CASE REPORT

A 34-year-old man was admitted to the Day Surgical Unit (DSU) for elective knee arthroscopy. Standard preoperative evaluation included an unremarkable medical history (derived from a health survey), physical examinations by the orthopedic surgeon and the anesthesiologist, and a complete blood count. He had no previous electrocardiogram or chest radiograph. No abnormalities were noted in his examination or laboratory studies. The patient played semi-professional ice hockey until 1 yr prior to surgery. He continued to play softball on weekends. There was no family history of cardiac disease. He was 6-feet tall and weighed 100 kg. Prior to induction of anesthesia blood pressure was 150/90, and an oscilloscope trace of the EKG revealed sinus tachycardia at a rate of 115, with infrequent premature ventricular contractions (less than one per min).

Anesthesia was induced with 500 mg sodium thiopental, 1 mg atropine, and 400 mg lidocaine. The patient was not breathing 100% oxygen prior to induction of anesthesia. An additional 100 mg sodium thiopental were administered when the patient failed to lose consciousness. No muscle relaxant was given because an inhalational anesthetic mask was planned. Shortly after receiving the second dose of pentothal, the patient developed a supraventricular tachycardia at a rate of 180 bpm, the reading on the pulse-oximeter (Nellcor®) decreased to less than 50, and the blood pressure was unable to be obtained by an automated blood pressure cuff (Dynamap®).

The patient’s lungs were easily ventilated while cricoid pressure was applied and he was then given 10 mg ephedrine, 100 mg succinylcholine, and the trachea was intubated without difficulty. The end-tidal CO₂ was 45. His blood pressure failed to increase, and the electrocardiogram showed bradycardia, and a wide-complex indeterminate rhythm (no paper readout is available on the DSU monitors). A total of 2 mg epinephrine, an additional mg of atropine, 44 meq sodium bicarbonate were given, and the lungs were ventilated with 100% oxygen via the endotracheal tube. The resuscitative efforts were successful, and the patient’s pulse increased to 120, with a blood pressure of 120/90. The surgical procedure was cancelled, and a subsequent chest film showed a mildly enlarged cardiac silhouette. A 12-lead electrocardiogram showed sinus tachycardia, right atrial enlargement, left axis deviation (~48 degrees), a nonspecific intraventricular conduction delay, and inverted anterolateral T waves.

The patient was admitted to the hospital and vital signs and electrocardiogram were monitored for 48 h. On further questioning, the patient denied illicit drug use, exercise intolerance, or a recent viral illness. He was employed as a carpet layer, and worked with unknown industrial solvents. His family claimed that the patient was a heavy alcohol drinker. A toxicology screen showed only those drugs administered during the resuscitative effort.

During the patient’s workup in the hospital, the electrocardiogram remained persistently abnormal, showing left axis deviation, left ventricular hypertrophy, and sinus tachycardia with inverted T waves in the precordial leads. An echocardiogram showed an enlarged left atrium and ventricle, global hypokinesia, moderate mitral regurgitation, and an ejection fraction of 35%. Serial cardiac isoenzymes showed no evidence of acute myocardial damage. Thyroid function tests were normal.

Further evaluation and cardiac biopsy were proposed to the patient who was unwilling to undergo more definitive investigation. He was discharged and refused follow-up studies. During the following year, the patient’s cardiac disease progressed to the point where he was unable to continue working. Noninvasive cardiac studies at another institution showed a decrease in ejection fraction with exercise, consistent with a moderate to severe cardiomyopathy.

DISCUSSION

This patient is typical of most same-day surgical patients, i.e., an apparently vigorous, young patient scheduled for a minor procedure requiring only short-term postoperative care. His screening health survey was unremarkable, as was his physical examination. The tachycardia and infrequent premature ventricular contractions seen immediately before surgery are not unusual in anxious and unpremedicated surgical outpatients.

Retrospective evaluation suggests a plausible explanation for this patient’s problems during anesthesia. His postoperative electrocardiograms, echocardiogram, and clinical course are consistent with a dilated cardiomyopathy. His initial failure to respond to relatively large doses of induction agents may have been due to a decreased cardiac output and increased circulation time.

