MISSOURI HOSPITALIST SOCIETY

Publisher:

Division of General IM

University of Missouri

Columbia, Missouri

Editor:

Robert Folzenlogen MD

Inside this issue:

MISSOURI HOSPITALIST

Issue 38 May-June, 2011

Hospitalist Update

Refeeding Syndrome & Hospitalists

Robert Folzenlogen MD

While refeeding syndrome was first described in prisoners of war and primarily studied in patients with anorexia nervosa, hospitalists encounter a wide range of patients who are at risk of this condition. Chronic alcoholics, debilitated patients and those with malignancies, chronic pancreatitis, malabsorption syndromes, inflammatory bowel disease, chronic malnutrition, AIDS , bariatric surgery or a complicated postoperative course are among those who might develop clinical problems associated with the refeeding syndrome.

Starvation, characterized by a diminished carbohydrate intake, results in reduced insulin production; while glucagon release is triggered, hepatic glycogen stores are depleted within 72 hours and the human body shifts to gluconeogenesis via utilization of muscle protein and adipose tissue. This process depletes intracellular phosphate, in concert with other electrolytes; due to homeostasis, the serum phosphate level may not reflect the severity of the intracellular depletion. Once carbohydrate intake is resumed, insulin release triggers an increased cellular uptake of phosphate, potassium and magnesum, which may produce a dramatic fall in the serum level of these electrolytes. This anabolic surge also increases the demand for thiamine and other vitamins and minerals which are already deficient in malnourished patients.

Early clinical signs of the refeeding syndrome may be subtle but, if they are not promptly addressed, lead to rhabdomyolysis, respiratory failure, cardiac arrhythmias, heart failure, seizures, coma and sudden death. This cascade of physiologic dysfunction usually begins within 4 days of refeeding and is often associated with a serum phosphate level below 0.50 mmol/L.; hypokalemia and hypomagnesemia are usually observed as well. The mainstay of treatment is prevention and the following recommentations should be considered in patients at risk of this condition:

1. Consult a nutritionist to assist with your refeeding protocol
2. Feeding should be restarted at no more than 25-50% of the patient's estimated daily caloric requirement (a maximum intake of 10 kcal/Kg/day); note that the syndrome

(continued) may develop in patients receiving either parenteral or enteral feedings.

3. Closely monitor the serum phosphate, potassium, magnesium, calcium, BUN and creatinine in patients at risk of refeeding syndrome and supplement accordingly; IV supplementation of phosphate is critical and may require 50 mmol/24 hours for several days.
4. Thiamine, at 200-300 mg/day, should be administered for at least 10 days; additional vitamin and mineral supplementation should also be provided
5. Leukocyte dysfunction is common in the early stages of refeeding syndrome and it is important to watch for signs of infection
6. In patients with acute or chronic renal failure, keep in mind that the serum phosphate may give false reassurance that phosphate repletion has been adequate
7. Patients with refeeding syndrome are at substantial risk for volume overload due to the effect of insulin on the renal tubules (causing fluid retention) and the presence of LV dysfunction; close monitoring for this complication is warranted.

Unfortunately, randomized-controlled studies for the management of refeeding syndrome are lacking. However, it is known that the risk for its development is greatest within the first 2 weeks of refeeding and that cardiac complications are the most common cause of mortality. Developing an appreciation for the potential development of refeeding syndrome and making every effort to avoid its occurrence should be the primary goals of the hospitalist.

REFERENCES:

1. Mehanna, HM et al., Refeeding Syndrome: What it is and how to prevent and treat it, BMJ 2008; 336: 1495
2. Crook, MA et al., The importance of Refeeding Syndrome, Nutrition 2001; 17: 632
3. Bermudez, O. and S. Beightol, What is Refeeding Syndrome?, Eating Disorders 2004; 12: 251

