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**Case Report**

# Low Dose Fentanyl Infusion Used for Sedation during Mechanical Ventilation Leading to Asynchronous Ventilation - A Case Report

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**Abstract**
*Fentanyl induced chest wall rigidity is more commonly observed on high bolus dose of intravenous fentanyl. We report here a case of asynchronous ventilation on low dose fentanyl infusion used for sedation in intensive care unit. Ventilation improved only with neuromuscular relaxation and on cessation of fentanyl. We suggest to have high degree of suspicion for this potentially life threatening side effect of fentanyl on use of this drug.*
**Keywords:** *Fentanyl; Mechanical ventilation; chest wall rigidity*
**Key Messages:** *Low dose intravenous fentanyl infusion is capable of producing chest wall rigidity.*

## Introduction

Fentanyl, a short acting opioid is one of the most common drug used in intensive care unit (ICU) for sedation and analgesia.[1] It is used mostly in low infusion rate after initial bolus dose. Chest wall rigidity (CWR) is a known complication of fentanyl but mostly with high bolus dose. [2] This CWR makes ventilation very difficult in these patients. Here we report a case of asynchronous difficult ventilation with low dose fentanyl infusion in ICU. A 25-year-old woman admitted to our surgical intensive care unit (SICU) immediately following a lower section cesarean section (LSCS) under general anaesthesia.

## Case Report

Approval for case report submission has been taken from the patient as a signed written informed consent. A 25 year old, primigravida at full term weighing 56 kg presented with active labour pain. She was unregistered case referred to our centre from peripheral district hospital. During her earlier routine antenatal check-up, she was found to have mid –diastolic murmur with loud $S_1$ at the apical area. She was investigated thoroughly and confirmed to have mitral stenosis. Her ECG showed bilateral atrial enlargement with right bundle branch block and right ventricular hypertrophy.

Echocardiography showed severely stenosed mitral valve (0.7 $m^2$), peak/mean pressure gradient across the valve 30/18 mm of Hg, grade II aortic regurgitation with severe pulmonary hypertension (PAP= 98 mm of Hg) with left ventricular ejection fraction of 35% and intact intratrial septum and intraventricular septum.

The patient was advised balloon mitral valvotomy by the cardiologist in 2nd trimester of pregnancy. She was on tab digoxin (0.25mg), tab lasix (40mg) and injection penidura every 21 days. She did not followed the advice of mitral valvotomy, but continued her medication. At 38 weeks she developed labour pain and had dyspnoea on rest accompanied with dry cough. She was referred to our centre and was taken for emergency LSCS.

On preanaesthetic examination done on the operation table, her pulse was 90/min, regular in rate and rhythm, blood pressure 150/90 mm Hg with engorged jugular vein and bilateral pedal oedema. Auscultation revealed mid diastolic murmur grade 5/6 with loud S1, opening snap at the apical area, loud P2 at pulmonary area and bilateral crept at lower lung fields. There was moderate hepatomegaly. Her routine investigations was within normal limit except liver function test was mildly deranged. Portable bedside echocardiography was done which showed increase in pulmonary artery pressure (PAP= 101 mm of Hg) and increase in mean and peak pressure gradient. It was planned to give her general anaesthesia (GA) followed by elective ventilation in surgical intensive care unit (SICU).

After taking informed written high risk consent, she was taken for LSCS. She was given GA and remained haemodynamically stable throughout the surgery. She was shifted to SICU for elective ventilation. On arrival to the SICU, she was put on fentanyl infusion (1 mcg/kg) and midazolam infusion (0.1mg/kg). After an hour when she was out of neuromuscular blocker her spontaneous breathing on the ventilator became laboured with a very prolonged expiratory phase. Lungs sounds remained vesicular. Chest radiography and electrocardiography revealed no abnormalities, and the patient's cardiac enzyme levels were within normal limits. On synchronized intermittent minute ventilation (TV-450 ml, low PEEP 8 cm H2O, FiO2 0.6, respiratory rate 22), inhalations and exhalations were markedly asynchronous. Chest wall movements were noted to be discordant with the ventilator, and the chest wall rose minimally with ventilator breaths. Also noted were a decrease in lung compliance to 8 L/cm H2O, a decrease in tidal volume to 2.7 mL/kg, an acute rise in airway resistance to 51 cm H2O/L/s, and an increase in end-tidal CO2 to 60 mm Hg. Arterial blood gas measurements indicated worsening respiratory failure (pH 7.24, pCO2 68 mm Hg). No endotracheal obstruction was noted on deep suctioning. Since we had to avoid any hypoxemia and hypercabia in this patient in view of high pulmonary artery pressure we paralyzed the patient with intravenous cisatracurium (0.1 mg/kg). There was marked improvement in the ventilation with arterial blood gas measurements (pH 7.44 and pCO2 40 mm Hg) at $Fi_2$ of 0.5 and PEEP of 10 cmH2O. We started the infusion of the same neuromuscular blocker. Patients lung compliance became good and we planned of weaning after stopping neuromuscular blocker on day 2 in SICU. Every attempt at weaning the patient off midazolam infusion while continued fentanyl infusion was met with periods of respiratory decompensation characterized by low minute ventilation, hypercarbia and breathe holding. Many of these intermittent episodes were associated with elevated blood pressure. On the morning of postoperative day 3, the patient's breath holding episodes became resistant to sedation. A propofol infusion (12.5-25.0 mcg/kg/min) was added to the continuous fentanyl and midazolam infusions. Despite this treatment, her breathing remained laboured; multiple ventilatory modes were attempted with no significant improvement. We reviewed all the drugs being given to the patients.

We decided to stop fentanyl infusion considering possibility of fentanyl induced chest wall rigidity since her ventilatory parameters used to improve

on paralysis. After stopping fentanyl infusion she improved and was weaned successfully from the ventilator.

**Discussion**

Opioid induced chest wall rigidity was first described in 1953 by Hamilton and Cullen. [3] This is seen more commonly during general anaesthesia, when high dose opioid is given.[4,5] There is difficulty with mask ventilation, respiratory arrest, and a rigid chest wall noted in this condition. Extremes of age, rapid injection and high dose of the drug increases the susceptibility of CWR.[6] It is a clinical diagnosis and commonly missed in non-susceptible patients especially on low dose fentanyl. We want to emphasize that it can also happen with very low dose of fentanyl infusion, as in our case. There was a similar case report of asynchronous ventilation on low dose fentanyl infusion caused due to CWR development in intensive care unit leading to prolonged stay and tracheostomy of the patient.[7] Apart from this case report there has been several case reports on low dose fentanyl given as intermittent boluses causing CWR.[8,9] The cause of this rigidity is not clearly defined with varied postulation in various studies.[10,11] Thus complete prevention of this complication is not possible. Fentanyl is one of the commonest used opioid for sedation. We therefore suggests to have high index of suspicion for opioid induced CWR in all the patients in background of opioid use in any dose, in any age group of patients.

In our case complication was recognized early and patient was successfully weaned of ventilator after stopping fentanyl.

**Conclusion**

Opioid induced CWR should always be one of the differential diagnosis of asynchronous ventilation when they are used for any indication in any dose.

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