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Anaphylaxis related to fentanyl citrate

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Abstract

Anaphylaxis is a fulminant, unexpected, immunoglobulin E-mediated allergic reaction that can be triggered by multiple agents. Common causative agents include neuromuscular blocking drugs, latex, antibiotics, colloids, hypnotics, and opioids. Fentanyl citrate, however, is an extremely unusual cause of anaphylaxis. Pulmonary edema, although uncommon in anaphylaxis, can be a prominent feature, as was in one of the patient. An adverse drug reaction is a noxious or unintended reaction to a drug that is administered in standard doses by the proper route for the purpose of prophylaxis, diagnosis, or treatment. Reactions are classified into two major subtypes: type A, which are dose dependent and predictable; and type B, which are not dose dependent and unpredictable. Unpredictable reactions include immune (allergic) or no immune drug hypersensitivity reactions and are related to genetic susceptibilities or undefined mechanisms (formally called idiosyncratic and intolerance reactions). A drug allergy is always associated with an immune mechanism for which evidence of drug-specific antibodies or activated T lymphocytes can be shown. In the last few years, many novel drugs have entered clinical practice (i.e., biologic agents) generating novel patterns of drug hypersensitivity reactions. As old drugs continue to be used, new clinical and biologic techniques enable improvement in the diagnosis of these reactions.

Keywords: Anaphylaxis, fentanyl citrate, immunoglobulin E, rashes

INTRODUCTION

Anaphylaxis is a medical emergency that is manifested by a broad spectrum of signs and symptoms that can be similar to those of other diseases. Drugs can cause immunoglobulin E (IgE)-mediated anaphylaxis. Vascular collapse and bronchospasm are hallmarks of this condition, but neither is universally present. Other findings, such as flushing or urticaria, are easily overlooked in surgical patients.

Numerous agents have been identified as triggers of intraoperative anaphylaxis, the most common being neuromuscular blocking drugs (NMBDs) and latex.^[1,2] In contrast, opioids rarely cause anaphylaxis. This report describes an unusual case of intraoperative anaphylaxis during a routine gynecologic procedure that was ultimately ascribed to intravenous fentanyl citrate.

CASE REPORTS

Case 1

A 31-year-old, 50-kg, G2P1 apprehensive woman with a history of abdominal ache from appendicitis

from the previous 4 days along with 101°F off and on fever was admitted in the hospital. She was planned to be operated for appendectomy. Her past medical and family history was not significant. Past surgical and anesthetic history included diagnostic hysteroscopy under conscious or moderate sedation (with propofol, midazolam, and fentanyl citrate), which was uneventful except the significant intraoperative nausea during diagnostic hysteroscopy which was treated with ondansetron and metoclopramide. She did not have any drug allergy in the past. She was premedicated with midazolam 1 mg intravenously. On physical examination, vital signs were BP = 104/60 mmHg, PR = 108/min, RR = 22/min, SpO₂ = 94% on room air. Her airway, cardiac, and chest examinations were normal. Laboratory studies including a complete blood count, electrolytes, glucose, and serum creatinine were also all within normal limits except the raised white blood cell counts. General anesthesia was planned on the patient's request and perspective. The patient was taken to the operating room and standard monitors were applied. After preoxygenation by using a face mask, anesthesia was induced intravenously with propofol 80 mg, lidocaine (preservative-free) 40 mg, fentanyl citrate 100 µg, and midazolam 0.5 mg. After mask ventilation was confirmed, vecuronium 6 mg was given intravenously. Laryngoscopy and tracheal intubation were uneventful. After confirmation of the tube placement, the patient was maintained on isoflurane <1.0% in 50% oxygen, 50% nitrogen. A latex-containing Foley catheter was then inserted into the bladder. Due to her past medical history of nausea and vomiting, metoclopramide 10 mg and ondansetron 4 mg were administered intravenously. Antibiotics were not given intraoperatively. She had one shot of antibiotics intravenously after skin sensitivity in the preoperative ward 30 min before coming to the operation theater (OT) room.