CASE OF THE MONTH Myriam Ensling MD

ABDOMINAL PAIN & NORMAL TEST RESULTS

A 56 year old Caucasian female was admitted to the IM service with complaints of dyspnea and LUQ abdominal pain. Her past medical history included COPD, MDD and lung cancer; she had undergone surgical excision of a left upper lobe lung nodule six weeks prior to this admission. She denied other trauma, fever or sick contacts prior to admission and her CXR was normal except for an elevated left hemi-diaphragm. A KUB was WNL and a CT of the chest revealed scarring vs. atelectasis at the left base, a trace left pleural effusion and no evidence of consolidation. Admission labs were remarkable for WBC 11.5 with 94%granulocytes, Hgb 12.9, Na 134, Cl 97, glucose 145, albumin 3.3 and a serum lactate of 2.3 mmol/L. Her ESR, D-dimer, cardiac enzymes, liver panel, UA, serum amylase and serum lipase were all within normal limits. Physical exam revealed T 36.8C, P 99, R 18, BP 114/79 and O2 saturation of 94%. Chest exam was remarkable for minimal wheezing and abdominal exam revealed marked tenderness of her LUQ and epigastric area; guarding was noted and the patient specifically asked not to be palpated in those areas. She consistently rated her pain at 10/10 and reported only mild relief with opioid medication.

The patient was treated for COPD exacerbation with oral steroids and antibiotics and was eventually transferred to the Psychiatric Unit for treatment of depression. There, she continued to complain of severe pain and refused to eat. As a result, she was transferred back to the IM Service where rib films demonstrated a displaced fracture of the left, lateral seventh rib.

DISCUSSION: A patient presenting with abdominal pain is assessed in accordance with their symptoms and their management is based on physical, laboratory and radiologic findings, which may point toward infectious, inflammatory or obstructive processes. When these studies are normal and the physician is unable to arrive at a specific diagnosis, the patient may be categorized as having a somatization disorder or experiencing medically unexplained symptoms (MUS). The medical literature includes an increasing number of studies that address the prevalence of MUS in primary care [1,2].

There are a number of conditions that cause abdominal pain but may present with normal basic testing (CBC, CMP, amylase, lipase, UA, cardiac enzymes CXR, KUB, CT of abdomen); physicians must keep these conditions in mind since specific tests will be necessary to insure an accurate diagnosis. The following tables provide an overview of these clinical diagnoses, grouped by localization (upper abdomen, lower abdomen, localized or diffuse):

Causes of upper abdominal pain with non-diagnostic basic tests

Condition	Diagnosis
CAD	EKG, Cardiac enzymes
Gastritits/ duodenitis/ gastroparesis	Trial with PPI?/EGD/ gastric emptying studies
Sphincter of Oddi dysfunction (usually RUQ pain)	Clinical: intermittent severe pain that improves on its own or with opiates. Intermittent mild elevation of bilirubin, liver enzymes, recurrent pancreatitis Imaging: HIDA scan, Gold standard: ERCP with Sphincter of Oddi manometry
Rib fracture (RUQ or LUQ)	X ray of rib cage
Slipping rib syndrome (RUQ or LUQ)	Hypermobile, subluxing 8th to 10th ribs, Dx physical examination

Causes of lower abdominal pain with normal basic tests

Gender	Condition	Diagnosis/clinical characteristics/ treatment
Male	IBD: Crohn's disease (usually RLQ pain)	Colonoscopy
	Pfannesnstiel Incision (hiliohypogastric nerve entrapment)	P Exam: Scar of prostatectomy, inguinal hernia, appendectomy Carnett's sign** /test Local Treatment: local injection with anesthetics and corticosteroids, effective 65-70% of the cases
	Traction symphysitis or pubalgia	Athletes, positive findings on MRI or scintigraphy
Female	IBD: Crohn's disease (usually RLQ pain)	Colonoscopy
	Ovarian and fallopian tube torsion	Abdominal pain with pelvic mass. Needs prompt diagnosis to avoid ovarian ischemia. Dx: Doppler Pelvic US, laparoscopy. Tx de-torsion with laparoscopy
	Endometriosis	Severe abdominal pain with menstrual period (Dysmenorrhea) dyspareunia, / Dx Laparoscopy
	Mittelschmertz	Pain in the middle of menstrual cycle. Tx: NSAIDs
	Pfannesnstiel Incision (hiliohypogastric nerve entrapment)	P Exam: Scar C section, inguinal hernia , appendectomy Carnett's sign** /test Local Treatment: local injection with anesthetics and corticosteroids, effective 65-70% of the cases
	Traction symphysitis or pubalgia	Athletes, positive findings on MRI or scintigraphy

