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Delayed Presentation of Gastric Perforation after Transesophageal Echocardiography for Cardiac Surgery

Wayne Soong, M.D., Sherif Afifi, M.D., F.C.C.M, F.C.C.P.,† Edwin C. McGee, M.D.‡*

TRANSESOPHAGEAL echocardiography (TEE) is an integral tool for intraoperative monitoring and diagnosis in patients undergoing cardiac surgery.[1] TEE remains a moderately invasive procedure with a very low incidence of complications, ranging from 0.2% to 1.2%.[2,3] The spectrum of complications has included injury to the gastrointestinal tract, obstruction of airways, dysrhythmias, hemorrhage, or entrapment of other upper airway tubes.[2–6]

A common concern is that placement of the TEE probe in the anesthetized patient eliminates signs of severe patient discomfort that might herald or result from damage to gastrointestinal tissues. Consequently, contraindications to insertion of a TEE probe have included extensive esophageal disease, such as strictures, masses, diverticula, or untreated varices, as well as recent gastric hemorrhage, ulcers, masses, or symptomatic hiatal hernias. A history of preexisting esophageal or gastric disorder usually alerts physicians to avert complications. We report a case involving delayed presentation of gastric perforation at a rare site by TEE probe after cardiac surgery.

Case Report

An 83-yr-old man was transferred to our institution with substernal chest pain. The initial workup revealed myocardial infarction and a coronary angiogram that showed significant three-vessel disease. A transthoracic echocardiogram revealed left-ventricular hypertrophy, a left-ventricular ejection fraction of 45%, and posterolateral akinesis.

The patient's medical history was significant for hypertension, atrial fibrillation, transient ischemic attacks, and prostatectomy. Medications before hospitalization included aspirin, atenolol, amlodipine, and benazepril, all of which were continued in the Cardiovascular Intensive Care Unit. A heparin infusion was started for his coronary occlusive disease, and esomeprazole was added for stress ulcer prophylaxis.

The patient was brought to the operating room for coronary revascularization. After induction of general anesthesia and tracheal intubation, an adult omniplane TEE probe (Omni III 21378A; Philips Medical Systems, Andover, MA) was inserted without difficulty. Imaging studies were easily obtained by both the resident and attending anesthesiologists. Three-vessel coronary artery bypass grafting, radiofrequency pulmonary vein isolation, and left atrial appendage stapling were performed, with an aortic cross-clamp time of 73 min and a cardiopulmonary bypass time of 103 min. The TEE probe remained *in situ* a total of 5 h, for the duration of the operation. Upon completion of surgery, the probe was removed without difficulty, with the tip in neutral position. Postoperatively, the patient was brought intubated and sedated to the Cardiovascular Intensive Care Unit.

On the second postoperative day, a routine chest radiograph showed significant pneumoperitoneum, which had not been present on previous studies. However, because the patient was asymptomatic and tolerating clear liquids, this finding was attributed to inadvertent intraoperative diaphragmatic injury, which had become radiographically evident only after removal of mediastinal drainage tubes. On the third postoperative day, the patient grew progressively disoriented. He also developed hypotension unresponsive to fluid resuscitation, eventually requiring vasopressin infusion. Despite continued absence of fever, anemia, leukocytosis, or abdominal pain, computed tomography of the abdomen demonstrated gross extravasation of oral contrast from the proximal stomach.

Broad-spectrum antimicrobials (ampicillin/sulbactam, metronidazole, and fluconazole) were started, and the patient was urgently taken to the operating room for laparotomy. Exploration revealed a 2-cm perforation at the lesser curvature of the stomach, near the gastroesophageal junction. The surrounding margins were clean, with minimal bleeding and no signs of ulceration or chronic granulation. The perforation was repaired with an omental patch, and a jejunostomy tube was inserted. The patient's postlaparotomy course was protracted, including mechanical ventilation for an additional 6 days and a total intensive care unit stay of 12 days. He subsequently developed intraabdominal sepsis, acute renal failure, and cognitive dysfunction, dying after 27 hospital days.

Discussion

Complications related to TEE use in cardiac surgery are infrequent, with upper gastrointestinal injury occurring in 0.04% to 1.2% of cases.[2,3] Bleeding is the typical presentation, diagnosed either upon withdrawal of the TEE probe or after finding anemia refractory to transfusion. Gastric perforation from TEE is an exceedingly rare event. In two studies reviewing data on a sum of more than 20,000 procedures, including patients undergoing TEE with conscious sedation, there were no incidents of gastric perforation.[4,5] Kallmeyer *et al.* [2] examined the complication rate in a series of 7,200 cardiac surgical patients and found gastrointestinal injury in 0.1%, the most severe of which resulted in esophageal perforation leading to hydropneumothorax. Esophageal abrasions and bleeding were the more common of the remaining complications.[2] More recently, Lennon *et al.* [3] reported six cases of "major gastrointestinal injury" in a series of 859 cardiac surgical patients, including three incidents of perforation at the cardia.

