Learning From Others:

A Case Report From the Anesthesia Incident Reporting System

Anesthesia Quality Institute
ANESTHESIA INCIDENT REPORTING SYSTEM (AIRS)

Detailed review of unusual cases is a cornerstone of anesthesiology education. Each month, the AQI-AIRS Steering Committee will abstract a case and provide a detailed discussion based on a submission to the national Anesthesia Incident Reporting System. Feedback regarding this item can be sent by email to **r.dutton@asahq.org**. *Report incidents to* **www.aqiairs.org**.

Case 2014-02: Win Some, Lose Some

"The great questions in medicine never change; the answers do with regularity."

– William Kelley, M.D., Chair, Department of Medicine, University of Michigan (1975-1989), and former CEO of the University of Pennsylvania Medical Center and Health System and Dean of the School of Medicine (1989-2000)

Case Presentation

A 55-year-old man with chronic low-back pain presented for an L4/5 laminectomy and fusion. Preoperative history included hyperlipidemia, sleep apnea, benign prostatic hyperplasia, chronic smoking and alcoholism. The patient was physically active and had a negative stress test two months prior. Preoperative EKG was normal. Medications included lorazepam, atorvastatin, amitryptaline, tamsulosin (Flomax), gabapentin and aspirin (stopped one week prior to surgery).

Anesthetic induction and intubation were unremarkable with propofol, succinylcholine, sevoflurane, fentanyl and vecuronium. The patient was turned prone and incision made. Anesthesia was maintained with sevoflurane. An hour into the operation, the patient experienced a short episode of self-limited ventricular tachycardia. ST elevation was also present; the surgeon was informed and the decision made to conclude surgery. Hypotension was treated with ephedrine, and FiO_2 increased to 1.0. Within a few minutes, ventricular tachycardia recurred. The patient was turned supine and CPR was initiated. The ACLS protocol was followed, and after defibrillation times 4, there was return of a pulsatile rhythm. Amiodarone and phenylephrine infusions were initiated. Transesophageal echocardiography demonstrated decreased cardiac contractility in an asymmetric pattern. A cardiology consultant recommended acute administration of bivalirudin and urgent transfer to the catheterization lab. The diagnosis was acute plaque rupture and thrombotic coronary occlusion. The patient underwent emergent angioplasty and placement of a stent, and subsequently recovered uneventfully.

Discussion

This case raises a number of interesting issues. The first is the very prosaic question of how to best manage the patient who suffers an acute coronary syndrome (ACS) intraoperatively; next, the question of whether the patient was appropriate worked up prior to the anesthetic; and finally, the question of the impact on the anesthesia team involved (the "second victims").

Perioperative myocardial infarction occurs in <0.5 percent of surgical patients without known coronary artery disease (CAD) and in 1-2.4 percent of those with a prior diagnosis.[1] The ability to infallibly identify patients at risk during the anesthesia preoperative evaluation process would obviously be of benefit but, as this case illustrates, we are not there yet. The treatment of an evolving or suspected myocardial infarction is outlined in the 2010 American College of Cardiology/American Heart Association (ACC/AHA) guidelines for management of ACS[2] and includes call for help, monitor, oxygen, prepare for CPR and defibrillation, administer aspirin and obtain 12 lead ECG. The clinicians in this case were fortunate that the surgeon was able to rapidly complete the procedure and turn the patient prior to his VF arrest. In cases where the patient arrests in the prone position, neither CPR nor defibrillation should be delayed, as both have been shown to be effective even in that position.[3-5]

Antiplatelet and thrombolytic agents are the cornerstone of early treatment of an ST segment elevation myocardial infarction (STEMI), but administration during and immediately postoperatively may result in extensive bleeding. Immediate consultation between the surgeon, cardiology and anesthesiology is critical, both to determine pharmacological treatment and to optimize decision-making regarding next steps.

Clearly, prevention of an MI is preferable to even the best intraoperative management, but the ideal approach to identification of the patient at risk has been steadily evolving for the past four decades. The anesthesiologists in this case followed the well-researched and well-defined evidence-based Guidelines for the Perioperative Management of the Cardiac Patient for

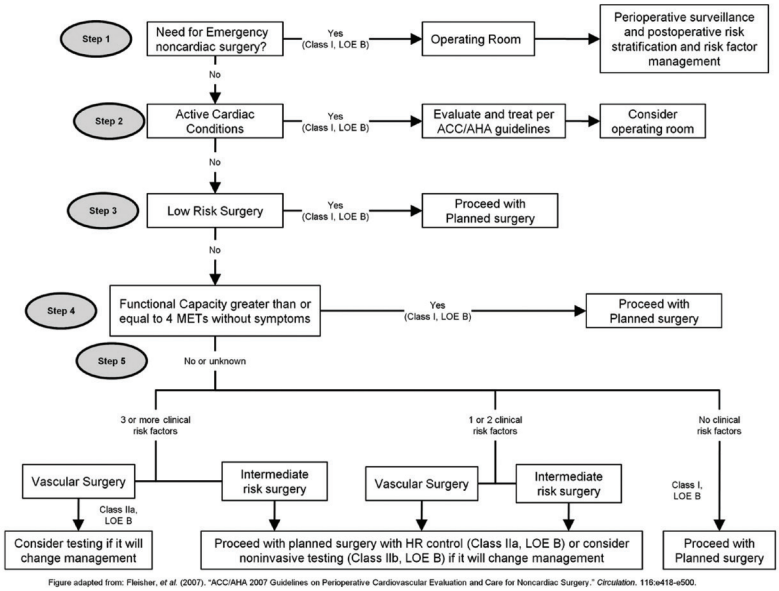

Figure 1: Algorithm used in the 2009 ACC/AHA Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery.[6]

