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Hospitalist Update

Curbside Consults: a tradition we ought to abandon

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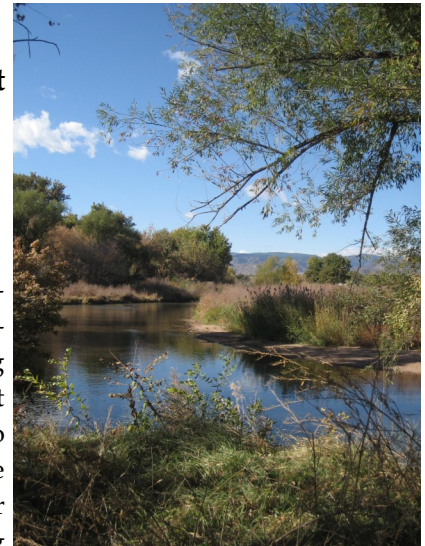
Physicians, having shared and survived the gauntlets of medical school and residency, tend to be supportive of one another. At the same time, having first hand knowledge of the time commitment that goes with our profession, we are often hesitant to ask for one another's assistance. These factors have been instrumental in the evolution of the curbside or sidewalk consult, a practice that is ingrained during the hectic years of residency.

Though widely utilized and well intentioned, the reliance on curbside consults has several limitations and pitfalls which, I suggest, make a case for abandoning this practice. Among these points are the following:

Limited Data leads to Bad Advice. As any consultant knows, the initial information provided by the consulting physician is rarely complete and not always accurate. Clinical assumptions based on a cursory review of the case may lead to off-the-cuff recommendations that are not in the best interest of the patient. A formal consult, on the other hand, invites the consultant to thoroughly review the chart, perform their own history and physical and produce a well-informed set of recommendations.

Curbside Consults are Legal Hearsay. It is one thing to leave a note in the chart that the patient's case was "discussed with Cardiology" and it is quite another to convince a court of law that such advice was obtained. Formal consults produce clear documentation of the consultant's assessment and recommendations and, assuming that such advice was followed, physicians can face the occasional bad outcome with the knowledge that appropriate consultations were obtained and documented.

Free Advice versus Time Saved. One of the primary reasons that physicians resort to curbside consults is to not burden the consultant with the need to see the patient and produce a formal note; this is, indeed, the driving force behind sidewalk consults between residents and fellows. Of course, this also deprives the consultant of the ability



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(continued) to bill for his/her services. Whether in an academic center or a private practice, having the opportunity to appropriately bill for one's expertise is essential to maintaining financial viability.

Patient Trust & Satisfaction. While patients in academic centers are often overwhelmed by the number of faculty, residents and students that attend to them, they always want to know who is in charge of their care. Few patients would be satisfied to hear that their physician has "run their case past the surgeon." Rather, despite their occasional proclamations to the contrary, patients are reassured by the personal attention that they receive from a consultant's visit; this, of course, does not occur when one resorts to curbside consults.

Reverse Curbside Consults. It is not uncommon that frenzied consultants might offer sidewalk advice (usually over the phone) even though a formal consult was requested. Though, once again, this practice is most common at the training level, those of us familiar with the private sector know that such bail-out attempts occur within all medical communities. For the reasons discussed above (billing considerations aside), it is advisable to insist on a formal consultation, including chart documentation of his/her recommendations. Should such a request meet with resistance, it is best to change consultants.

Young physicians, faced with the numerous demands and time constraints of their career, may dismiss these points as the trivial ramblings of an aging practitioner. While I plead guilty to the aging part, experience teaches all of us that shortcuts in the care of our patients often lead to unnecessary complications. The curbside consult is one of these shortcuts and, in my opinion, ought to be abandoned.

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CASE OF THE MONTH

Bishnu Devkota MD & Fereidoon Shafiei MD

A 40 year old African American male, with a history of diabetes, presented to the emergency department with persistent diarrhea over the past few months. Soon after arrival, the patient developed respiratory failure, requiring mechanical ventilation. Labs at the time of presentation revealed severe hypokalemia (K 1.0), metabolic acidosis (HCO₃ 10), prerenal azotemia (BUN 150, Cr 11) and marked hypoalbuminemia (Alb 1.0). IV hydration and electrolyte supplementation were initiated, he was soon extubated and he had a relatively uneventful hospital course. A non-contrast CT of the abdomen was normal and a colonic biopsy showed only nonspecific, mild inflammation.

