Journal of Clinical Anesthesia (2016) **34**, 344–347

ELSEVIER

Journal of Clinical Anesthesia

Case Report

Anesthetic considerations for renal transplant surgery in patients with mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes syndrome: a case report☆,☆☆

Michelle L. Humeidan MD, PhD[a,*], Julia Dalia BS[b], Wanye D. Traetow MD[a]

[a]*Department of Anesthesiology, The Ohio State University Wexner Medical Center, Columbus, OH, USA*
[b]*Saint Louis University School of Medicine, St Louis, MO, USA*

Received 24 February 2016; revised 4 May 2016; accepted 4 May 2016

Keywords:
Mitochondrial myopathy;
Renal transplant;
Anesthesia

Abstract Mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes syndrome is a progressive syndrome with variable involvement of multiple-organ systems. These patients require special consideration for preoperative optimization, intraoperative management, and postoperative care. The medical literature regarding perioperative management of these patients relies heavily on case reports. Here we present a novel experience providing care for a patient with mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes syndrome who underwent renal transplantation for focal segmental glomerulosclerosis and end-stage renal disease.
© 2016 Elsevier Inc. All rights reserved.

1. Case

A 33-year-old, 5-ft 5-in., 67-kg woman with mitochondrial myopathy, encephalopathy, lactic acidosis, and stroke-like episodes (MELAS), and history of focal segmental glomerulosclerosis with end-stage renal disease on hemodialysis presented for living unrelated renal transplant. The patient had hypertension managed with a β-blocker and angiotensin receptor blocker therapy. She also had diet-controlled type 2 diabetes mellitus and symptoms of sensorineural hearing loss likely related to maternally inherited diabetes and deafness, a condition associated with the same mitochondrial mutation as MELAS. [1] The patient was diagnosed as having MELAS due to high clinical suspicion for a mitochondrial mutation in the context of focal segmental glomerulosclerosis, diabetes, and sensorineural hearing loss. The patient had no significant history of cardiovascular disease, confirmed with a normal transthoracic echo and a normal Lexiscan stress electrocardiography and perfusion scan obtained within the 6 months prior to surgery. There were no other significant neurologic manifestations of MELAS.

Preoperative laboratories revealed anemia with hemoglobin and hematocrit levels of 10.3 g/dL and 29.6%, respectively. There was no evidence of coagulopathy. Sodium, chloride,

☆ Disclosures: The authors have no disclosures. The patient discussed here has reviewed the manuscript and given permission for publication.
☆☆ The patient discussed here has reviewed the manuscript and given permission for publication.

* Corresponding author at: Department of Anesthesiology, The Ohio State University Wexner Medical Center, 410 W. 10th Avenue, Columbus, OH 43210, USA. Tel.: +1 614 293 7308; fax: +1 614 293 8153.
E-mail address: michelle.humeidan@osumc.edu (M.L. Humeidan).

http://dx.doi.org/10.1016/j.jclinane.2016.05.025
0952-8180/© 2016 Elsevier Inc. All rights reserved.

magnesium, aspartate aminotransferase, alanine aminotransaminase, and bicarbonate were within normal limits; therefore, a preoperative lactate was not checked. Potassium was elevated (5.2 mmol/L), along with blood urea nitrogen (60 mg/dL) and creatinine (5.52 mg/dL). Fasting glucose was 112 mg/dL. The patient denied gastric reflux symptoms. Airway and pulmonary examination was unremarkable. Preoperative vital signs were as follows: blood pressure 137/102 mm Hg, heart rate 70 beats/min, temperature 36.6°C, and room air oxygen saturation with pulse oximetry 98%. The patient had no known drug allergies, and surgical history included tonsillectomy and dilation and curettage, for which the patient received general anesthesia without incident. A type and screen was completed with 2 units of packed red blood cells held for the operating room. A dialysis catheter provided intravenous access for the surgery.

Preinduction sedation was provided with 2 mg midazolam. Propofol and lidocaine were given for induction of anesthesia after placement of standard monitors. Neuromuscular blockade was initiated with 10 mg cisatricurium and no additional blockade was required during the case. The patient was maintained with sevoflurane and fentanyl boluses. An esophageal Doppler probe was placed after induction, and stroke volume variation and cardiac index were monitored. If the stroke volume variation was >15%, crystalloid was administered. Intraoperative fluid resuscitation was provided with normal saline and plasmalyte. An infusion of D10 was provided continuously to avoid hypoglycemia. Intraoperative blood glucoses were checked frequently; no insulin administration was required. Normothermia was maintained with a forced-air warming blanket. L-Arginine was immediately available for treatment of any stroke-like symptoms postoperatively.

