CASE REPORTS

avoid this, one may opt to use the largest endotracheal tube available, inflate the cuff as usual, and then pack the oropharynx with enough gauze to create an airtight seal. This is useful for patients in whom muscle relaxation and mechanical ventilation are needed and for those who may have a full stomach to reduce the risk of aspiration. For patients in whom this risk is not present, a brain laryngeal mask may be satisfactory. Ankylosing spondylitis has been listed as a contraindication to the insertion of a laryngeal mask. In a case report of a patient with ankylosing spondylitis, it was reportedly impossible to insert a laryngeal mask because of a severely acute angle between the oral and pharyngeal axes. The neck of our patient had pathologically fused at an angle greater than 90°, making insertion of the laryngeal mask easy.

In conclusion, we have presented the case of a patient with ankylosing spondylitis who was difficult to mechanically ventilate because of an unusually large trachea and subsequent leakage from around the cuff. The laryngeal mask was effectively used as an alternative to the endotracheal tube.

References


Unusual Hypotension and Bradycardia in a Patient Receiving Fenfluramine, Phentermine, and Fluoxetine


FENFLURAMINE§ and phentermine are used for the management of obesity. Phentermine acts via central catecholamine pathways. Fenfluramine and fluoxetine act via serotonergic pathways. Additionally, with chronic use, fenfluramine has been reported to have catecholamine-depleting effects. Reported side effects, in patients taking fenfluramine and phentermine, range in severity from bothersome to fatal. We report a case of a patient receiving a long-term course of fenfluramine, phentermine, and fluoxetine who experienced hypotension and bradycardia during regional anesthesia with sedation.

Case Report

A 50-yr-old, 90-kg, 185-cm, American Society of Anesthesiologists' physical status II man presented for PIP fusion of the right second toe. Medical history was positive for a distant episode of hepatitis A and cessation of smoking 20 yr previously. Baseline electrocardiograph (ECG) revealed sinus bradycardia of 56 beat/min. Preoperative laboratory studies were unremarkable. Personal and family history were negative for anesthesia-related complications. Although initially denied, medication history was positive for phentermine, 18.75 mg/day, combined with fenfluramine, 20-40 mg/day for approximately...
18 months. Additionally, for the past 24 months, the patient had been taking fluoxetine, 20 mg/day, for depression. He denied use of alcohol or illicit drugs. All medications were prescribed by the patient’s primary care physician.

Because of the fenfluramine package insert recommendations, we chose not to use general anesthesia. The patient refused to have a spinal anesthetic but agreed to an ankle block with sedation.

Baseline vital signs included a blood pressure (BP) of 139/96 mmHg, heart rate (HR) of 73 beat/min, and respirations of 14 breath/min. After application of routine monitors and oxygen by nasal cannula at 4 l/min, the patient was sedated with midazolam, 2 mg, in divided doses. Additionally, fentanyl was administered to a total titrated dose of 150 μg over approximately 20 min while a right ankle block was administered. Twenty-five milliliters of preservative-free lidocaine, 2%, was injected with a 10-ml supplement 20 min later to block residual sensation. After surgical prep, the case proceeded without incident until nearly completed.

Approximately 25 min after start of surgery and 55 min after administration of any medication, the patient experienced sudden bradycardia (HR, 45 beat/min) and hypotension (BP, 67/44 mmHg). After a divided dose of 150 μg of phenylephrine, blood pressure was restored to 110/80 mmHg. Heart rate returned to 70 beat/min after intravenous administration of glycopyrrolate, 0.2 mg. SpO₂ increased from 96% to 99%. Before this, the average HR was 72 beat/min, and average systolic BP was 120 mmHg. At the time of the hypotension and bradycardia, the patient appeared calm and was responsive to verbal stimuli. He denied surgical discomfort, chest pain, shortness of breath, nausea, or vomiting. No change in ST segments, mental status, or onset of diaphoresis was noted.

Surgery was completed without further incident. Fifteen minutes post-episode, his initial postanesthesia care unit (PACU) presentation was BP, 126/85 mmHg; HR, 77 beat/min; and SpO₂, 98%. He was receiving an FiO₂ of 0.4 by face mask, lying in mid-fowler’s position, in no apparent distress, and without complaints of any type. A 12-lead ECG was obtained with an increase in HR to 85 beat/min being the only change noted from preoperative ECG.

