

Fentanyl-Induced Chest Wall Rigidity In a 6-Year-Old Child: A Case Report

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ABSTRACT

INTRODUCTION: Chest wall rigidity is muscle rigidity affecting the thoracic and abdominal muscles following the administration of high doses of opioids including Fentanyl. In our case, we present a rare case of chest wall rigidity induced by fentanyl administration.

CASE PRESENTATION: A 6-year-old male patient presented to the E.N.T. department in the University Teaching Hospital of Butare with a 3-year history of recurrent tonsillitis with obstructive sleep apnea and Grade IV adenoid and grade III tonsils on physical examination. Elective adenotonsillectomy was planned and preoperative induction of general anesthesia with Halothane and Ketamine, Fentanyl and Propofol was done. The child experienced persistent chest rigidity that was immediately treated with IV Naloxone.

CONCLUSION: Fentanyl-induced chest wall rigidity is an uncommon but severe complication, especially in children. Once treated very early, the condition successfully improves.

Keywords: Chest Rigidity, Fentanyl, General Anesthesia, Case Report

INTRODUCTION

Opioid chest wall rigidity has been a well-documented condition since 1953 [1]. It is muscle rigidity particularly affecting the thoracic and abdominal muscles following the administration of high doses of opioids such as fentanyl. This leads to blockage of ventilation, hypoxemia and respiratory acidosis. The exact incidence is not well known, especially in young children. The most known cases are reported in newborns and elderly patients [2,3,4].

We present this case of chest wall rigidity in a 6-year male child after administering a low dose of fentanyl. This case report highlights the need

for clinicians to have a high suspicion of the phenomenon in children, even with a low dose of fentanyl, because once diagnosed and treated very early, the condition improves with good outcomes.

CASE REPORT

A 6-year-old male patient presented to the ENT /OPD department in the University Teaching Hospital of Butare (UTHB/ CHUB) located in Rwanda's southern province, with recurrent tonsillitis, snoring, sleep disturbances and a dry cough. There was a three-year history of chronic recurrent tonsillitis with obstructive sleep apnea,

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and there was no reported chronic disease in the family. His physical examination concluded a grade IV adenoid and grade III tonsils. For case management, adenotonsillectomy was planned.

The Preoperative evaluation was done with conserved general state and weight of 16.7 kg. He was anxious, with normal vital signs: SaO₂ 96%, heart rate 123 beats per minute, clear chest, normal heartbeats, and normal airway evaluation, The American Society of Anesthesiologists (ASA) physical status class I. Laboratory findings were in normal range: hemoglobin 13g/dl, hematocrit 38.7%, platelets 296 x10³/μl and white blood cells 7x 10³/μl.

During induction of general anesthesia with halothane, IV-line placement done with a 22G catheter then IV drugs are given: Ketamine 10mg, Fentanyl 40mcg, and Propofol 100mg (80mg then 20mg) were administered. Uneventful intubation was done with a cuffed endotracheal tube (ETT) 4.5 secured at 16cm and ventilation VCV- PEEP5 TV 150 RR 25 Fio₂ 1.0. Maintenance was established on Halothane MAC 1.0.

Around 10 minutes after the confirmation of uneventful endotracheal intubation, a member of the anesthesia team noted a loss of capnography waveform with the absence of chest movements and increased Peak Inspiratory Pressure (PIP), very difficult manual bagging with too much pressure, and desaturation to 60-70%. Hemodynamic status was conserved. Severe bronchospasm was immediately suspected, an emergency declared, call for help given, and aggressive management started. The team administered deepening anesthesia O₂ 100% with halothane, several doses of sub-cutaneous Epinephrine 200mcg, several doses of albuterol puffs via ETT, Dexamethasone IV 4mg (due to a lack of Hydrocortisone), switched off mechanical ventilation, and used bag-mask for ventilation/ connected to E-cylinder.

After five minutes without any response, a short-acting muscle relaxant was added: IV suxamethonium 20mg. The patient continued to desaturate on oxygen with persistent chest wall rigidity. Therefore, a Fentanyl- induced chest wall rigidity was highly suspected, and the condition was immediately treated with several doses of IV Naloxone 40mcg. Eventually, after 15 minutes, chest movement restarted and oxygen saturation improved.

The patient maintained spontaneous breathing. The ENT team agreed to postpone the case for

stabilization of the patient. Eventually, after one hour, extubation was done, and the patient was kept in the ICU for close monitoring. Intravenous Dexamethasone was prescribed for 48 hours. A chest X-ray was requested to rule out any associated chest pathology, but the chest X-ray was normal. Two days later, the patient was discharged and returned home. Surgery was planned for one week after on April 17, 2019.

General anesthesia without opioids was planned; keeping in mind the Fentanyl-induced chest wall rigidity. The adenotonsillectomy was rescheduled to be performed under general anesthesia without opioids.

