# MISSOURI HOSPITALIST SOCIETY

# MISSOURI HOSPITALIST

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Photos: South Platte River, Colorado

# 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 pro-

cedure 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**:

- 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
- 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 <u>not</u> 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

#### **REFERENCES:**

- 1. Gibbs, J et al., Preoperative serum albumin level as a predictor of operative mortality and morbidity; results from the National VA Surgical Risk Study, Archives Surgery 1999; 134:36-42
- 2. Arozullah, AM et al., Multifactorial risk index for predicting postoperative respiratory failure in men after major non-cardiac surgery: The National VA Surgical Quality Improvement Program, Ann Surgery 2000; 232:242-253
- 3. Bapoje, S et al., Preoperative Evaluation of the Patient with Pulmonary Disease, CHEST, 2007, Vol 132, No. 5, 1637-1645
- 4. Qaseen, A et al., Risk Assessment for and Strategies to reduce Perioperative Pulmonary Complications for patients undergoing non-cardiothoracic surgery: A Guideline from the ACP; Ann Int Med 2006; 144:575-580

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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During the hospitalization, further history was obtained which revealed the potential etiology of his infarction. One week prior to admission, while injecting a drug, he missed the vein and removed the needle, noticing a clot in the syringe; nevertheless, he proceeded to inject the drug into another vein and his presenting symptoms developed within 12 hours of that event. The patient remained afebrile throughout his hospital course; he was treated with IV saline, morphine and oxycodone-acetaminophen for pain control and lisinopril for his hypertension. His pain improved significantly over the next 72 hours and he was discharged on oral analgesics.



SPLENIC INFARCTION MARKED BY ARROW

#### **DISCUSSION:**

Splenic infarcts are usually limited to one segment or pole of the organ because the lobar arteries that supply the spleen do not anastomose with one another, thus giving rise to lobes known as segments. For this reason, conservative surgery of the spleen is possible, when indicated.

A literature review turned up multiple possible causes for splenic infarction including coagulation disorders such as antiphospholipid syndrome, autoimmune disorders such as Wegener's granulomatosis, and infectious causes such as HIV, CMV, aspergillosis, EBV, salmonella and malaria. Splenic infarction has also been described as a complication of pancreatitis, a microvascular complication of diabetes mellitus or a consequence of systemic emboli. Laxity of ligaments supporting the spleen can lead to a "wandering spleen" which is another cause of infarction, resulting from torsion.

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The classic presentation of splenic infarction includes left upper abdominal pain, nausea, vomiting and early satiety. CT angiography with contrast is the modality of choice for diagnosing splenic infarction, revealing a hypodense, wedge-shaped region as illustrated above. Leukocytosis and anemia are commonly found. Standard management of splenic infarction includes hydration, oxygenation and pain control. Depending on the size of the infarct, symptoms typically resolve within 7-14 days.

Complications of splenic infarction include abscess formation, pseudocyst development, hemorrhage, subcapsular hematoma or splenic rupture; however, all of these complications are uncommon. In the majority of cases, the ischemic tissue undergoes fibrosis and heals completely, thus precluding the need for splenectomy. Preserving the spleen is especially important due to its role in preventing infections; following splenectomy, patients are at significant risk for overwhelming infections, including sepsis, from encapsulated bacteria such as Strep pneumonia, Haemophilus influenza and Neisseria species; patients who have massive splenic infarcts and/or must undergo splenectomy should thus be vaccinated against these organisms.

**CONCLUSION:** This case highlights a rare complication of IV drug abuse and demonstrates the value of CT angiography in the diagnosis of splenic infarction. As discussed above, splenectomy is not often necessary and should be avoided to prevent medical complications associated with hyposplenism.

#### REFERENCES:

Devitt et al., An Unusual Cause of Abdominal Pain, Ir Med J, 2005 Mar; 98(3): 88-89

Salvi et al., Splenic Infarction, Rare Cause of Acute Abdomen, only seldom requires splenectomy, Ann Ital Chir., 2007; 78: 529-532

 $Ahmet\ et\ al.,\ A\ rare\ cause\ of\ Acute\ Abdomen:\ Splenic\ Infarction,\ Hepato-gastroenterology\ 2001;\ 48(41):1333-6$ 