Two weeks after discharge, the patient presented with worsening diarrhea (up to 40 times per day) and severe weakness; he denied hematochezia or melena and had no abdominal pain. His past medical history was remarkable for diabetes mellitus, hypertension, a seizure disorder (thought to be related to alcohol use) and a long history of alcohol and tobacco abuse. Surgical history was limited to amputation of a toe due to gangrene. He admitted to noncompliance regarding his medications. Family history was also positive for hypertension and diabetes.

His admission exam revealed that he was alert and oriented to time, person and place. He was pale and his mucous membranes were dry. Orthostatic vitals revealed a BP of 132/97 and P 81 when supine, BP 87/45 and P 115 when standing. Chest was clear to auscultation. Abdomen was soft, non-tender and non-distended; bowel sounds were diffusely hyperactive. Skin turgor was decreased, generalized muscular weakness was noted but no neurologic deficits were found.

Admission labs revealed WBC 13,300 (with 63.5%N, 29.6%L, 1%Eos, 5%M), Hgb 7.8, Hct 22, and platelet count of 213,000. Chemistries returned Na 136, K 1.7, Cl 115, HCO₃ 14, BUN 12, Cr 2.4, Gluc 153, Ca 6.1, Alb 2.6, AST 65, ALT 34 and AlkPhos 168. His serum amylase and lipase were normal. EKG demonstrated T wave inversion and U waves, reflecting his severe hypokalemia. A CXR was normal and plain abdominal films were unremarkable.

IV hydration and electrolyte repletion were initiated. Based on the conviction that this patient may have chronic pancreatic insufficiency, he was started on pancreatic enzyme supplementation. His electrolyte abnormalities and diarrhea soon resolved and he was doing well at the time of followup, 1 month later; he did report that his diarrhea would redevelop if he stopped taking the enzyme supplements for a few days. At a followup visit 2 years later, the patient has remained compliant with his medications and has had no further diarrhea.

Discussion: Pancreatic insufficiency usually causes severe protein and fat malabsorption; patients generally become symptomatic once 90% of the pancreatic exocrine function has been lost. One of the most common causes of pancreatic insufficiency is chronic pancreatitis, usually secondary to a long history of alcohol abuse.

Chronic pancreatitis is a syndrome that involves progressive inflammatory change in the pancreas, eventually resulting in permanent structural damage; this leads to impairment of endocrine and exocrine function. Patients with chronic pancreatitis often complain of epigastric pain (which may radiate to the back), weight loss and diarrhea; nausea and vomiting may also occur. Our patient, though plagued by severe diarrhea, did not experience abdominal pain; in one study, 20% of patients with chronic pancreatitis presented with endocrine or exocrine dysfunction in the absence of abdominal pain. Forty five percent of asymptomatic alcoholics have been found to have evidence of chronic pancreatitis on postmortem examination [1].

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The true prevalence of chronic pancreatitis is not known, although estimates range from .04 to 5 percent [2]. A long history of heavy alcohol consumption (at least 6-12 years) accounts for 60-70% of cases in developed countries [3]. Obstruction of the pancreatic duct can also lead to chronic pancreatitis; this is characterized by dilation of the pancreatic duct and exocrine insufficiency. Tropical pancreatitis, most often seen in Africa and Asia, appears in young persons and is characterized by pancreatic insufficiency, diabetes mellitus and recurrent abdominal pain; it is uncertain whether this condition is caused by protein malnutrition or by ingestion of toxic substances such as the cyanogens in cassava root [4]. Mutations in serine protease inhibitor SPINK1 have been identified as a cause in some patients [5-6]. Chronic pancreatitis can also occur in association with cystic fibrosis or hyperparathyroidism [3].

Autoimmune pancreatitis is most often seen in the Far East and is associated with other autoimmune disorders such as Sjogren's syndrome and SLE; these patients have autoantibodies such as ANA and increased gamma globulin in their blood and often have no or minimal symptoms [7]. Approximately 30-40% of patients with chronic pancreatitis have no apparent underlying cause and are thus classified as idiopathic chronic pancreatitis. Regardless of the cause, the pathogenesis of chronic pancreatitis, though poorly understood, has been attributed to an increased secretion of ductal protein without a compensatory increase in ductal bicarbonate; histologic examination may reveal patchy inflammatory changes within the exocrine pancreas.