Causes of abdominal pain localized to a discrete area of the abdomen with non-diagnostic basic tests

Condition	Diagnosis/characteristics
Acute	
Herpes Zoster	Typical Clinical skin findings (may be delayed) Tx: antivirals
Myofascial pain syndrome	Myofascial strain, history of trauma
Chronic	
ACNES (abdominal cutaneous nerve entrapment syndrome)	Prominent abdomens, obesity, pregnancy Abdominal pain localized on cutaneous nerve foramina on lateral border of rectus abdominis muscles (T7-T12) Positive Carnett's sign** Treatment: local injection with anesthetics and corticosteroids, effective 65-70% of the cases
CAWP (chronic abdominal wall pain)*	Usually localized on scars. Positive Carnett's sign** Treatment: local injection with anesthetics and corticosteroids, effective 65-70% of the cases
Thoracic lateral cutaneous nerve entrapment	Surgical scar on T7-T12 cutaneous nerve territory; positive Carnett's sign** Treatment: local injection with anesthetics anesthetics and corticosteroids, effective 65-70% of the cases
Thoracic nerve radiculopathy	Pain along T7-T 12 territory. Dx: nerve conduction studies i.e. diabetic neuropathy of thoracic nerves. Tx: amitriptyline, gabapentin, etc
Thoracic disc syndrome	T7-T 12 nerve root compromise on thoracic spine: Dx: x ray, MRI, Tx: specific cause

Causes of diffuse abdominal pain with non-diagnostic basic tests

Surgical		
Condition	Clinical characteristics	Diagnosis/treatment
Ischemic colitis **(may be fatal if not diagnosed promptly)**	History of chronic postprandial periumbilical pain or intestinal "angina", weight loss, patient refuses to eat due to pain. Pt. at risk: vasculopaths, PVD, CHF, A fib, elderly patient, pt in ICU on vasopressors	Gold-standard: Angiogram Other: MRA of mesenteric vessels, mesenteric duplex ultrasonography. CT scan with IV contrast for mesenteric venous thrombosis
Celiac artery compression syndrome	Chronic abdominal pain: Postprandial pain or intestinal "angina", weight loss	Mesenteric duplex ultrasonography. Angiogram
Medical		
Medication related	Opiates: OBD/ opiate related chronic abdominal pain/ NBS Other: digitalis, topiramate, quietiapine, lamotrigine, duloxetine, ziprasidone, etc.	Tx: Decrease dose or DC medication, use transdermal opiates, Methylnaltrexone Reduce dose or DC medication
Irritable Bowel	Crampy pain and altered bowel habits. Relieved with defecation, abdominal bloating. Does NOT awake pt from sleep and does NOT cause weight loss.	Dx: Exclusion of other conditions,
Sickle cell crises	DD: Pain crisis vs opiate bowel disease vs Constipation	Dx: Clinical Tx of underlying condition
Acute intermittent porphyria (AIP)	Age 30-40. Acute intermittent attacks of abdominal pain with N/V, acute neuropathy, mental disturbances., Sometimes: seizures, HTN, tachycardia and hyponatremia. Could be life threatening, may become chronic with intermittent attacks of pain.	Dx: Urine porphibilinogen PBG Treatment: carbohydrate loading (glucose 10%), hemin, opiates, ondasentron, etc.

***CAWP (Chronic abdominal wall pain):** present in 10-30% of patients presenting with chronic abdominal pain; often confused with visceral pain. Diagnosed in 5-10% of patients referred to gastroenterologists and in 20% of patients admitted to surgical services for nonspecific abdominal pain [4]. More common in women (4:1); all ages may be affected but peak incidence from 30-50 years; workups for this condition have cost $900-$6700 [4] even though it can often be treated inexpensively in a physician's office. The local injection of 0.5-1.0 ml 2% lidocaine or 2 cc of 0.25 bupivacaine plus 20-40 mg triamcinolone have been used for CAWP and ACNES with a high rate of success [5]. Neurolysis and fasciotomy around the compromised nerve are alternative therapies if the injections are not effective [4].