There were no difficulties with management of the patient’s airway or with ventilation of his lungs, as confirmed by the low end-tidal CO₂ after intubation which suggests
that ventilatory insufficiency is not an adequate explanation for his dysrhythmias and desaturation upon induction. Like many anxious, presumably healthy day surgical patients, this patient did not breathe oxygen before induction of anesthesia and this clearly contributed to the prompt decrease in saturation. There are two factors in this case that impaired the patient's already compromised cardiac performance and may have potentiated the decrease in arterial saturation and caused his profound hypotension. The first is drug-induced myocardial depression from thiopental and lidocaine. The second is tachycardia and diminished filling time in a dilated and consequently preload dependent heart. The exact etiology and nature of his SVT is unclear, but it is almost certainly due to some interaction between the induction agents and his diseased myocardium. Patients with a cardiomyopathy and decreased cardiac output may have a low mixed-venous oxygen saturation, and desaturation of arterial blood may therefore readily occur with any further decrease in cardiac output.

The choice and dosage of induction agents are standard in our day surgery unit, where sodium thiopental (5–7 mg/kg), atropine, and lidocaine (2–4 mg/kg) are used in combination to prevent vagal and upper airway responses to rapid induction with pungent inhalational agents. This patient received 6 mg/kg of thiopental, a dose that is acceptable and often necessary in day surgery patients who have received no preanesthetic medication, but well in excess of that which should be given to a patient with a cardiomyopathy. The dose of lidocaine, 4 mg/kg, is larger than that used in most studies of lidocaine as an induction agent, but should lead to a serum concentration of approximately 4 μg/ml in a healthy 100-kg male, within the therapeutic concentration of this agent. The volume of distribution of lidocaine in congestive heart failure is reduced, however, and the peak serum concentration of lidocaine may have exceeded the toxic level of 10 μg/ml in this patient. A regimen that we have safely used for 7 yr in our DSU was clearly inappropriate in this patient with an unappreciated cardiomyopathy.

A recent review of dilated cardiomyopathies describes a large variety of potential causes: toxic, metabolic, infectious, and idiopathic.2 Electrocardiographic findings frequently show ventricular extrasystoles, left bundle branch block, or left ventricular hypertrophy with poor R wave progression. There are often nonspecific T wave changes. Many of these features were prominent on our patient's postoperative EKG. Echocardiographic criteria for the diagnosis of dilated cardiomyopathy include an ejection fraction of less than 40%, a dilated and hypokinetic left ventricle, and mild to moderate mitral regurgitation, all of which were present in this patient.

The two most likely etiologies for a dilated cardiomyopathy in this patient appear to be idiopathic and alcoholic (which may appear in patients 30–55 yr of age without other manifestations of long-term alcohol abuse such as cirrhosis or peripheral neuropathy). A recent study of 50 asymptomatic alcoholic men (mean age 38.5 yr) showed that one-third had ejection fractions of 55% or less.3 Any further speculation as to cause is unwarranted given the absence of biopsy data.

In addressing the question of screening for patients with this disorder in its presymptomatic phase, we attempted to identify signs and symptoms that might have marked this patient as a potential anesthetic risk. Consistent with this patient's cardiomyopathy are preoperative premature ventricular contractions, postoperative electrocardiographic abnormalities and echocardiographic changes. The ventricular extrasystoles are a known normal finding in healthy adults in hyperadrenergic states, and neither specific nor sufficient enough to change most anesthetic plans. The usefulness of preoperative EKG's as a sensitive, specific screening test in young apparently healthy patients under forty years of age is limited.4 An echocardiogram is almost never warranted as a preoperative test in an otherwise healthy adult.

In a retrospective evaluation, no remediable flaws in the preoperative screening methods for the DSU or same day surgical admissions were detected. While some of the drug dosages and choices may be questioned, they are usually not only tolerated but required in most patients seen in the DSU. One lesson to be taken from this case is that the lack of response to an initial dose of induction agents, usually interpreted as a need for additional drug in a healthy patient, may in fact indicate a significantly prolonged circulation time and warrant caution instead.

Several years ago, this patient would have been admitted to the hospital on the night prior to surgery, and a routine chest film, electrolyte panel, complete blood count, urine analysis, and electrocardiogram performed. The borderline abnormalities of his chest film and electrocardiogram might have led to a more exhaustive history and physical and ultimately to the diagnosis of his occult cardiomyopathy. In today's era of necessary cost containment, however, the enormous expense of routine preoperative admission and screening studies, with the attendant rate of false-positive reports, must be weighed against the potential benefit to a single patient.

This patient represents an anomalous example of a potentially occult disease process with significant anesthetic ramifications that might have been avoided by a more rigorous preanesthetic evaluation. Cardiomyopathy should be considered in the evaluation of patients with a history of significant alcohol intake or an unusual reaction to usual anesthetic induction regimens.