The surgical procedure lasted 160 minutes. Throughout the case, plasma glucose levels were stable and never greater than 158 mg/dL. Early in the surgery, the patient developed a metabolic acidosis (pH 7.25, bicarbonate 16 mEq/L) requiring bicarbonate supplementation totaling 200 mEq. This was likely secondary to the renal failure and quickly resolved. The pH of the blood remained normal throughout the remainder of the surgery, and lactic acidosis did not develop. Electrolyte levels were monitored with frequent venous blood gases. Estimated blood loss was 50 mL. Fluid administration totaled 3 L. The patient was making urine at the conclusion of the surgery (800 mL). After confirmation of sustained tetany without fade following stimulation of the ulnar nerve, the patient was extubated at the end of the case and taken to the postanesthesia recovery unit. Her postoperative course is uncomplicated, and she is currently doing well. Her immunosuppression regimen includes everolimus and cyclosporine.

2. Discussion

MELAS is a variably progressive, multiorgan system syndrome. Inherited in a maternal pattern, mitochondrial DNA mutations result in disrupted electronic transport chain function, followed by pathologic response of high-energy organs to oxidative stress. The most common mutation is an adenine to guanine change at position 3243 in the mitochondrial genome. [2] Prevalence of this mutation is reported to be 236/100,000 by Manwaring and colleagues [3], almost 15 times greater than previous estimates. In addition to stroke and elevated lactate levels, ragged red fibers on muscle biopsy, seizures, and onset prior to 40 years of age are considered cardinal features, but MELAS symptoms can vary greatly between patients. Frequent manifestations include migraines, vomiting, limb weakness, and dementia, and less frequently peripheral neuropathy and functional cardiac abnormalities can be seen [4]. Medical management can include administration of coenzyme Q_{10} to treat oxidative enzyme deficiencies, free-radical scavenging with idebenone, use of L-arginine to promote nitric oxide–mediated vasodilation, and attenuation of lactate overproduction with dichloroacetate. Some patients may benefit from exercise training and ketogenic diets, although clear evidence supporting or refuting effectiveness of any of these therapies is lacking [2].

We conducted a PubMed search for "MELAS" and "anesthesia" which yielded 19 manuscripts; of these 6 were not in English and 4 provided no abstract. Of the remaining 9 articles, none specifically addressed anesthetic management in MELAS patients undergoing transplant surgery. In general, MELAS patients seem to tolerate commonly used anesthetics, which is reassuring because a case series by Gurrieri et al [5] reported on 20 anesthetics provided to 9 MELAS patients, 12 of which were done prior to syndrome diagnosis. Urine analysis is the most clinically useful method of mitochondrial DNA testing, but muscle biopsy remains critical for diagnosis in patients without an identifiable mutation [1]. Renal transplant is considered an intermediate-risk surgery, and optimal preoperative evaluation and anesthetic management warrants consideration of the systemic effects of MELAS syndrome and insight into the impact of surgical stress on MELAS patients. Electrolyte abnormalities and comorbid diabetes mellitus are not uncommon in patients having renal transplant surgery, and MELAS patients should have standard evaluation for these issues. However, cardiomyopathy and conduction abnormalities, gastroenterologic disturbances, stroke, and seizures in MELAS patients warrant special consideration [5,6,7]. In addition to documentation of preexisting neurologic deficits and thorough assessment of aspiration risk, consideration of advanced cardiac testing beyond electrocardiography seems prudent. Despite a good functional status, our patient underwent a stress electrocardiography and perfusion scan.

Case reports have indicated that MELAS patients may be at increased risk for malignant hyperthermia (MH), but the evidence is not clear. It is certainly reasonable to avoid triggering agents in MELAS patients [7]. Use of total intravenous anesthesia with propofol [8] with or without remifentanil has been described [9], but these reports used only 1 to 2 hours of total intravenous anesthesia, and prolonged infusion for a longer case duration could in theory put MELAS patients

Download English Version:

https://daneshyari.com/en/article/5884611

Download Persian Version:

https://daneshyari.com/article/5884611

Daneshyari.com