* Fellow, Critical Care Medicine, Northwestern University Department of Anesthesiology. † Associate Professor, Northwestern University Department of Anesthesiology, and Chief, Section of Critical Care Medicine. ‡ Assistant Professor, Northwestern University Department of Cardiothoracic Surgery. Surgical Director, Advanced Heart Failure Program of the Bluhm Cardiovascular Institute of Northwestern Memorial Hospital, Chicago, Illinois.

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Address correspondence to Dr. Soong: 251 East Huron Street, Feinberg 5-704, Chicago, Illinois 60611. w-soong@northwestern.edu.

Our case involved a late and unusual presentation of perforation near the gastroesophageal junction, in a patient without underlying gastrointestinal pathology. Because benign pneumoperitoneum after cardiac surgery is not uncommon,[7] it was only after the patient's clinical condition had deteriorated that computed tomography and subsequent laparotomy were performed. Based on the gross appearance of surrounding gastric tissue during laparotomy, the surgeon concluded that the injury was likely to have resulted from the TEE probe. The location and lack of underlying pathology were inconsistent with perforation from peptic ulcer disease.[8]

Transesophageal echocardiography can lead to upper gastrointestinal injury from a combination of two mechanisms.[6] Mechanical trauma may occur during probe insertion or manipulation, with upper esophageal injury being more common than lower esophageal or gastric.[5] Alternatively, sustained contact between probe and esophagus, as frequently occurs during cardiac surgery, can lead to pressure on the surrounding tissues, with the potential for ischemia and thermal injury.[6,9] Based on the operative findings, we suspect that the former mechanism was responsible for gastric perforation in our case.

Complications from TEE use are rare but potentially severe. The late and uncommon presentation of this case, in the context of previously reported incidents of trauma from TEE, underscores the need for a high index of suspicion after cardiac surgery. Postoperative complaints of dysphagia or odynophagia, as well as evidence of leukocytosis, fever, refractory anemia, or pneumoperitoneum, should be thoroughly investigated, even if significant time has elapsed between TEE and presentation.

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Antifibrinolytic Therapy Use to Mitigate Blood Loss during Staged Complex Major Spine Surgery: Postoperative Visual Color Changes after Tranexamic Acid Administration

Grant T. Cravens, M.D., Michael J. Brown, M.D.,† Daniel R. Brown, M.D., Ph.D., F.C.C.M.,† C. Thomas Wass, M.D.‡*

TRANEXAMIC acid (TEA) is an antifibrinolytic medication that inhibits plasmin and plasminogen activation, thereby promoting hemostasis and thrombosis. Since the 1960s, it has been used for a number of bleeding dyscrasias, including gastrointestinal hemorrhage, menorrhagia, epistaxis, urinary tract bleeding, hyphema, and hemophilia. It is also used for bleeding prophylaxis during many surgical procedures, including complex neuraxial surgery. We report an unusual case of color vision change after TEA administration.

Case Report

A 58-yr-old, 80-kg man presented in 2003 with new-onset back pain and was found to have a thoracic chordoma for which he underwent surgical resection. The patient underwent repeat surgery in 2004 for tumor recurrence. In 2006, leg weakness developed, and neuraxial imaging confirmed recurrence of his thoracic chordoma. Because of the progressive nature of his symptoms, he was scheduled for en bloc vertebral resection to be staged on two separate days.

Aside from his neurologic disease, the patient had no other significant medical history. After standard monitoring, general anesthesia was induced with intravenous midazolam and fentanyl, and muscle relaxation was achieved with vecuronium. After the patient's airway was secured, general anesthesia was maintained with an infusion of fentanyl, midazolam, and atracurium. Blood glucose was strictly controlled (goal 80–110 mg/dl) with an insulin infusion. Because of anticipated

* Resident in Anesthesiology, † Assistant Professor of Anesthesiology, ‡ Associate Professor of Anesthesiology.

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Address correspondence to Dr. Brown: Department of Anesthesiology, Mayo Clinic, 200 First Street, Southwest, Rochester, Minnesota 55905. brown.michael3@mayo.edu.

dissection through multiple previously operated tissue planes, tranexamic acid (10-mg/kg loading dose, 1-mg $\cdot$ kg^{-1} $\cdot$ h^{-1} infusion) was used to minimize blood loss and transfusion requirements. The patient was placed in pinions, was positioned prone, and underwent T4–T8 laminectomies, mobilization of the epidural tumor from T6 to T8, and posterior spinal instrumentation from T2 to T11. The total time in the prone position was 9 h. The total tranexamic acid dose was 1,360 mg, and the estimated blood loss was 550 ml. The patient was awakened at the conclusion of surgery, moved all his extremities to command, and was transferred to the intensive care unit. He was extubated the following morning and had an uneventful recovery.