Edwards et al., Acute Splenic Infarction, CMAJ 2006 Aug; 175(3): 247

# HOSPITAL MEDICINE VIRTUAL JOURNAL CLUB

# WASHINGTON UNIVERSITY SCHOOL OF MEDICINE

Abstracts & Full Links from recent journals of interest to Hospitalists

http://beckerinfo.net/JClub

### **NEW INSIGHTS ON AORTIC STENOSIS**

DR. ALAN ZAJARIAS

DIVISION OF CARDIOVASCULAR DISEASE, WASHINGTON UNIVERSITY SCHOOL OF MEDICINE SOCIETY OF HOSPITAL MEDICINE, ST. LOUIS CHAPTER, FEBRUARY 23, 2012

See listing in Hospitalist Calendar, page 7

# FROM THE JOURNALS

KATE AHMED, MD

#### Clostridium difficile infection in patients with inflammatory bowel disease

Saidel-Odes, L et al., Annals of Gastroenterology, North America, November 24, 2011

http://www.annalsgastro.gr/index.php/annalsgastro/article/view/1010/739

This review focuses on the epidemiology, pertinent clinical aspects, standard and newer diagnostic methods, established and novel therapies and prevention of infection. Emphasis is placed on clinical awareness, rapid detection and appropriate therapy.

# Rheumatological manifestations in inflammatory bowel disease

Voulgari, P, Annals of Gastroenterology, North America, July 24, 2011

http://www. Annalsgastro.gr/index.php/annalsgastro/article/view/915/720

Rheumatologic manifestations of inflammatory bowel disease are frequent and include peripheral arthritis, axial involvement, peripheral enthesitis, secondary osteoporosis and hypertrophic osteopathy; septic arthritis may also be a complication. This article discusses diagnositic and treatment modalities as well as potential adverse effects of the therapeutic agents.

#### Alcoholic Liver Disease: Pathogenesis and New Therapeutic Targets

Bin Gao, Ramon Bataller, Gastroenterology, Vol 141, Issue 5, November, 2011, pages 1572-1585

http://www.gastrojournal.org/article/S0016-5085(11)01228-5/abstract

Hepatic cirrhosis is the 12th leading cause of death in the U.S. and 48% of cases are alcohol related. This article reviews new data on the pathogenesis of alcoholic liver disease and identifies some promising therapeutic targets.

#### AGA technical review on the evaluation of liver chemistry tests

Green, RM and S Flamm, Gastroenterology, Vol 123, Issue 4, October 2002, pages 1367-1384

http://www.gastrojournal.org/article/S0016-5085(02)00241-X/abstract

Abnormal liver chemistries occur in 1-4% of asymptomatic patients. There interpretation must be in context with the patients history, risk factors, physical findings and other lab data.

# **ID CORNER**

WILLIAM SALZER, MD

C DIFF....AGAIN

The AHRQ commissioned a systemic review of C diff. Bottom line....vancomycin (\$1300) is probably not better than metronidazole (\$20); also, fidaxomicin (\$3400) appears to reduce relapse vs vancomycin for non-NAP1 strains.

Drekonja, DM et al., Comparative effectiveness of Clostridium difficile treatments. A systemic review. Annals Intern Med 2011; 155:839-847

http://www.annals.org/content/155/12/839.full.pdf+html

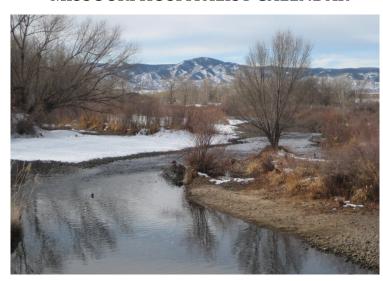
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# MISSOURI HOSPITALIST SOCIETY

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# MISSOURI HOSPITALIST CALENDAR



**New Insights on Aortic Stenosis,** Dr. Alan Zajarias, Cardiovascular Division, Washington University School of Medicine, Thursday, February 23, II Bel Lago Restaurant, 11631 Olive Blvd., Creve Coeur, MO. Hors d'oeuvres at 6:30, Dinner at 7:30 PM. RSVP to mrussell@dom.wustl.edu or call 314-362-1707; we hope you can join us **LOCAL** 

**Fundamentals of Mechanical Ventilation for Providers**, February 23, Northbrook, Illinois; register via www.chestnet.org

**Hospital Medicine 2012**, Society of Hospital Medicine, April 1-4, San Diego Convention Center; register via www.hospitalmedicine.org

**American Geriatrics Society Annual Meeting**, May 2-5, Seattle, Washington; register via www.americangeriatrics.org

Please direct all comments, ideas and newsletter contributions to the Editor:

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Please forward this newsletter to Hospitalists that you might know!