

Fentanyl Induced Coughing Caused Life-threatening Airway Obstruction in a patient with arteriovenous malformation of tongue and hypopharynx

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Citation

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Abstract

Fentanyl induced coughing (FIC) is common with the incidence ranges from 18 to 65%; however, it has not been perceived as a serious problem. The FIC is not always benign and brief and may be spasmodic and explosive to attract immediate emergency intervention. We report a 12 years old patient scheduled to undergo ultrasound guided needle aspiration cytology of intra-abdominal lymph nodes developed severe spasmodic cough after receiving i.v. fentanyl (50 µg). The FIC lead to massive engorgement of tongue and hypopharynx that caused acute airway obstruction and severe hypoxia. The management and suggestions to prevent such an episode in future are presented.

INTRODUCTION

Opioids have been used for hundreds of years to allay anxiety and decrease pain in surgical patients. They are routinely used as premedicants and analgesics perioperatively. Fentanyl, a synthetic opioid, is commonly administered prior to induction of anaesthesia because of its rapid onset, short duration of action, intense analgesia, cardiovascular stability and no histamine release. However, various studies [1,2,3,4,5,6,7] have found that preinduction bolus dose of fentanyl causes coughing in 18% to 65% of patients. Fentanyl induced cough (FIC) is common but has not been perceived as serious anaesthetic problem. The FIC is not always brief and benign and may be explosive at times that may require immediate intervention. Tweed and Dakin [8] reported a case of explosive, spasmodic coughing after intravenous injection of fentanyl that was severe enough to produce periorbital petichiae and was only relieved after induction of anaesthesia. We report a 12 year old child scheduled to undergo ultrasound guided needle aspiration cytology of intra-abdominal lymph nodes developed severe spasmodic coughing after i.v. fentanyl leading to massive engorgement of tongue and hypopharynx due to pre-existing arteriovenous malformation that caused acute upper airway obstruction and life threatening hypoxia.

CASE REPORT

A 12-yr old boy, height 132 cm, weight 42 Kg, known case

of arteriovenous malformation of tongue and oropharynx was scheduled to undergo ultrasound guided needle biopsy of intra-abdominal lymph nodes of unknown aetiology, under sedation and local anaesthesia (Fig.-1). There was no history of fever, cough or sore throat in last three months. He had no previous history of any surgical procedure or general anaesthesia. There was no history of environmental allergies or penicillin sensitivity. Arterial blood pressure and heart rate were 100/60 mm.Hg and 98 bpm, respectively. He was breathing comfortably through nose with respiratory rate of about 20/min. Auscultation of chest revealed no adventitious sounds. Routine laboratory results were within normal limits: white blood count 8900/mm³, haemoglobin 12 g/dL, platelet count of 159000/mm³, and prothrombin time 14.2 seconds (lab normal 13.2- 14.6 s).

In ultrasound room, the child became anxious, restless and unco-operative. Anaesthesiologist was called-in to manage the patient. A #20 IV cannula was inserted on the dorsum of his left hand under local anaesthesia. Midazolam (1mg) and fentanyl (50µg) were administered intravenously and flushed with normal saline. Within a minute the patient begun to cough explosively. The cough became quite vigorous and spasmodic. Oxygen was administered with face mask; however, the spasmodic cough persisted and in next two minutes the patient became cyanotic. Intermittent positive pressure ventilation with 100% oxygen on face mask showed no air entry into the lungs and child lost his consciousness

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with cessation of respiratory efforts. Peripheral haemoglobin saturation dropped (SpO₂) to 75% and soon became unrecordable. Endotracheal intubation was attempted with Macintosh laryngoscope but failed due to nonvisualization of larynx with massive intra-oral swelling of tongue and hypopharynx. Laryngeal mask airway (Size 2.5 and 3) was tried but failed to establish patent airway. Meanwhile, the patient developed severe bradycardia (heart rate 36-40/min). Atropine (0.6mg) was administered intravenously. Emergency airway access was achieved by inserting percutaneous cricothyroid cannula (12G) and 100% oxygen was administered. Oxygen saturation improved to 93-95% and heart rate increased to 140/min. In five minutes the patient started breathing with some inspiratory stridor. He became comfortable with return of normal breathing in next 15 minutes as intraoral swelling got decreased spontaneously; the cricothyrotomy cannula was removed. There was no further adverse event and patient recovered without any problem.