Approximately 10 min after induction but before skin incision, the electrocardiogram (ECG) showed brief episodes of supraventricular tachycardia (5–10 beats per episode). The patient otherwise appeared stable. The anesthetic was deepened and a total of 20 mg of esmolol was given intravenously in incremental doses. Over the next few minutes, the heart rhythm became sinus tachycardia at 140 beats/min. Generalized rashes could be seen all over the body. The patient's blood pressure decreased precipitously from a baseline of 104/60 to 60/30 mmHg. Intravenous fluids were opened to gravity and ephedrine 12 (6 + 6) mg was given intravenously without effect. Phenylephrine 200 µg was then given intravenously, which increased blood pressure only slightly to 70/50 mmHg. At this point, an ascending slope was noted on the capnogram, and the peak airway pressure was 30 cm H₂O, up from a baseline of 20 cm H₂O. Chest auscultation revealed bilateral wheezing, and the patient's oxyhemoglobin saturation (SpO₂) decreased from 100 to 90%. The inspired oxygen concentration was increased to 100%. An anaphylactic or anaphylactoid reaction was suspected, and epinephrine 20 µg was given intravenously in intermittent incremental doses.

The patient's blood pressure improved with epinephrine (110/80 mmHg), although peak airway pressures remained near 35 cm H₂O. An examination of accessible skin revealed flushing of the neck and upper torso, but no other signs. Her blood pressure plummeted again, and copious frothy secretions were noted emerging from the endotracheal tube. Prominent crepitations in lower lobes were heard on chest auscultation on each side. A positive end-expiratory pressure of 7.5 cm H₂O was applied in an effort to maintain alveolar recruitment.

Additional epinephrine was given, a radial arterial catheter was inserted, and arrangements were made to transfer the patient to our intensive care unit (ICU). The latex-containing Foley catheter was replaced with a polyvinyl chloride substitute. Arterial blood gases on a fractional inspired oxygen concentration (FiO₂) of 1.0 demonstrated metabolic acidosis and relative hypoxemia: pH 7.26, pCO₂ = 42 mmHg, PO₂ = 83 mmHg, with bicarbonate of 18.2 mEq/l (normal 22–26 mEq/l). An epinephrine infusion was started and titrated to maintain systolic blood pressure greater than 90 mmHg. Hydrocortisone 200 mg, diphenhydramine 50 mg, and ranitidine 20 mg were given intravenously.

The surgery was canceled, and the patient was transferred to the ICU. Intravenous fluids given up to this point consisted of approximately 3 l of Ringer's lactate solution. Chest X-ray showed bilateral infiltrates consistent with pulmonary edema. Ventilation was continued with positive end-expiratory pressure increased to 10 cm H₂O. The patient continued to require epinephrine for blood pressure support. In light of the significant pulmonary edema, a central venous catheter was inserted to follow filling

pressures as a guide for fluid management. Central venous pressures were normal, ranging from 8 to 10 cm H₂O.

A transthoracic echocardiogram revealed normal chamber size and cardiac function. An ECG showed sinus tachycardia without ST-segment or T-wave changes. Serial troponin I values were abnormal, with a peak value of 1.33 ng/ml at 8 h (normal < 0.05 ng/ml). Serial arterial blood gases were also obtained.

Blood and sputum cultures were obtained, which yielded no growth. An allergist was consulted, who concurred with the diagnosis of anaphylactic or anaphylactoid reaction. A latex radioallergosorbent test was negative. A serum tryptase concentration was ordered but not obtained.

Over the next two days, the patient's oxygenation improved and the FiO₂ was reduced. She continued, however, to have a metabolic acidosis, which slowly resolved. The peak lactic acid concentration was 7.2 mmol/l (normal < 2 mmol/l). The epinephrine infusion was tapered off after about 24 h. After 48 h of mechanical ventilation, the patient was conscious, hemodynamically stable, and well oxygenated. Her trachea was extubated, and the patient was continued on face-mask oxygen. She remained in the hospital for another 24 h and was discharged home without apparent sequelae.