** **Carnett's Sign:** increased local tenderness with muscle tensing. The supine patient is asked to raise his head or legs to increase tension in the rectus abdominis muscles while the examiner's finger presses on the site of pain; this increases pain due to an entrapped nerve while visceral pain is decreased.

The above tables are not complete but serve to point out the wide range of conditions that may cause abdominal pain while failing to affect our usual mix of diagnostic tests. Hospitalists should maintain a high index of suspicion for these problems and avoid rushing to the diagnosis of a psychiatric cause for the symptoms. At the same time, a careful history and physical examination may negate the need for expensive testing and ineffective therapies.

(References on the following page)

REFERENCES:

1. Fornaro, M et al., Prevalence and diagnostic distribution of medically unexplained painful somatic symptoms across 571 major depressed outpatients, Neuropsych Dis and Treat 2011; 7: 217-221
2. Jackson, JL and K Kroenke, Prevalence, Impact and Prognosis of Multisomatoform Disorder in Primary Care: a 5 year follow-up study, Psychosomatic Med 2008; 70: 430-434
3. Tuteja, AK et al., Opioid-induced bowel disorders and narcotic bowel syndrome in patients with chronic non-cancer pain, Neurogastroenterol Motil 2010; 22: 424-e96
4. Lindsetmo, R and J Stulber, Chronic abdominal wall pain—a diagnostic challenge for the surgeon, Am J Surg 2009; 198: 129-134
5. Srinivasan, R and DS Greenbaum, Chronic abdominal wall pain: a frequently overlooked problem, Am J Gastroenterol 2002; 97: 824-830

FROM THE JOURNALS

Deepika Jain MD

Tiotropium versus Salmeterol for the prevention of exacerbations of COPD

NEJM 2011; 364: 1093-1103

This study investigated the use of tiotropium vs salmeterol in reducing the exacerbations in patients with moderate to severe COPD. The current treatment guidelines recommend the use of long acting bronchodilators but do not specify beta agonist vs anticholinergic therapy. In this one year, randomized, double-blind study, a total of 7376 patients were randomly assigned to receive treatment with either salmeterol (50 mcg) or tiotropium (18 mcg). The authors concluded that tiotropium, as compared to salmeterol, significantly increased the time to the first moderate or severe exacerbation of COPD (187 days for tiotropium vs 145 days for salmeterol) and significantly decreased the annual rate of exacerbations among patients with moderate to severe COPD (reductions of 14% and 28% in moderate and severe COPD patients, respectively). This benefit was consistent and independent of the concomitant use of inhaled glucocorticoids.

Characteristics and short-term prognosis of perioperative myocardial infarction in patients undergoing non-cardiac surgery.

Devereaux, PJ et al., Annals Int Med 2011, April 19; 154(8): 523-528

The authors examined the characteristics and short-term outcome of perioperative MI; 8351 patients enrolled in the POISE trial were studied. The perioperative MI was defined as either autopsy findings of acute MI or an elevated biomarker of infarction and at least one of the following features: ischemic symptoms, new pathologic Q waves, ischemic changes on the EKG, coronary intervention or cardiac imaging evidence for an MI. The results showed that, within 30 days of random assignment, 5% of the patients had a perioperative MI, of which (cont)

(continued) 74.1% occurred within 48 hours of surgery; 65.3% of the patients did not experience ischemic symptoms. The 30 day mortality rate was 11.6% for those who had a perioperative MI vs 2.2% for those who did not have an MI; the mortality rate in the MI group was similar for those with and without ischemic symptoms. Since cardiac markers were not monitored beyond 3 days of the surgery, additional asymptomatic MIs may have been missed. The authors conclude that routine monitoring of cardiac markers in high-risk patients is warranted to detect perioperative MIs since the majority of these patients will not have ischemic symptoms.

