Journal of Clinical Anesthesia (2009) **21**, 131–134

Journal of Clinical Anesthesia

Case report

Transient anesthetic-induced worsening of existing left-sided weakness in a patient undergoing elective anterior cervical discectomy

Aaron M. Joffe DO (Resident)[a,*], Azam Ahmed MD (Resident)[b], Gregory R. Trost MD (Professor)[b], Karl Willman MD (Assistant Professor)[a]

[a]*Department of Anesthesiology, University of Wisconsin Hospital and Clinics, Madison, WI 53792, USA*
[b]*Department of Neurosurgery, University of Wisconsin Hospital and Clinics, Madison, WI 53792, USA*

Received 23 May 2007; revised 22 June 2008; accepted 25 June 2008

Keywords:
Intravenous anesthetics;
Opioids;
Benzodiazepines,
Transient neurologic deficits

Abstract The differential diagnosis of new or worsening focal neurologic deficits on emergence from anesthesia is broad. Cerebral ischemia or hemorrhage, focal seizures, and acute metabolic abnormalities can all result in similar neurologic findings. Intravenously administered anesthetic agents also have been reported to cause new or worsening focal neurologic deficits in patients with a history of preexisting deficits. A patient who suffered such a reversible deficit related to anesthesia is presented.

1. Introduction

The presence of new or worsening neurologic findings on emergence from anesthesia is a troubling sign. Immediate concern centers over the possible occurrence of acute cerebral ischemia or hemorrhage, or spinal cord compression. The differential diagnosis of focal neurological findings is broad and includes such entities as focal seizures, complicated migraine headaches, and metabolic abnormalities including hypoglycemia, hyponatremia, hypocalcemia, and hypomagnesemia. While volatile anesthetics themselves have never been directly implicated, intravenously (IV) administered benzodiazepines and opioids used as part of balanced anesthesia or by themselves unmask or worsen prior neurological deficits in diverse groups of neurologically injured patients [1-6].

A 52-year-old woman who underwent an elective anterior cervical discectomy and fusion (ACDF), with profound worsening of her preexisting neurological deficits, is presented.

2. Case report

A 52-year-old, 62 kg woman presented for ACDF from level 4 through 7. The patient had a history of carotid disease with prior transient ischemic attacks (TIAs) presenting as parasthesias in the extremities on the right, for which she underwent a left carotid endarterectomy 6 months previously. She underwent a duplex ultrasound study of her carotid arteries 10 days prior to undergoing the ACDF that

* Corresponding author. Tel.: +1 608 262 8328; fax: +1 608 263 8114.
E-mail address: ajoffe@uwhealth.org (A.M. Joffe).

0952-8180/$ – see front matter © 2009 Elsevier Inc. All rights reserved.
doi:10.1016/j.jclinane.2008.06.034