One week after discharge, the patient saw the allergist for skin testing. Scratch and intradermal tests were negative for midazolam, propofol, rocuronium, and cisatracurium. Positive reactions (wheal and flare) were noted with both fentanyl citrate and succinylcholine. The allergist advised the patient to avoid fentanyl citrate and all paralytic agents, if possible. She was also seen by the consulting cardiologist and underwent an exercise stress echocardiogram, which was normal. She returned 1 month after the initial event for her hysterectomy. With intravenous premedication with midazolam 1.0 mg, a spinal anesthetic containing 2 ml of 0.5% hyperbaric bupivacaine along with clonidine 15 µg was given at the L₂₋₃ level. Cefuroxime 1 g was given intravenously without incident. Antiemetic prophylaxis was given intravenously, including dexamethasone 4 mg, metoclopramide 10 mg, and ondansetron 4 mg. Moderate sedation with dexmedetomidine (0.2 µg/kg/h) was continued throughout the case. Following surgery, the patient awakened readily and was transferred to the postanesthesia care unit without incident. She had an uneventful hospital course and was discharged home on the second postoperative day.

Case 2

A 26-year-old male patient with intertrochanteric fracture of left leg with history of road traffic accident 1 day before planned for open reduction and internal fixation. On preanesthetic checkup, his past medical and surgical history was nonsignificant except for the incision and drainage done for right gluteal abscess under local anesthesia. He was a casual smoker and alcoholic.

On physical examination, he was found to have a small laceration wound on forehead for which suturing and aseptic dressing were applied without any history of nausea, vomiting, ear, nose, throat (ENT) bleed, or loss of consciousness at the time of accident. His vital parameters were within normal limits. After taking the patient's written informed consent and nil per orally (NPO) history, his surgery was planned under a subarachnoid block. All the vital parameters were monitored inside the OT. He was found to be quite apprehensive on the day of surgery. After applying local anesthesia topically at the site of L₃₋₄ vertebral bodies, spinal anesthesia was performed in the sitting position with the help of the 26-Gauge Quincke needle after confirming free cerebrospinal fluid outflow 3 ml of 0.5% hyperbaric bupivacaine with clonidine 15 µg as adjuvant was introduced in the subarachnoid space. The effect was found to be adequate. The patient was sedated with the injection of midazolam 1 mg and fentanyl citrate 50 µg intravenously after repositioning the patient according to surgeon's perspective and demand in the left lateral position.

After 5 min of the administration of sedatives, the patient was found to have desaturated progressively with initial 98% SpO₂ to 70% along with the rise in his pulse rate and BP from the baseline 86 to 130 per min and from 126/86 to 150/100 mmHg, respectively. He was complaining of generalized pruritus all over the body. On physical examination, he was found agitated, restless, and tachypnoic at 40 breaths/min, with cyanosed coloration of lips and face. Pruritic rashes were seen on his arms and face after 10 min of spinal anesthesia and 5 min after administering fentanyl citrate intravenously. The

patient was not allowed to undergo surgery at that moment of time and managed conservatively with 100% O₂ via a tight mask on intermittent positive pressure ventilation (IPPV) manually in the 15° propped up position, injecting diphenhydramine 20 mg, dexamethasone 8 mg, hydrocortisone 100 mg, and betaprolol 5 mg intravenously every 10 min in incremental doses till vital parameters settled down to within normal limits. After 30 min of these conservative measures, he was found to be stable but still tachypnoic at 28 breaths/min. His arterial blood gas analysis (ABG) showed Ph = 7.28, paO₂ = 56 mmHg, paCO₂ = 28 mmHg, HCO₃⁻ = 30 mmHg.

Surgery was postponed on the same day as the patient shifted to ICU for further monitoring and close observation for motor and sensory blockade in lower limbs. He was kept on bilevel positive airway pressure noninvasive ventilation via mask electively for 2 h. He was later found to be conscious, oriented, and comfortable having vital parameters (PR, BP, SPO₂, ECG, RR, and pattern) and ABG within normal limits (Ph = 7.38, paO₂ = 86 mmHg, paCO₂ = 32 mmHg, HCO₃⁻ = 28 mmHg, and diminishing of rashes overnight when in ICU).