Four Nephrology myths debunked

Rachoin, JS and EA Cerceo, J Hospital Med, 6: n/a. doi: 10.1002/jhm.703

This article reviews some important and often debated issues related to renal disease in hospitalized patients: 1. Hypothyroidism, unlike myxedema, is not a cause of hyponatremia; the simultaneous presence of both disorders is often noted since both are widely prevalent but additional investigations are needed to determine if there is a true correlation. 2. Sodium bicarbonate is effective for the treatment of hyperkalemia primarily by enhancing renal potassium elimination rather than by translocating potassium into cells. 3. Acetaminophen can be a cause of metabolic acidosis by causing 5-oxoprolinuria. 4. Furosemide (and sulfa containing diuretics) can be safely used in patients with an allergy to sulfa-containing antibiotics.

ID CORNER

WILLIAM SALZER MD

PREVENTION OF IV CATHETER INFECTIONS

The IDSA has just released its updated, evidence-based practice guidelines for the prevention of IV catheter infections. This will be useful for your infection control program.

O'Grady, NP et al., Guidelines for the prevention of intravascular catheter-related infections

Clin Infect Dis 2011; 52: e1-e32

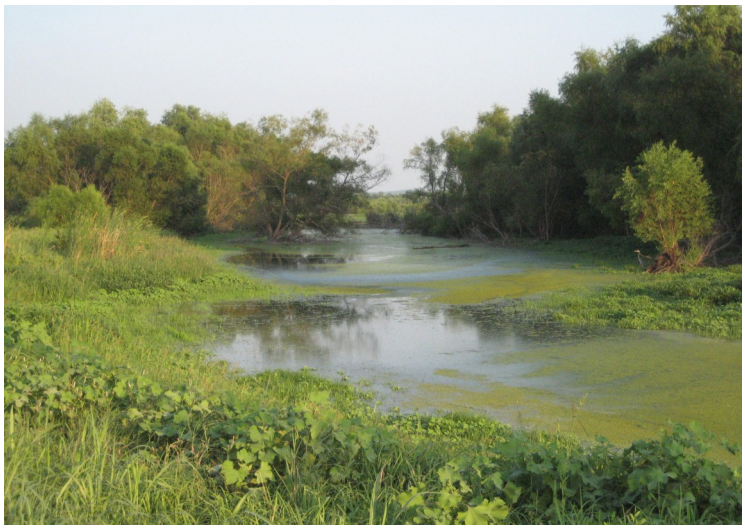
http://cid.oxfordjournals.org/content/early/2011/04/01/cid.cir257.full.pdf+html

MISSOURI HOSPITALIST SOCIETY

University of Missouri
Division of General Internal Medicine DC043
1 Hospital Drive
Columbia, MO 65212

folzenlogenr@health.missouri.edu

MISSOURI HOSPITALIST CALENDAR



Difficult Airway Management, American College of Chest Physicians, July 22-24, Northbrook, IL; register via www.chest.org

21st Annual Caring for the Frail Elderly, Missouri Association of Long-Term Care Physicians, August 19-20, Holiday Inn Select, Columbia, MO; register online at: http://medicine.missouri.edu/cme **LOCAL**

12th Annual St. Louis Critical Care Update, Drury Plaza, Chesterfield, September 10; information & registration via http://cme.wustl.edu **LOCAL**

2011 Missouri Chapter Scientific Meeting, American College of Physicians, September 15-18, Tan Tar A Resort, Osage Beach; **Hospitalist Conference Lunch** at 12:15 on Saturday, September 17; contact Patrick Mills 573-636-5151 or via email: pmills@msma.org; details to follow **LOCAL**

7th Annual Health Ethics Conference: The Ethics of Effective Communication in Health Care, Center for Health Ethics, University of Missouri School of Medicine, Columbia, MO, October 6-8, 2001; information & registration call 573-882-4105 or visit the website: http://medicine.missouri.edu/cme **LOCAL**

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

Robert Folzenlogen MD, folzenlogenr@health.missouri.edu

Please forward this newsletter to Hospitalists that you might know!