ORIGINAL RESEARCH PAPER

Anaesthesiology

FENTANYL INDUCED CHEST WALL RIGIDITY IN A THREE YEAR OLD CHILD: A CASE REPORT

KEY WORDS: Fentanyl, Chest wall rigidity, Child

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ABSTRACT

Chest wall rigidity is muscle rigidity affecting the thoracic and abdominal muscles following the administration of high doses of opioids including fentanyl. We are reporting a rare case of chest wall rigidity induced by low dose of fentanyl. A three year-old male patient presented to the paediatrics department of our institute with history of seizure disorder and his MRI brain was planned. On the day of MRI scan, child was induced with IV fentanyl and propofol. ProSeal laryngeal mask airway (PLMA) of size 2 inserted and connected to Bains circuit for ventilation. The child experienced persistent chest rigidity that was immediately treated with IV Naloxone.

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. Majority of cases are reported in newborns and elderly patients[2-4].

We present this case report of chest wall rigidity in a three year male child after administering a low dose of fentanyl. This report highlights the need for clinicians to have a high suspicion of this phenomenon in children, even with a low dose of fentanyl, because once diagnosed and treated very early, the condition improves with good outcome.

CASE REPORT

A three year old male patient presented to the Paediatrics department with complains of seizure disorder and was advised to get MRI scan of brain done. As the child was apprehensive induction with anaesthesia was planned before scan.

Evaluation prior to scan was done. Weight of child was 12 kg. He was anxious, with normal vital signs; SpO2 100%, heart rate 120 beats per minute, clear chest, normal heart sounds and normal airway evaluation. The American Society of Anesthesiologists (ASA) physical status class I. Laboratory findings were in normal range: hemoglobin 13g/dl, hematocrit 39.7%, platelets 326 x103/µl and white blood cells 6x 103/µl. During induction, IV line was secured with a 22G cannula and fentanyl 20 mcg and propofol 30 mg (20 mg followed by 10 mg) were administered. After proper jaw relaxation PLMA of size 2 was inserted and ventilation started with Bains circuit. Bilateral air entry was found equal and adequate. MRI scan was started and finished in 10 minutes.

After completion of MRI scan, around 15 minutes later to induction, we noticed a loss of capnography waveform with the absence of chest movements and very difficult manual bagging with too much pressure. Hemodynamic status and oxygen saturation was conserved. Severe bronchospasm was immediately suspected, an emergency declared and call for help asked. Anaesthesia was deepened with additional doses of propofol and 2 puffs of salbutamol inhaler were given via PLMA. IV hydrocortisone 40 mg was also given immediately. Patient's condition didn't improve. IV succinylcholine 20mg was administerd. The patient continued to desaturate on oxygen with persistent chest wall rigidity. Therefore, Fentanyl induced chest wall rigidity was highly suspected and the condition was immediately treated with several doses of IV Naloxone 20mcg. Eventually, after 15 minutes, chest movement restarted and oxygen saturation improved.

The patient maintained spontaneous breathing. Eventually after one hour, extubation was done and the patient was kept in the recovery room for close monitoring. A chest X-ray was requested to rule out any associated chest pathology, but was found normal.

DISCUSSION

Opoids are commonly used during induction of anaesthesia. However, fentanyl induced chest wall rigidity is an uncommon complication, especially during general anesthesia for a short procedure in children. The most known cases are reported in newborns and elderly patients[2-4]. Fentanyl is widely used in the neonatal intensive care unit for sedation and analgesia[5]. 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[2,6]. *P.L. Bailey et al* reported that high doses of fentanyl could cause neuronal excitation and rarely seizure-like activity[6]. In our case, this was not similar; chest wall rigidity occurred after 15 minutes of induction of general anesthesia with a low IV dose of 2 µg/ kg of fentanyl in a three year-old child. Other authors reported chest wall rigidity after a lower dose of fentanyl administration in infants[7]. The phenomenon of the chest wall rigidity was also reported in preterm and term infants.

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[2]. 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[8,9].

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[9]. For a three 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[10]. *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[11]. Our 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 20 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

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.

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