The same surgery was planned for the next day under general anesthesia, avoiding fentanyl citrate as analgesic. The patient was premedicated with an intravenous injection of ranitidine 50 mg and glycopyrrolate 0.2 mg. After preoxygenating for 5 min, he was induced intravenously with morphine 5 mg, propofol 100 mg, and vecuronium 6 mg. Anesthesia was maintained on O₂:N₂O (40:60) and isoflurane 1% and vecuronium 1 mg intermittently. On completion of surgery and after adequate spontaneous respiration, he was reversed normally by injecting neostigmine 2.5 mg and glycopyrrolate 0.4 mg intravenously. At the end of anesthesia, he was fully conscious to obey the verbal commands.

DISCUSSION

Anaphylaxis is a fulminant, unexpected IgE-mediated allergic reaction that can be triggered by multiple agents. A clinically indistinguishable syndrome, anaphylactoid reaction, can only be differentiated with later testing and is treated identically. The incidence of intraoperative anaphylaxis has ranged from 1 in 3500 to 1 in 20,000 anesthetics.[3] The initial presentation can vary, potentially involving numerous organ systems. The most common system involved is cardiovascular (hypotension, arrhythmia, myocardial depression, cardiovascular collapse), followed by cutaneous (flushing, urticaria) and respiratory (bronchospasm, laryngeal edema) manifestations.[2] The intraoperative period, however, poses numerous challenges to diagnosing anaphylaxis. Patients receive multiple drugs in close succession, are often unconscious and unable to relate symptoms, and are usually surgically draped, delaying recognition of cutaneous signs. This requires the anesthesiologist to be vigilant for what may initially be subtle indications.

The pathophysiology of anaphylaxis begins with the binding of an allergen to IgE on the surface of mast cells and basophils with the cross-linking of receptors and subsequent cell activation leads to the resultant massive release of mediators such as histamine, leukotrienes, kinins, and eosinophil. The chemotactic factor leads to bronchoconstriction, vasodilation, and increased capillary permeability. This process can continue, with progressive inflammation leading to a delayed 'second wave' of symptoms six to eight hours later.[4]

Numerous drugs and substances have been implicated in triggering anaphylaxis. A recent survey found that NMBDs, most commonly rocuronium and succinylcholine, accounted for 58.2% of perioperative events. These were followed by latex (16.7%), antibiotics (15.1%), colloids (4.0%), hypnotics (3.4%), and opioids (1.3%). Outside the perioperative period, insect stings and certain foods (peanuts, shellfish, and tree nuts) are also common triggers.

NMBDs have received much attention as the most frequent intraoperative trigger. A feature common to all NMBDs is two linked quaternary ammonium groups. This arrangement can bind to two separate IgE molecules and initiate the immune cascade. Individuals may be sensitized without prior exposure to NMBDs, because quaternary ammonium moieties are present in many products including over-the-counter drugs, food products, and cosmetics.[3]

Latex fluid contains several proteins that are believed to be the immunologic triggers of anaphylactic

reactions. Increasing awareness of bloodborne pathogens and use of barrier precautions has exposed more individuals to this agent. Consequently, it is now a major trigger of anaphylaxis.

Opioid-induced anaphylaxis, however, is rare. To date, there have been three reported cases of fentanyl citrate-induced anaphylaxis,^[5–7] one of which was subsequently retracted due to an undiagnosed latex allergy.^[8] In each of these cases, the reaction presented as hypotension and urticaria.

The treatment of perioperative anaphylaxis begins with removing the likely trigger and aborting the procedure, if possible. One hundred percent oxygen should be applied. Intravenous fluids should be given to compensate for vasodilation. Epinephrine should be administered intravenously, both for its β_2 effects (bronchodilation) and for its α_1 effects (increased vascular resistance). In addition, the β -agonist effect of epinephrine can inhibit further mediator release from mast cells and basophils. An epinephrine infusion may be necessary to support blood pressure. H_1 and H_2 antagonists and corticosteroids are useful to blunt later recurrence of the reaction. Invasive monitoring such as arterial blood pressure and central venous pressure measurements can help with hemodynamic management. In unclear cases, echocardiography or pulmonary artery catheterization may be of benefit. Regardless of the monitors used, admission to an ICU is warranted due to the possibility of airway edema or recurrent symptoms.