The pre-operative evaluation of the patient was uneventful with normal laboratory findings and chest X-ray. General anesthesia induction was performed with O₂/Halothane 3%, use of standard monitoring: NIBP, ECG, SatO₂ and Capnography. Peripheral IV access was performed with a 22GCatheter. Intravenous Ketorolac 9mg, Ketamine 20mg, and Vecuronium 2mg were administered, and there was then uneventful intubation and cuffed ETT 4.5/secured at 16cm. The patient was put on a Ventilator: VCV/TV 150/PEEP 5/FiO₂0.5. He received IV Paracetamol 200mg and the maintenance was done with Isoflurane- MAC 2, fluids were given with Ringer Lactate-Dextrose 2% 60ml/h. Within one hour of surgery, the patient was stable in hemodynamics with a normal sinus rhythm, heart rate and Capnography waves. Extubation was uneventful, and the patient was admitted to the ICU for close monitoring. After a few hours without any clinical issues, the patient was discharged from the ICU to the ENT ward. The child was discharged from the ENT ward around 48 hours post adenotonsillectomy without any reported clinical complication.

DISCUSSION

Fentanyl was synthesized by Janssen Pharmaceutica in the 1950s and 1960s, to produce an opioid analgesic with potent analgesic activity and lesser adverse effects than morphine [5]. Between 1975 and 1981, it was adopted widely as a strong intra-operative analgesic agent with relatively few side effects. In low to moderate bolus doses (3 to 5 μg /kg), it was combined with different intravenous supplements to produce “balanced” anesthesia [6].

Fentanyl has the following side effects: euphoria, confusion, respiratory depression, drowsiness, nausea, visual disturbances, dyskinesia, hallucinations, delirium, analgesia, constipation, narcotic ileus, muscle rigidity, addiction, loss of consciousness, hypotension, coma, and even death [7,8].

In the University Teaching hospital of Butare, we routinely induce general anesthesia with halothane and then top up with intravenous hypnotics such as propofol, and/or ketamine, as well as opioids such as Fentanyl. To achieve this effect, close cardiac and pulse oximeter monitoring is mandatory. This is well tolerated in the majority of cases. The risks of general anesthesia mainly include variability of the heart activity, hemodynamic instability and respiratory depression.

However, fentanyl- induced chest wall rigidity is an uncommon complication, especially during general anesthesia for adenotonsillectomy in children. The most known cases are reported in newborns and elderly patients [2,3,4]. Fentanyl is widely used in the neonatal intensive care unit for sedation and analgesia [9]. In the majority of cases of chest wall rigidity induced by fentanyl use were in neonates, and occurred after high doses of fentanyl used in anesthetic induction [2,10,11,12]. P.L. Bailey et al. reported that high doses of fentanyl could cause neuronal excitation and, rarely, seizure-like activity [10]. For our case, this was not similar; chest wall rigidity occurred after 10 minutes of induction of general anesthesia with a low IV dose of 2.43µg/kg of fentanyl in a 6-year-old child. Other authors reported chest wall rigidity after a lower dose of fentanyl administration in infants [11].

The phenomenon of the chest wall rigidity was also reported in preterm and term infants [2]. Fentanyl plasma concentration decreases slowly in infants. The rapid and cumulative redistribution of fentanyl into fat and muscle depots may cause fentanyl toxicity in neonates [13]. The neonatal liver has lower expression of CYP3A4 responsible for fentanyl metabolism. This is the cause of the longer half-life of fentanyl in neonates and a lower rate of elimination [14,15].

In our case, this risk factor of age cannot contribute to the occurrence of fentanyl-induced chest wall rigidity because the expression of CYP3A7 and CYPA4 in the human liver occurs immediately after birth [15]. For a 6-year - old child, the liver

should be mature enough to metabolize fentanyl. In the previous studies, the other risk factors for the development of chest wall rigidity were a high dose of fentanyl, increased age, critical illness, and antidepressant use [16,17,18]. The average dose of 19 ± 1.9 µg/kg of fentanyl was administered at the rate of 200 µg/ min and chest rigidity occurred in 20 of 21 patients [17]. Phua CK et al. reported its occurrence after 25 µg of IV fentanyl administration for moderate sedation during a routine bronchoscopy in a 55-year-old man with lung cancer and mediastinal lymph nodes [3]. It was reported by Peh WM et al. with high-dose intravenous fentanyl administration for sedation and analgesia in the intensive care unit for management of pneumonia and asthma in an 80-year-old woman [4]. Even a small repeated bolus dose of fentanyl (50 µg once, the total dose of 200-250) was reported to cause chest wall rigidity in adults [19,20]. Our 6-year-old patient was in good condition with normal vital signs and normal laboratory findings, therefore increased age and critical illness were excluded as risk factors of chest wall rigidity induced by fentanyl administration in our case.

This complication of fentanyl was treated in our case by repeated boluses of IV naloxone 40 µg and respiratory support. Naloxone acts as a competitive antagonist at μ , κ , and σ opiate receptors in the central nervous system with a higher affinity for the μ receptor [8].

CONCLUSION

In this case study, we present an uncommon severe complication: chest wall rigidity following administering a low dose of fentanyl in a 6-year-old male child during general anesthesia induction. The experience shared in this case report underlines the consideration of chest wall rigidity as a rare, severe complication of fentanyl use, even with low dose in children during general anesthesia. The recognition, prompt diagnosis and management of this phenomenon are crucial to avoid subsequent severe complications. With a delayed diagnosis, it may lead to death. However, once diagnosed very early, the condition can be treated with success. In our case, a child had chronic recurrent tonsillitis with obstructive sleep apnea. Further study is needed to rule out the role of chronic obstructive sleep apnea in the development of chest wall rigidity induced by fentanyl administration.

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