Several minutes post-PACU admission, he complained of feeling ‘tired.’ His HR had decreased to 58 beat/min, and systolic BP was 98 mmHg with an SpO₂ of 95%. He was placed in the supine position and noted to be diaphoretic. Glycopyrrolate was titrated to effect over the next 15–20 min. A dose of 0.8 mg was administered before diaphoresis and bradycardia subsided. HR then held steady between 76 and 84 beat/min, and systolic BP ranged between 110 and 120 mmHg. SpO₂ ranged between 96% and 98%.

After being symptom-free for about 40 min, diaphoresis was again noted when the patient resumed the mid-fowler’s position but without change in vital signs. A tilt-test was performed, and the results were negative. In addition, he denied malaise or nausea. After diaphoresis resolved, the remainder of recovery was uneventful.

Cardiology service evaluated the patient but found no cardiac-related reason not to proceed with same-day discharge. The patient returned 18 h later for postoperative evaluation and to have a troponin I level drawn. He denied recurrence of any postoperative symptomatology.

Discussion

Although multiple etiologies of perioperative hypotension exist, many potential causes may be ruled out as a result of our patient’s lack of medical history, lack of physical findings, and normal preoperative laboratory values. We considered the possibility of a vasovagal-type reaction, although our patient manifested no signs of psychological distress and denied any emotional or physical discomfort. Even though a substantial period of time had elapsed since the administration of the local anesthetic or sedation-analgesia, the patient remained comfortable, adequately sedated, and denied surgical pain. Considering this, we could find no satisfactory mechanism of vasovagal-mediated hypotension or bradycardia. We considered the possibility of myocardial ischemia. The patient’s troponin-I level was negative. Cardiology service found no evidence of a cardiac event and provided a diagnosis of autonomic dysfunction of unknown origin.

We recognize the tenuous connection between this patient’s use of fenfluramine, phentermine, and fluoxetine and the delayed cardiovascular responses after regional anesthesia with sedation-analgesia. This delay may point to the degree to which catecholamine reserves were decreased in relation to the amount of depression induced by the sedation-analgesia or local anesthetic. Catecholamine reserves could have been consumed more rapidly had a more potent anesthetic been used. Autonomic dysfunction as a result of catecholamine depletion has been documented with general anesthesia. Fenfluramine associated autonomic dysfunction appears similar to that seen with other catecholamine-depleting drugs. However, this has been reported as problematic only during induction of general anesthesia. No reports of synergism between fluoxetine and fenfluramine or phentermine could be found.

Indirect-acting sympathomimetics completely depend on sufficient neuronal stores of norepinephrine. Therefore, because of suspected catecholamine depletion from chronic drug therapy, we treated the intraoperative hypotension with a direct-acting sympathomimetic. Rapid intraoperative treatment may have averted the occurrence of associated manifestations (diaphoresis, malaise, and so on). In contrast, delayed postoperative management of borderline hypotension and bradycardia may have allowed the occurrence of the symptoms that developed in the PACU.

Considering the dose of glycopyrrolate necessary to resolve the postoperative bradycardia and the length of time required before symptoms abated, the presence of autonomic dysfunction cannot be excluded. Addi-
tionally, one cannot rule out catecholamine depletion as a result of decreased reserves of norepinephrine from chronic drug therapy.\(^3\) We postulate that fenfluramine, phentermine, and fluoxetine, acting separately or in combination, precipitated the hypotension, bradycardia, and accompanying symptoms manifested by our patient.

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Unusual Psychological Manifestation of Systemic Local Anesthetic Toxicity

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SYSTEMIC local anesthetic intoxication is a potentially life-threatening complication that has been repeatedly described. Low plasma concentrations of local anesthetics may produce dizziness, drowsiness, numbness of tongue, tinnitus, dysarthria, and visual disturbances, whereas higher blood levels may result in seizures, coma, and respiratory arrest. Whereas these neurologic symptoms of central nervous system toxicity are familiar to all anesthesiologists, the psychological manifestations of local anesthetic toxicity are less well known. It has been proposed that apprehension about imminent death or the delusion that death has actually occurred is a specific feature of local anesthetic toxicity.\(^1\) We present a case in which overt systemic local anesthetic toxicity was associated with a profound psychological experience.

Case Report

A 59-year-old woman was scheduled for surgery of Dupuytren’s contractures of the fourth and fifth finger of her right hand, using axillary plexus block. Preoperatively, she did not take any medication, and examination revealed no cardiac, pulmonary, neurologic, or mental disease. Oral midazolam, 7.5 mg, was given 1 h

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