Tryptase, a protease released from activated mast cells, can be used as a marker of immune activation. An elevated serum tryptase concentration in blood drawn within several hours after the event can be helpful, although it cannot differentiate between anaphylaxis and an anaphylactoid reaction. A normal result, however, does not exclude these diagnoses.^[9,10] Subsequent outpatient skin testing may also be useful but suffers from variations in methodology and both false-positive and false-negative results.^[11,12] If utilized, skin testing should include both prick and intradermal tests, as each has its weaknesses: prick tests suffer from concentration dependence of the antigen and a lack of standardization, while intradermal tests are more prone to false-positive results.^[13] A positive skin test (wheal and flare) to a suspected agent suggests the presence of relevant IgE antibodies.

In the first case, pulmonary edema is rarely associated with anaphylaxis, but is not without precedent.^[14,15] Its rapid presentation in a relatively young woman with no cardiac disease, a normal echocardiogram, and normal central venous pressures suggests a noncardiac etiology. Since the patient was tracheally intubated and paralyzed, negative-pressure pulmonary edema from airway obstruction seems improbable. Without any increased risk for aspiration or a witnessed event, aspiration pneumonitis also seems unlikely. It is thus reasonable to suspect that anaphylaxis itself was the cause of pulmonary edema, especially as increased capillary permeability is a hallmark of the disorder.

The metabolic acidosis that developed in the first patient was pronounced. Elevated lactic acid levels usually imply tissue ischemia. In the setting of profound vasodilation requiring epinephrine infusion, acidosis was likely due to a distributive shock pathophysiology. As the inflammatory process diminished, the acidosis resolved slowly.

The elevation in troponin concentrations was concerning. The patient had no history of coronary artery disease and had good functional capacity. A lack of wall motion abnormalities on the postoperative echocardiogram and a subsequent negative stress test suggests that transient ischemia caused subendocardial myocardial injury. Severe vasodilation with insufficient coronary blood flow in the setting of aggressive epinephrine use may have created a significant energy supply–demand imbalance in the myocardium.

Subsequent skin testing (performed according to the recommendations of Mertes *et al.*^[12]) revealed positive reactions to both fentanyl citrate and succinylcholine. Given that the patient did not receive succinylcholine, fentanyl citrate seems the likely cause. If all skin test results were negative, the diagnosis of anaphylactoid reaction would be more appropriate. The positive results, however, argue for the presence of causative antibodies. The patient was not tested for the antiemetic drugs she received, but her uneventful reexposure to these same drugs argues against their triggering her anaphylaxis. The reaction to succinylcholine may also have been spurious, as there is a 9.3% prevalence of positive skin test results to NMBDs in the general population, without clinically apparent allergic reactions.^[16,17] Although the skin test results suggest she could safely receive rocuronium or cisatracurium, the allergist

instructed the patient to avoid NMBDs in the future, if possible.[18] Consequently, neuraxial anesthesia was chosen for appendicectomy in the first case.

In the second case, the development of cyanosis with generalized pruritic rashes after intravenously administering fentanyl citrate warrants further investigation related to anaphylaxis. The initial rise in vitals parameters (PR, BP, RR) might be suggestive of counter sympathetic stimulation of body immune defense system in response to progressively decreasing SpO₂. Since the patient received fentanyl citrate as sedative analgesic during the subarachnoid block, it was better decided to have total amnesia intraoperatively so the need of general anesthesia (GA) in place of regional anesthesia (RA) was advisable next time inside OT, along with morphine as analgesic in place of fentanyl citrate.

CONCLUSION

In brief, these cases describe otherwise healthy patients who experienced intraoperative anaphylaxis most likely due to fentanyl citrate allergy. Anesthesiologists should remain vigilant of the risk of intraoperative anaphylaxis as well as its treatment. We advocate the skin sensitivity test of the opioids (fentanyl) prior to their intravenous administration on routine basis for the patients posted for various surgeries.

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1st and 2nd Authors had contributed equally in the manuscript.