The second stage of the patient's surgery was performed 38 h thereafter. Anesthesia was induced with midazolam and fentanyl, and muscle relaxation was achieved with vecuronium. General anesthesia was maintained with an infusion of midazolam, fentanyl, and atracurium. Tranexamic acid was again used (same dose as above). The patient was placed in the left lateral decubitus position (no pressure on eyes noted throughout case) and underwent en bloc tumor resection at T5–T7, spine stabilization with a titanium cage, and vascularized rib graft through a combined anterior (right thoracotomy) and posterior approach. In addition, a latissimus dorsi flap was used for hardware coverage. The total anesthesia time was 13 h. The total tranexamic dose was 1,840 mg, and the estimated blood loss was 1,300 ml. The patient was awakened, moved his upper and lower extremities to command, and was transferred to the intensive care unit sedated and mechanically ventilated. He remained hemodynamically stable, and the trachea was extubated the next day. After extubation, he was alert and oriented to person, place, and time; however, he reported color vision disturbances; specifically, all objects appeared green. As his color vision improved, his perception changed from objects being all green, to objects having a slight green tinge, to a green-tinged halo effect around objects that were now normal in color, to return of normal color vision. A formal ophthalmologic examination, including Ishihara color vision testing for both eyes, was normal. Unfortunately, this examination was performed after significant subjective improvement had already occurred in his color vision. By the second postoperative day, his color visual changes had dissipated. Throughout the remainder of his hospital stay, there was no recrudescence of his ophthalmologic symptoms. He was discharged to a rehabilitation facility on postoperative day 8.

Discussion

We present an unusual case of transient color vision change after TEA administration during major spine surgery. To our knowledge, such an occurrence has not previously been reported in the medical literature.

Tranexamic Acid and Blindness

It is known that TEA can cause ophthalmologic complications, including blindness. For example, two women receiving TEA for menorrhagia developed central venous stasis retinopathy-induced blindness after 1 week of therapy.[1] In both cases, visual acuity and funduscopic examinations returned to normal after concomitant TEA withdrawal and treatment with systemic corticosteroid and dipyridamole. In another case, a 56-yr-old dialysis patient experienced blindness on two separate occasions during prolonged TEA therapy for gastrointestinal hemorrhage management.[2] On both occasions, visual acuity returned after withdrawal of TEA, although some defect in night vision persisted. Because TEA is renally excreted, blindness was attributed to a TEA overdose in this particular case.[2] Whether the "TEA overdose" resulted in thrombosis of retinal circulation or a direct drug or metabolite effect on retinal cone cells remains uncertain. In yet another case, a patient experienced blindness while receiving TEA for hemorrhagic cystitis.[3] In all of the above cases, the etiology has yet to be fully elucidated. Although a thromboischemic mechanism would seem to be the most plausible explanation, there is no definite link between TEA and blindness.

Tranexamic Acid and Color Vision Change

In regard to color vision change, we were unable to find any case reports or clinical trials citing such observation. Interestingly, Micromedex (Thomson Micromedex, Greenwood Village, CO) cautions that "acquired defective color vision" is a contraindication to TEA use. However, Micromedex provides no substantiating laboratory or clinical evidence as to why this claim is made.

Color vision requires the intact function of several structures. Cone cells within the retina contain pigments that allow for differential responses to various light wavelengths. The optic tracts then transmit this data to the visual cortex of the occipital lobe. In addition, color vision is processed in area V4 of the temporal lobe. Disruption of function in any of these areas could presumably affect color vision.

In our case, ischemia as the cause of color vision change seems unlikely. An ischemic injury to any of the structures or pathways involved in color vision would also be expected to result in additional findings such as changes in visual acuity or visual field defects. Rather, the nature of our patient's morbidity was not total loss of color vision, but a green tinge and green halo effect. Visual changes (*e.g.*, transient or permanent blindness) have been reported in patients after major spine surgery.[4] More specifically, the American Society of Anesthesiologists Practice Advisory Board has reported prolonged surgical duration (*i.e.*, exceeding an average of 6.5 h) and substantial blood loss (*i.e.*, when loss reaches an average of 44.7% of estimated blood volume) as high-risk contributing factors to developing postoperative visual acuity changes after spine surgery.[5] Regardless of whether these criteria were fulfilled, our patient experienced a completely different entity in that visual acuity was spared. Although it is impossible to definitively identify a pathogenic link between TEA and color vision disturbance, we speculate that our observation could represent a pharmacodynamic effect on one or more of the pigments involved in color differentiation by retinal cone cells. The likelihood of a pharmacodynamic effect of some kind is supported by the fact that the manufacturer lists acquired color vision defects as a contraindication to TEA use.

Last, this patient received several other drugs during his anesthetic, none of which seem likely causes of his impairment. Midazolam is not known to cause color

vision problems *per se*. It does have hallucinogenic potential; however, our patient did not demonstrate any psychotic behavior, altered consciousness, or confusion associated with his color vision impairment that would indicate hallucination. Hallucinations due to fentanyl would be an unlikely cause for similar reasons. Other side effects of fentanyl do include blurred vision, but not color vision disturbance. Vecuronium and atracurium are not known to have visual side effects.

In summary, we report an unusual case of transient color vision disturbance after TEA administration in a patient undergoing staged complex major spine surgery. Although the etiology has yet to be fully elucidated, we speculate that our observations resulted from a pathogenic link between TEA and retinal cones.

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