Figure 1

Figure 1: Patient with arteriovenous malformation of tongue and hypopharynx two hours after the incident



DISCUSSION

Fentanyl-induced coughing, though quite common, has not

been viewed as a serious anaesthetic complication. The mechanism involved in the production of FIC is not yet well understood but various theories have been proposed. Fentanyl has been shown to inhibit central sympathetic outflow, causing vagal predominance, inducing cough and reflex bronchoconstriction. It has also been shown to elicit cough by stimulating irritant receptors in tracheal smooth muscles. In a demographic study, Oshima et al [9] reported higher incidence of FIC in young patients and in patients who received benzodiazepine premedication while lower incidence of FIC was reported in light smokers. In this report, our patient was quite young, nonsmoker and had received benzodiazepine (midazolam) before iv fentanyl, the factors may have favoured occurrence of FIC.

In the first reported controlled study, Bo'hrer et al [1] found that 46% of patients coughed after receiving 7µg/kg through a central venous catheter. Phua et al. [2] observed that 28% of patients coughed after 1.5µg/kg IV of fentanyl injected through a peripheral cannula and that coughing was prevented by morphine but not by atropine or midazolam.

The rapid response of the reflex and morphine's efficacy in preventing cough suggest that a pulmonary chemo reflex is the likely mechanism, mediated by either irritant receptors (rapidly adapting receptors) or by vagal C-fibre receptors (Juxta receptors) that are close to pulmonary vessels [3]. However, fentanyl-induced vocal cord spasm and vagally mediated bronchoconstriction may also be important. Lui et al. [4] demonstrated that inhalation of a selective beta 2-adrenergic bronchodilator, terbutaline, suppressed the reflex. Bo'hrer et al. [1] reported that usually there were two to four coughs in sequence but in 4 of the 17 patients who coughed after fentanyl there was a staccato series of 8-15 cough efforts. Gin and Chui [5] observed a young patient with acute extradural haematoma who had continuous coughing for five seconds before suppression by thiopentone. Tweed and Dakin [8] reported a case of explosive coughing after peripheral injection of IV fentanyl (2µg/kg) that produced periorbital petechiae and was only relieved after induction of anaesthesia. The present case, however, is the first report of explosive spasmodic coughing with a life threatening morbidity, that is, engorgement of occult dormant sublingual arteriovenous malformation presenting as upper airway obstruction and severe hypoxia. Although a cause and effect relationship is impossible to prove, the temporal relationships and absence of other causative factors support the conclusion that this was an exaggerated reflex response

to fentanyl. The problem got exacerbated by the presence of intra-oral arteriovenous malformation that got engorged and caused upper airway obstruction with difficulty in ventilating the patient.

As per available literature there is no contraindication of fentanyl in patients with arteriovenous malformation. Explosive coughing increases intrathoracic pressure, intraocular pressure and intracranial pressure. In our case this increase in intrathoracic pressure and arterial pressure may have caused engorgement of intraoral arteriovenous malformation and produced a swelling almost occupying the whole of oral cavity extending up to oropharynx causing acute upper airway obstruction and subsequently severe hypoxia.

Various workers [1,2,3,4,5,6] have realized the gravity of potential harmful effects of fentanyl induced coughing and therefore several agents (i.e. i.v. lignocaine, ephedrine, dexamethasone or inhalation of salbutamol, beclomethasone or sodium chromoglycate) have been used and found successful in suppressing fentanyl induced coughing in significant number of patients. Therefore, pretreatment with one of the above mentioned agents may be used to prevent or minimize FIC. In situations, where coughing could be detrimental (i.e.; open eye injury, arterio-venous malformation in the airway or brain) fentanyl may not be suitable or should be used with caution for sedation or anaesthetic induction sequence.

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