Non-Cardiac Surgery, which state that no further workup is necessary in the patient with a negative stress test.[6] Indeed, even if this patient had known CAD with stable angina and a positive stress test, no further work-up or intervention would be indicated prior to this intermediate-risk surgery unless severe three-vessel or left main disease was suspected. Patients with acute cardiac conditions (acute myocardial ischemia due to current or recent MI, severe aortic stenosis, acute heart failure and certain unstable arrhythmias) clearly need work-up; management of the rest is as defined in Figure 1 of the ACC/AHA 2007 Guidelines on Perioperative Cardiovascular Evaluation and Care for Noncardiac Surgery: **circ.ahajournals.org/content/116/17/e418.full.pdf+html?sid=0b04e74c-078a-4378-8117-0620850f000d.**[6] (Used with permission from *Circulation*. 2007; 116:e418-e500. ©2007 American Heart Association, Inc.)

This guidance comes from evidence that preoperative revascularization of patients with known CAD does not improve outcomes over medical management. The CARP study randomized 510 patients with CAD (including three-vessel disease, but excluding left main) to undergo revascularization or not prior to major vascular surgery: there was no difference in immediate, 30-day, or two-year mortality or major adverse cardiac events.[7] The potential benefit of preoperative intervention is even less well supported for lower-risk surgeries such as this spine operation.

The absence of benefit of revascularization in the face of known CAD may be explained, first, by the overall skill with which anesthesiologists manage perioperative stressors and, second, by the nature of perioperative MI. Ischemia can occur due to global or regional excess oxygen demand versus supply (Type 2 MI)[8] as triggered by hypertension, increased wall tension, tachycardia, hypoxia, anemia, hypovolemia or hypotension. Anesthesiologists can and do manage these conditions both intra- and postoperatively. However, ischemia can also occur with acute plaque rupture and thrombosis formation with abrupt closure of the vessel, as occurred in this patient.[8] These events occur more often in the operative setting due to increased shear forces (hypertension, tachycardia) and are aided and abetted by hypercoagulability and the systemic inflammation associated with major surgery. Which plaques are vulnerable cannot be predicted with accuracy, as a patient with both 40 percent and 80 percent lesions is actually more likely to have severe ischemia related to abrupt closure of the 40 percent lesion (the 80 percent will have collateral support while the 40 percent will not).[9] While the lesser stenosis is more likely to be the culprit in a perioperative MI, there is no basis for revascularization of these minor lesions.

The reporter in this case noted the event as "preventable," but this may represent hindsight bias. Anesthesiologists should manage the hemodynamic issues that can precipitate an MI, but the triggers of inflammation and hypercoagulation are poorly understood. The extent of surgical tissue trauma, the duration of the operation and the patient's genetic predilection all play a role. Interventions that have been tested to decrease the incidence of perioperative MI include prophylactic revascularization (no benefit, as described above), aspirin, beta-blockers and statins. Beta-blockers have been and continue to be the most controversial. Mangano kicked off the debate in 1996 when he showed that preoperative atenolol reduced not only perioperative mortality but six-month mortality as well.[10] In 2007, the POISE study was conducted, in which 8,331 patients with cardiac disease undergoing noncardiac surgery were randomized to receive metoprolol or placebo two to four hours prior to surgery and to continue through the postoperative period.[11] The cardiac event rate was 5.8 percent in the metoprolol group versus 6.9 percent in the placebo group (p=0.4). However, overall mortality was greater in the metoprolol group at 3.1 percent versus 2.3 percent, largely due to a doubling of the stroke rate in the beta-blocked patients.

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These data have been supported through several smaller trials and meta-analyses.

The impact of metoprolol in POISE may have been driven by the relatively large dose used or to the fact that metoprolol was tested. There is growing evidence that not all beta-blockers are created equal. Atenolol has been shown to have a better risk profile than metoprolol,[12] and a recent observational study showed that while metoprolol is associated with an increased stroke rate, bisoprolol and atenolol are not, presumably due to the cardio-selectivity of the latter agents.[13] At this point, the guidelines recommend continuation of beta-blockers in patients already on them, but to not initiate them in the preoperative period. Beta-blockers should still be used to manage acute hypertension and tachycardia perioperatively in patients at risk for myocardial supply and demand imbalance.

Finally, this case brings up the question of the "second victim" in such events. Most anesthesiologists will experience at least one catastrophic event in the course of their careers, and the emotional impact of such an event is now recognized, but few resources are currently available to the clinicians involved. Ganzoni's survey found that nearly as many anesthesiologists involved in non-preventable events felt personally responsible as those involved in preventable events.[14] Worse yet, the reporting anesthesiologist in this event marked it as preventable, when it clearly was not, indicating that s/he felt personally responsible for an event experts would classify as "not preventable."

Conclusion

Medical practice, and human behavior in general, is based on frequency gambling. Evidence-based guidelines are developed on the likelihood of an event occurring in a population and provide little information about a specific patient's outcome. In addition, they are based on what we know now, and thus change regularly as our knowledge increases. In many cases, as with this patient, even strict adherence to evidence-based best practices does not guarantee a perfect outcome. Clinical vigilance remains important, as well as the anesthesiologist's ability to rescue the patient from unusual life-threatening events. Finally, we must acknowledge the emotional impact such events have and build robust support systems to provide critical resources – by the numbers, we will all need this support at one time or another.

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