REFERENCES


A single dose of intrathecal fentanyl 25 μg can extinguish established phantom limb pain and restore normal sensations for about 8 h.¹ Acute phantom pain unmasked in an amputee undergoing elective cesarean section during epidural anesthesia, was effectively treated with two doses of epidural fentanyl (75 μg), each providing relief for 3–4 h.² Consequently, spinal opioids were recommended for acute phantom pain associated with epidural or spinal anesthesia.² We describe the management of an amputee with severe and intractable acute postamputation phantom foot pain refractory to conventional forms of acute pain therapy, using spinal opioids administered over an extended period.

REPORT OF A CASE

A 61-yr-old man with adult onset diabetes mellitus and hypertension presented for a right transmetatarsal amputation because of necrotic ulcers at the base of his second and third toes. He had no pain operatively and was not taking any pain medication. Other medications included chlorpropamide 250 mg daily and propranolol 40 mg every 6 h.

The procedure was conducted uneventfully with spinal anesthesia (tetracaine 6 mg with epinephrine 0.2 mg in dextrose). When the effects of the anesthetic receded 5 h postinjection, the patient noted severe pain in his stump and right phantom foot. It was a constant, throbbing pain that involved his whole foot from the ankle distally. It felt as if the whole foot below the ankle (including the toes) was in a vise. Frequent unpredictable paroxysms of lancinating pain were superimposed. They lasted a few seconds and radiated down the dorsal aspect of the foot into the toes, which felt as if they were clamped by a powerful, sharp pincer (“bitten by a giant crab”).

In the ensuing 48 h, a variety of pain treatments were tried without success. These included morphine 10 mg im every 2 h, meperidine 100 mg with hydroxyzine 50 mg im every 3 h, iv patient-controlled analgesia (PCA) with morphine (incremental bolus 1.5 mg; lockout 6 min), and oxycodone with acetaminophen (percoct) two tablets every 4 h.

After obtaining written informed consent, the patient was brought to the operating room area. The plan was to give a diagnostic intrathecal injection of fentanyl which, if successful, would be followed with the insertion of an indwelling epidural catheter to be used for continued morphine therapy. Using a verbal numerical pain rating system (0 = no pain and 10 = worst pain imaginable), the baseline pain score was 7/10. An iv cannula was inserted, monitoring established and oxygen administered via nasal cannulae. The patient was placed in the left lateral decubitus position and the subarachnoid space located at the L2/3 level with a 22-G spinal needle inserted under sterile conditions. Fentanyl 25 μg (0.5 ml) was injected, the needle removed, and the patient placed supine.

Significant effects were present within 2 min of the injection. The patient initially noted a sensation of warmth in his lower trunk and legs. The phantom foot felt warm, relaxed and of normal proportions. By 3 min there was no discomfort (0/10). By 15 min the phantom foot sensations were absent and the stump felt warm and comfortable. There was no pruritus, nausea, vomiting, urinary retention, excessive drowsiness, or clinical respiratory depression associated with the fentanyl injection.

Spinal opioids were superior to any of the other treatments tried, and the patient expressed enthusiasm for pursuing this type of therapy. Consequently, 30 min after the fentanyl injection, an indwelling epidural catheter was inserted in the L3/4 interspace, under sterile conditions. Epidural injections were, however, withheld until the effects of the fentanyl began to recede. Four hours postinjection the patient felt a mild tingling sensation in his phantom right big toe, which by 4.5 h involved all the toes. The discomfort was mild (2/10).

Preservative-free morphine (Duramorph®, Elkins-Sinn, New Jersey) 5 mg was injected via the epidural catheter. Fifteen minutes later the patient was without discomfort (0/10) and remained so for 15 h, when he again described tingling in his phantom toes associated with mild discomfort (1/10). Subsequently, duramorph 5 mg was injected every 12 h via the epidural catheter. Urinary retention requiring catheterization for 3 days after the initiation of epidural morphine was the only adverse effect. Eleven days of therapy ensued during which time there was no recurrence of phantom pain (0/10).

The stump was healing well and the patient was ambulating and participating in physical therapy. He was being considered for a walking cast and discharge from the hospital. Therefore, slow discontinuation of the epidural morphine was initiated. One dose of epidural morphine was omitted and the pain did not recur. The epidural catheter was left in place and the patient was started on percorct two tablets every 6 h. After a further 24 h without phantom pain, the epidural catheter was removed. The patient remained in the hospital another 10 days, during which time percorct was discontinued with no recurrence of phantom pain. Subsequent to discharge he has had no phantom limb pain.