Footnotes

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REFERENCES

1. Mertes PM, Laxenaire MC, Alla F. Anaphylactic and anaphylactoid reactions occurring during anesthesia in France in 1999-2000. *Anesthesiology*. 2003;99:536–45. [PubMed: 12960536]
2. Laxenaire MC, Mertes PM. Anaphylaxis during anaesthesia. Results of a two-year survey in France. *Br J Anaesth*. 2001;87:549–58. [PubMed: 11878723]
3. Hepner DL, Castells MC. Anaphylaxis during the perioperative period. *Anesth Analg*. 2003;97:1381–95. [PubMed: 14570656]
4. Roizen MF, Fleisher LA. Anesthetic implications of concurrent diseases. In: Miller RD, editor. *Anesthesia*. 6th ed. Philadelphia: Elsevier; 2005. pp. 1092–3.
5. Bennett MJ, Anderson LK, McMillan JC, Ebertz JM, Hanifin JM, Hirshman CA. Anaphylactic reaction during anaesthesia associated with positive intradermal skin test to fentanyl. *Can Anaesth Soc J*. 1986;33:75–8. [PubMed: 3948052]
6. Fukuda T, Dohi S. Anaphylactic reaction to fentanyl or preservative. *Can Anaesth Soc J*. 1986;33:826–7. [PubMed: 3779505]
7. Zucker-Pinchoff B, Ramanathan S. Anaphylactic reaction to epidural fentanyl. *Anesthesiology*. 1989;71:599–601. [PubMed: 2802218]
8. Zucker-Pinchoff B, Chandler MJ. Latex anaphylaxis masquerading as fentanyl anaphylaxis: Retraction of a case report. *Anesthesiology*. 1993;79:1152–3. [PubMed: 8239002]
9. Kroigaard M, Garvey LH, Menne T, Husum B. Allergic reactions in anaesthesia: Are suspected causes confirmed on subsequent testing? *Br J Anaesth*. 2005;95:468–71. [PubMed: 16100238]
10. Fisher MM, Baldo BA. Mast cell tryptase in anaesthetic anaphylactoid reactions. *Br J Anaesth*. 1998;80:26–9. [PubMed: 9505773]
11. Nasser SM, Ewan PW. Opiate-sensitivity: Clinical characteristics and the role of skin prick testing. *Clin Exp Allergy*. 2001;31:1014–20. [PubMed: 11467991]

12. Mertes PM, Laxenaire MC, Lienhard A, Aberer W, Ring J, Pichler WJ, et al. Reducing the risk of anaphylaxis during anesthesia: Guidelines for clinical practice. *J Investig Allergol Clin Immunol*. 2005;15:91–101.
13. Dhonneur G, Combes X, Chassard D, Merle JC. Skin sensitivity to rocuronium and vecuronium: A randomized controlled prick-testing study in healthy volunteers. *Anesth Analg*. 2004;98:986–9. [PubMed: 15041585]
14. Gallerani M, Manzoli N, Fellin R, Simonato M, Orzincolo C. Anaphylactic shock and acute pulmonary edema after a single oral dose of acetazolamide. *Am J Emerg Med*. 2002;20:371–2. [PubMed: 12098191]
15. Low I, Stables S. Anaphylactic deaths in Auckland, New Zealand: A review of coronial autopsies from 1985 to 2005. *Pathology*. 2006;38:328–32. [PubMed: 16916722]
16. Matthey P, Wang P, Finegan BA, Donnelly M. Rocuronium anaphylaxis and multiple neuromuscular blocking drug sensitivities. *Can J Anesth*. 2000;47:890–3. [PubMed: 10989860]
17. Porri F, Lemiere C, Birnbaum J, Guilloux L, Lanteaume A, Didelot R, et al. Prevalence of muscle relaxant sensitivity in a general population: Implications for preoperative screening. *Clin Exp Allergy*. 1999;29:72–5. [PubMed: 10051704]
18. Cummings KC, III, Arnaut K. Case report: Fentanyl-associated intraoperative anaphylaxis with pulmonary edema. *Can J Anaesth*. 2007;54:301–6. [PubMed: 17400983]

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