fentanyl analogs which may resemble generalized seizures is not associated with electroencephalographic evidence of seizure activity and its mechanism of origin is probably similar to the mechanism of opioid-related tonic rigidity[4,5]. The occurrence of both chest rigidity and myoclonus is dependent on the dose and rate of administration of the drug and is more commonly seen at the time of induction. Chest wall rigidity in the postoperative period occurs due to a secondary rise in plasma concentration due to mobilization of opioid from tissue compartments (muscle, fat etc) when high doses of fentanyl or fenatnyl analogs are used intra-operatively[7]. In the present case fentanyl was used as intra-operative analgesic in a relatively low dose. Though there are anumber of cases reported, where muscle rigidity was experienced with lower doses of opioids, most of them occur at the time of induction within few minutes of administration of the drug[3–10]. Postoperative rigidity with the use of fentanyl and analogs in analgesic doses is rare. We came across three reports of post-operative chest wall rigidity when opiods were used in low doses for patients undergoing general surgery[11–13]. Only one of these reports describes the occurrence of myoclonus along with chest rigidity like we have experienced in our case[11].

It has been suggested that opiod related myoclonus is closely associated with opoid related rigidity[4,5]. Smith et al. reported "seizure-like" movements in association with rigidity in forty-sevenpatients receiving large doses of fentanyl, sufentanil, or alfentanilwith normal simultaneous EEG recordings[14].

Presumably,rigidity is relatively unusual in the postoperative period with low doses because fentanyl concentrations are typically belowthe level required to produce rigidity. However, in a study conducted by Streisand et al on twelve healthy volunteers- it was demonstrated that rigidity and unconsciousness occurred in 50% of the subjects on an infusion of fentanyl 150 mcg/min and no difference in the plasma concentrations were detected between subjects who developed rigidity and those who did not[15].

Paucity of information exists with regard to the underlying mechanism(s) of development of opioid associated chest rigidity. Vankova et al., demonstrated that opioid-induced muscular rigidity is primarily due the activation of central mu receptors[16]. Another study by Soares et al reported

that central dopaminergic pathway might be partially responsible for the opioid-induced chest wall rigidity[17]. Lui et al in an experimental study on rats suggested the possible Involvement of locus coeruleus of pons and noradrenergic neurotransmission in fentanyl-induced muscular rigidity[18]. Some patients developed rigidity or myoclonus with low dose of fentanyl after emergence from anesthesia, as long as 5 hours after surgery, makes the understanding more obscure[12].

CONCLUSION

Fentanyl induced chest wall rigidity can be life threatening if not recognized and treated promptly. It may occur postoperatively within minutes to hours after reversal and extubation. It can prove to be perilous if occurs when the patient has been transferred to the surgical ward and is not closely monitored. Although no clear mechanism has been described in literature this delayed effect is thought to be due to the secondary rise in plasma concentration of fentanyl due to reentry of fentanyl into plasma from tissue deposits. Management includes prompt action to prevent de-saturation and reversal of the effects of fentanyl with naloxone.

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