Diagnostic Considerations. An accurate history and diagnostic evaluation is required to confirm the presence of chronic pancreatitis. Steatorrhea may be assessed qualitatively by looking for fat globules or by staining stool samples with Sudan red; quantitative results are obtained by fecal fat excretion (>20 grams per 24 hours). Dynamic testing, such as the secretin stimulation test and the bentiromide (chymex) test, and quantitative tests such as stool chymotrypsin are now infrequently used due to a significant improvement in imaging technology [8]. Diffuse, intraductal calcium deposition, pathognomonic of chronic pancreatitis, will be seen on plain abdominal radiographs in up to 30% of cases [9]. The diagnostic sensitivity and specificity of ultrasound is 60-70% and 80-90% respectively [10]. CT scanning has a higher sensitivity and is the imaging modality of choice to look for complications of chronic pancreatitis [11]. Endoscopic ultrasonography is becoming a popular tool for the diagnosis and ERCP remains the gold standard for confirming chronic pancreatitis secondary to ductal abnormalities [12].

When the diagnosis remains uncertain, other causes of chronic abdominal pain, such as peptic ulcer disease, gallstones, irritable bowel syndrome and endometriosis, should be considered. The possibility of pancreatic carcinoma must be kept in mind since it can mimic chronic pancreatitis and may develop against the background of pancreatitis, often delaying the diagnosis until late in the disease process [8].

Treatment Considerations. The treatment of chronic pancreatitis involves the elimination of its cause, symptomatic therapy and pancreatic enzyme supplementation. Cessation of alcohol is imperative; while this may or may not relieve pain, it does reduce mortality [2]. Dietary changes, especially the consumption of small meals with low fat content, often improves symptoms; pancreatic enzyme supplementation may also reduce pain in some patients [13]. Oral trypsin deactivates intrinsic cholecystokinin and thus decreases pancreatic stimulation of digestive peptides; this appears to be the mechanism by which they help to relieve pain [14]; however, this therapy remains controversial, has not been consistently supported in studies and is most likely to benefit women and those with mild disease. Steroid therapy has been shown to dramatically improve symptoms in patients with autoimmune pancreatitis [15].

Many anecdotal reports, including our own, have documented significant improvement in clinical symptoms with the use of pancreatic enzyme supplementation and either H2 blockers or proton pump inhibitors; pancreatic enzyme supplementation is devoid of major side effects although, very rarely, allergic reactions have been reported. Colonic stricture is a rare complication of pancreatic enzyme therapy in patients with cystic fibrosis [16].

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According to clinical trials, pancreatic enzyme supplements are most likely to relieve pain in patients with small duct disease [17]. Treatment with octreotide, a potent pancreatic enzyme secretion inhibitor, is not widely recommended. The use of antioxidants has shown some benefit but further studies are warranted. CCK receptor antagonists (MK329) and the treatment of pain with tricyclic antidepressants and gabapentin are other promising options [17]. Endoscopic ductal dilatation, in patients with mild pancreatitis, and endoscopic ultrasound or CT guided celiac plexus blockade are other modalities being used for the relief of chronic pain. Finally, surgery is reserved for the management of intractable pain.

Conclusion: The accurate diagnosis of chronic pancreatitis relies on a constellation of imaging and laboratory studies. When such studies are unavailable or contraindicated (e.g. the presence of acute renal failure), a therapeutic and diagnostic trial of pancreatic enzyme supplementation may be warranted; indeed, in our patient, this therapy completely controlled his symptoms and protected him from the potential complications of dehydration and electrolyte imbalance. When the clinical suspicion of chronic pancreatitis is high and the resources are limited, this intervention may prove to be an excellent strategy.

References:

1. Layer, P et al., *The different courses of early and late onset idiopathic and alcoholic chronic pancreatitis*, *Gastroenterology*, Nov 1994; 107(5):1481-1487
2. Steer, ML et al., *Chronic Pancreatitis*, *NEJM*, June 1, 1995; 332(22):1482-1490
3. Owyand, C et al, Ed, *Chronic Pancreatitis*, *Textbook of Gastroenterology*; No.2, JB Lippincott, NY 1991
4. Sarles, H et al., *Pancreatic lesions and modifications of pancreatic juice in tropical chronic pancreatitis (tropical calcific diabetes)*, *Dig Dis Sci.*, June 1994; 39(6):1337-1344
5. Bhatia, E et al., *Tropical Calcific Pancreatitis: strong association with SPINK1 trypsin inhibitor mutations*, *Gastroenterology*, Oct 2002; 123(4):1020-1025
6. Schneider, A et al., *SPINK1/PSTI mutations are associated with tropical pancreatitis and type II diabetes mellitus in Bangladesh*, *Gastroenterology*, Oct 2002; 123(4):1026-1030
7. Ozaki, K and T Chiba, *Autoimmune related pancreatitis*, *Gut*, Jul 2002; 51(1): 1-4
8. Mergener, K and J Baillie, *Chronic pancreatitis*, *Lancet*, Nov 8 1997; 350(9088):1379-1385
9. Grendell, JH et al., ed, *Chronic Pancreatitis*, 5th Edition, *Gastrointestinal Disease*, WB Saunders Co, Phil. PA
10. Bolondi, L et al., *Sonography of Chronic Pancreatitis*, *Radiol Clin NA*, Jul 1989; 27(4):815-833
11. Maynor CH NR, ed *Imaging of the Pancreas*, Malden:Blackwell; 1997; Dimarino, AJ, BS eds, *Gastrointestinal Disease: an endoscopic approach*
12. Malfertheiner, P and M Buchler, *Correlation of imaging and function in chronic pancreatitis*, *Radiol Clin NA*, Jan 1989; 27(1):51-64
13. Whitcomb, DC et al, *Pancrealipase delayed-release capsules (CREON) for exocrine pancreatic insufficiency due to Chronic Pancreatitis or Pancreatic Surgery: a Double-blinded Randomized Trial*, *Am J Gastroenterol* 2010
14. Owyand, C., *Negative feedback control of exocrine pancreatic secretion: role of cholecystokinin and cholinergic pathway*, *J. Nutrition*, Aug 1994; 124(8 Suppl):1321S-1326S
15. Noor, MT et al., *Autoimmune pancreatitis: a report from India.*, *JOP* 2010; 11(3):213-219
16. Smyth RL et al., *Strictures of ascending colon in cystic fibrosis and high-strength pancreatic enzymes*, *Lancet* Jan 8, 1994; 343(8889):856-89
17. Singh VV and PP Toskes, *Medical therapy for chronic pancreatitis pain*, *Curr Gastroenterol Rep*, Apr 2003; 5(2):110-116

FROM THE JOURNALS

The following journal articles should be of interest to hospitalists:

Diabetic Gastroparesis

Camilleri, M., *New England J Med* 2007; 356:820-829

Cytomegalovirus reactivation in critically ill immunocompetent patients

Limaye, A. et al, *JAMA* 2008, Vol 300, No 4, pages 413-422

Pulmonary Hypertension in Hemodialysis Patients: an unrecognized threat

Yigla, M et al., *Seminars in Dialysis*, 2006, Vol 19, No 5, pages 353-357

Thrombotic Thrombocytopenic Purpura

George, JN, *New England J Med* 2006; 354:1927-1935

Sepsis & Septic Shock in Pregnancy

Sheffield, JS, *Critical Care Clinics* 20, 2004, pages 651-660

ID CORNER

William Salzer MD

PREVENTION & TREATMENT OF NOSOCOMIAL INFECTIONS

Here is a recent article from the NEJM that nicely and concisely reviews nosocomial infections, their prevention and their treatment:

Peleg, AY and DC Hooper, Hospital acquired infections due to gram-negative bacteria
New England J Med 2010; 362:1804-1813

<http://content.nejm.org/cgi/reprint/362/19/1804.pdf>

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MISSOURI HOSPITALIST CALENDAR

20th Annual Conference: Caring for the Frail Elderly, August 20-21, Holiday Inn Select Executive Center, Columbia, MO: call 573-882-5661 or register at som.missouri.edu/CME **LOCAL**

Brain Attack! 2010, Comprehensive Stroke Care Door-to-Door, Saturday, October 9, Eric P. Newman Education Center, Washington University Medical Center, St. Louis, register at <http://cme-online.wustl.edu> **LOCAL**

Chest 2010, October 30-November 4, Vancouver, BC, register online via: www.chestnet.org/accp/chest/chest-annual-meeting

Hospitalist Lunch Conference, Missouri ACP Meeting, September, 2010; presentations from MU, UMKC, Washington University; the luncheon will be on Saturday, September 25. **LOCAL**

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!