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Hospitalist Update

Perioperative Pulmonary Complications: Risk & Prevention

Robert Folzenlogen MD

We hospitalists are often asked to provide perioperative risk assessments and much of our focus is on cardiovascular risk. Yet, perioperative pulmonary complications increase the length of hospital stay twice as much as cardiovascular complications; indeed, the development of postoperative pulmonary complications increases the LOS six times the duration expected for a surgical procedure.

While the occurrence of these complications varies widely, depending on the type of procedure and the underlying condition of the patient, the overall rate is in the neighborhood of 6.8%. Most studies have defined the primary postoperative pulmonary complications to be atelectasis, pulmonary infection, bronchospasm and respiratory failure, any of which might lead to prolonged mechanical ventilation.

Our ability to assess risk for perioperative pulmonary complications (PPC) is a bit more subjective than it is for cardiovascular complications but the following have been consistently found to be **independent PPC risk factors for non-cardiac surgery patients**:

1. Age of 65 or greater
2. A history of COPD: increases risk by a factor of 2-6; 56% of these patients have PPC if undergoing major abdominal surgery, 38% have PPC if the surgery lasts over 2 hours and 73% have PPC if the procedure exceeds 4 hours
3. A history of CHF
4. Functional dependence
5. ASA Class II or greater
6. Hypoalbuminemia (< 3.5) - Gibbs et al. [1] showed that this is the best predictor of morbidity and mortality in the 30 days post surgery, the best predictor of postoperative infections and sepsis (quadruples the pneumonia rate) and is associated with a dramatic increase (5X) in the failure to wean.



Other **factors which increase the risk for PPC** (in non-cardiac surgery patients) but have **not been found to be independent** include: chronic tobacco and/or alcohol use, altered mental status, a weight loss of >10% in 6 months, a history of CVA, the need for perioperative transfusion and a high or low BUN. Any additional risk from documented obstructive sleep apnea remains controversial but obesity, controlled asthma and a history of cardiac arrhythmias do not appear to augment risk. Current evidence suggests that the **added risk of chronic tobacco use is reduced only if cessation occurs at least 6-8 weeks prior to the surgery.**

Procedure-related risk for PPC is significant and includes the following:

1. Surgery lasting more than 3 hours
2. Emergency surgical procedures
3. AAA and other vascular procedures
4. Thoracic and upper abdominal procedures (especially esophagectomy)
5. Neurosurgical procedures
6. Neck surgery
7. Need for General Anesthesia
8. Use of long-acting neuromuscular blockade

Note: Cardiac surgery dramatically increases the risk of PPC but is beyond the scope of this discussion

The only significant effort to establish an **index for PPC risk** in non-cardiac surgery patients was by Arozullah et al in 2000, a prospective study of 81,719 patients undergoing major non-cardiac surgery [2]. The index is heavily weighted by the nature of the procedure itself (with an AAA receiving the most points); once again, a low serum albumin received the most points of any non-procedure factor. Use of this index, which includes all of the independent risk factors and procedure related risks listed above, is controversial but, to date, no more reliable tool is available. However, over the past decade, **evidence seems to be emerging that the presence of documented pulmonary hypertension or interstitial lung disease may prove to be independent risk factors for PPC .**

For patients undergoing non-cardiac surgery, the following **preoperative tests/procedures** have not been shown to be helpful in assessing the risk for PPC:

1. CXR—useful only if new, acute symptoms are present
2. Spirometry or PFTs - useful only in efforts to maximize control of COPD/asthma exacerbations prior to surgery or to assess expected tolerance of planned lung tissue resection
3. ABGs
4. Right heart catheterization - this recommendation may change in light of newer evidence related to the presence of pulmonary hypertension
5. TPN or enteral supplementation

Postoperative efforts to reduce PPC are limited but should include the following:

1. Incentive Spirometry
2. Early Ambulation
3. Use of CPAP as indicated
4. Adequate pain control to prevent splinting and atelectasis
5. Avoiding placement of NG tubes which increase risk of aspiration
6. Maintenance of bronchodilator regimen for patients with COPD or asthma
7. Appropriate VTE prophylaxis

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CASE REPORT Molly Lewandowski, MD & Samantha Nohava, Medical Student, UMKC

SPLENIC INFARCTION

Splenic infarction is a rarely encountered disorder, usually occurring as a complication of another disease process. Due to the distribution of the splenic vasculature, infarcted areas are limited to specific segments of the spleen and rarely extend to all of the parenchyma. Wedge-shaped, hypodense regions are the characteristic appearance of splenic infarction on imaging studies.

CASE:

A 57 year old African American male presented to Truman Medical Center with a five day history of severe left upper quadrant abdominal pain, radiating to his left shoulder and neck. He also complained of nausea, dyspnea and mild chest pain but denied fever; the patient had visited the ER two days prior with similar complaints and his symptoms were partly relieved with a GI cocktail and ranitidine. However, the above symptoms worsened and he returned for further evaluation. His past medical history was remarkable for hepatitis C, hypertension, IV drug use, chronic tobacco use, BPH and a history of medication noncompliance.

On presentation, his vital signs revealed a BP of 170/79, P 90, R 20 and a temperature of 98 F. Labs demonstrated a leukocytosis of 12.2 but were otherwise unremarkable. A CXR had findings suggestive of COPD and an EKG was normal. A CT of the abdomen with contrast was obtained; this showed a medial upper pole wedge-shaped area of hypodensity in the spleen, consistent with a splenic infarct (**image on the next page**). Given his history of IV drug use and his current leukocytosis, blood cultures were obtained and a trans-thoracic echocardiogram was ordered; the latter was essentially normal and did not demonstrate evidence of endocarditis. The blood culture grew only a non-Bacillus species that was determined to be a contaminant. Additional testing included a monospot and lupus anticoagulant, both of which were negative.

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