showed no significant stenoses in either artery. No new neurological symptoms had been reported since the carotid endarterectomy. By history, the patient had experienced chronic radicular pain and weakness of the left upper extremity attributed to her cervical spine disease. The patient also suffered from essential hypertension and hypercholesterolemia, treated with metoprolol extended-release 100 mg daily, amlodipine 5 mg daily, and atorvastatin 20 mg daily. On the morning of surgery, a baseline neurologic examination showed a cooperative, alert, and oriented woman without aphasia, dysarthria, cranial nerve deficits, or abnormal cerebellar function. Strength testing revealed mild deficits on the left; 4/5 in the deltoid, 4+/5 in the biceps, 4/5 hand grasp, and 4/5 plantarflexion and dorsiflexion. The lower extremity weakness was surprising to the patient as she was unaware of it prior to the examination. Cardiovascular examination was normal, carotid bruits were absent, heart rate was 68 beats per minute and regular, and blood pressure was 126/86 mmHg. Pre-anesthetic medication consisted of one mg of IV midazolam. No neurologic changes were noted after administration. Anesthesia was induced with fentanyl 100 mcg and propofol 190 mg administered IV. Intubation was facilitated with vecuronium 6 mg. Anesthesia was maintained with 4% to 5% end-tidal desflurane in two L of fresh gas flow consisting of one L of 100% oxygen and one L of air. Additional doses of fentanyl and vecuronium were given as needed. Routine ASA monitoring was performed. Blood pressure was measured non-invasively every 2.5 minutes during induction of anesthesia and then continuously following insertion of a 20-gauge peripheral arterial catheter in the left radial artery. Degree of neuromuscular block was monitored with a hand-held peripheral nerve stimulator and maintained at one to two twitches. Hypotension was treated with titrated doses of ephedrine 5 mg and/or phenylephrine 100 mcg while tachycardia and hypertension were treated with esmolol 10 mg and labetolol 10 mg, respectively. Blood pressure reached a nadir after induction and was then stable. The patient was in normal sinus rhythm. End-tidal carbon dioxide tension was maintained between 31 and 35 mmHg, and oxygen saturation by pulse oximetry was 99-100%. Intraoperative sodium, potassium, ionized calcium, glucose, and hemoglobin concentration obtained two hours and 15 minutes into the case were within normal limits. The case proceeded uneventfully and the patient's trachea was extubated in the operating theater after reversal of neuromuscular block with neostigmine three mg and glycopyrrolate 0.4 mg. The surgical procedure itself involved a right-sided approach, was uncomplicated, and proceeded without undue compression of the spinal cord. In the recovery area, the patient complained of difficulty in moving her left arm and leg. No concomitant sensory changes were noted. A non-contrast computed tomogram of the head, as well as magnetic resonance images (MRI) of the head and cervical spine, showed no acute intracranial or spinal pathology. In addition, magnetic resonance arteriography (MRA) of the intracranial and extracranial vessels did not show any flow-limiting stenoses or aneurysms. The patient was admitted to the neurosurgery service where her deficits gradually improved and returned to baseline after 6 hours.

3. Discussion

Anecdotal reports of transient neurological symptoms in patients with cerebral ischemia, intracranial mass lesions, and cervical spine disease temporally related to anesthesia has been reported for more than 25 years [1-3,5]. The clinical observation of evolving lateralizing neurologic signs on awakening from anesthesia and the subsequent return to complete function has been termed "differential awakening" [7]. It is theorized to result from differential pharmacokinetics of anesthetic drugs between normal and injured brain, hypersensitivity of injured neurons to sedative medications, or loss of neural network redundancy with focal injury leaving a limited reserve in the presence of a centrally acting drug.

Although the theoretical mechanisms imply that any central nervous system depressant, including inhaled anesthetics, may lead to this phenomena, no implicit evidence exists in the literature. On the contrary, direct clinical observation and prospective human study has shown that IV opioids can reproduce neurologic deficits. Baskin and colleagues reported elevated levels of endogenous opioids such as immunoreactive β-endorphin-like substance and leucine enkephalin obtained from the cerebrospinal fluid of patients with ischemic brain injury and the improvement or complete reversal of neurological deterioration after administration of an opioid receptor antagonist (naloxone) [1]. Other authors have also reported cases of transient neurologic deficits that improved or were reversed by administration of naloxone. Subsequently, diazepam and, by default, gabaminergic transmission, also were implicated [5]. The ability of both opioids and benzodiazepines to reproduce or worsen neurological deficits has been directly studied and confirmed in earlier clinical observations [6]. Thal and colleagues administered either midazolam or fentanyl in divided doses to achieve light sedation after a baseline neurologic examination of 54 patients scheduled for elective surgery to remove a supratentorial mass lesion or undergo carotid endarterectomy. When the neurologic examination was repeated 5 minutes later, deterioration was seen in 30% of patients overall; 35% of those receiving midazolam and 22% receiving fentanyl. Most notable was the finding that 73% (16/22) of the patients with preexisting motor deficits on study entry experienced a significant deterioration in their motor examination after medication administration. These observations have been extended by Lazar and colleagues [4]. Eight patients who suffered a stroke as recently as one week to 6 years before study entry were given midazolam in