CLINICAL MANAGEMENT

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Alcoholic Patient With Constant Abdominal Pain and Normal Amylase

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Clinical Case

A 32-year-old man is admitted to the hospital with 2 days of constant, moderately severe epigastric pain, radiating to the back and associated with nausea and vomiting. He denies fevers, chills, sweats, change in weight, hematemesis, melena, hematochezia, or jaundice. He has consumed 12–18 beers per day for the past 10 years, but his past medical history is otherwise unremarkable. He takes no medications and denies recent trauma. On physical examination he is afebrile and diaphoretic. His heart rate is 125 per minute with a blood pressure of 145/90. He is anicteric with no stigmata of chronic liver disease. His abdomen is flat with moderate tenderness in the mid-epigastrium but without peritoneal findings. Hemoglobin, white blood cell count, electrolytes, liver tests, and serum amylase are normal.

Background

Multiple diagnoses can be entertained in this patient presenting with moderately severe abdominal pain, nausea, and vomiting but with normal laboratory tests (Table 1). The principal differential diagnoses should always include those with potentially serious morbidity, such as perforated ulcer. While most young patients with gut perforations do present with severe abdominal pain and classic "peritoneal signs," contained or chronic perforations in patients with altered mental status and/or alcohol or drug usage not infrequently present with more subtle symptoms and physical signs. Acute alcoholic "gastritis" is a diagnosis conveniently offered in many of these patients on the basis of clinical presentation and relatively nonspecific endoscopic features of erythematous mucosa. While alcohol does alter gastric mucosal permeability, the majority of the endoscopic features are either nonsignificant or suggestive of portal hypertensive gastropathy, a condition not associated with abdominal pain.

Given the patient's lifestyle, even despite the absence of trauma, a missed splenic or hepatic laceration, hematoma, or infarction should be considered. The epigastric location of his abdominal pain and the limited physical findings in this area would also mandate that acute cholecystitis and spontaneous bacterial peritonitis be considered. Despite the epigastric location of the pain and absence of impressive physical findings in the right lower quadrant, any patient presenting with abdominal pain who has not had a prior appendectomy needs to be evaluated for possible acute appendicitis. On occasion, patients with lower lobar pneumonia, particularly pneumococcal pneumonia, will present with peritoneal findings rather than more typical pulmonary manifestations of pneumonia. Other less likely diagnoses that need to be considered would include retroperitoneal or mesenteric hemorrhage, renal, or adrenal hemorrhage or infarction, renal calculus, or even an internal hernia, volvulus, or viscus infarct.

Recognizing the reported subjective location of the pain, the tenderness in the epigastrium, and the heavy extensive long-term alcohol abuse, pancreatitis certainly should be considered in the differential diagnosis despite the normal serum amylase level. Could this be, for example, the first bout of acute pancreatitis presenting belatedly in this 32-year-old alcoholic with a 10-year history of heavy alcohol consumption? Might we be dealing with established chronic alcoholic pancreatitis now presenting with its first episode of abdominal pain—perhaps related to a complication such as the develop-

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Table 1. Multiple Diagnoses for Alcoholic Patients with Abdominal Pain

Diagnosis	Pro	Con	Additional testing needed
Peptic ulcer disease	Young age/alcoholic/sudden onset	No past history/no response to antacids/no overt bleeding	CT scan/abdominal series/endoscopy
Alcoholic "gastritis"	Alcohol usage/sudden onset	Absent melena/hematemesis	Endoscopy after negative CT to rule out other diagnoses
Acute pancreatitis	Heavy alcohol usage/pain pattern	Normal amylase/no prior history	Abdominal CT (oral and IV contrast) & serum lipase level
Chronic pancreatitis	Chronic alcohol usage/pain pattern	No prior pancreatitis/normal amylase	Abdominal CT/serum lipase level
Acute appendicitis	No prior appendectomy/young age/ sudden onset	Normal WBC/epigastric pain pattern	Abdominal CT/repeat physical assessments and WBC
Splenic/hepatic laceration or hematoma	Alcoholic/sudden onset/upper abdominal pain	Normal HCT and WBC/absent peritoneal signs	Abdominal CT/repeat physical assessments and CBC
Splenic/hepatic infarct	Young age/severe upper abdominal Pain	Absent IV drug usage	Abdominal CT
Spontaneous bacterial peritonitis	Chronic alcoholic/abrupt onset of pain	No gross ascites/normal albumin/normal WBC	Paracentesis if ascites on CT
Acute cholecystitis	Epigastric pain	Normal liver tests/epigastric, not right upper quadrant pain/male sex	Abdominal ultrasound
Internal hernia/volvulus	Abrupt onset of emesis	No prior surgery/epigastric pain	Abdominal CT
Retroperitoneal/mesenteric hemorrhage	Sudden onset/pain out of proportion to physical findings	Bland physical exam/normal HCT	Abdominal CT
Renal/adrenal infarct	Sudden onset/pain disproportion to exam	Normal WBC/unimpressive physical findings	Abdominal CT
Lower lobar pneumonia	Alcoholic/sudden onset of upper abdominal pain	Absent cough or sputum/normal temperature and WBC	Chest PA & lateral radiographs
Obstructive uropathy	Young male/sudden onset of pain	Epigastric pain/absent hematuria/normal/WBC	Abdominal/pelvic CT

ment of a pseudocyst? While both lipase and amylase are sensitive and specific in the majority of patients with pancreatitis, a substantial number of patients will present with pancreatitis or with one of many its complications with normal pancreatic enzyme levels. This is particularly more common in patients who have presented late in the course of their illness and/or have long-standing chronic pancreatitis and are presenting with one of multiple flares of the disease.

Potential Management Strategies

One can approach this patient using several different strategies. A "minimalist" approach might consist of few additional diagnostic tests and expedited hospital discharge. Recognizing the short-term duration of illness in a previously healthy individual with epigastric tenderness but normal initial laboratory tests, a minimalist imaging approach would also likely consist of a plain abdominal series (upright radiograph of chest and abdomen plus a supine abdominal film). The abdominal plain radiographs would be helpful in excluding gross abdominal findings such as sub-diaphragmatic air; gallbladder, pancreatic or renal calcifications; and intramural air or intramural edema of the bowel.

It would also be reasonable to request a "routine" posterior-anterior (PA) and lateral chest x-ray to exclude lobar pneumonia–particularly a lower lobe pneumonia presenting with abdominal pain.

Additional laboratory work-up taking a "minimalist" approach could be justified in this patient including urinalysis looking specifically for the presence of blood or white cells and a serum lipase determination. In addition, to the laboratory and radiographic testing outlined previously, several additional physical diagnostic tests might be appropriate at a minimum level for this patient including a rectal examination with a stool sample for occult blood and, if positive, a nasogastric aspirate for fresh blood or melenic material.

A "maximal" diagnostic strategy in this patient would be to draw additional blood samples for glucose, calcium, phosphate, and magnesium, and also arrange for an abdominal and pelvic CT scan with oral, rectal, and intravenous contrast. In addition to the CT scan, given the complaint of epigastric pain associated with nausea and vomiting, one could consider a right upper quadrant ultrasound to look at the gallbladder to exclude cholesterol stones, which well might be missed on a CT scan of the abdomen.

A "middle ground" approach on this patient would be to add to the minimal evaluation of the patient an abdominal CT scan with oral and intravenous contrast. Should the abdominal CT scan prove negative and the

additional laboratory tests are normal or nonspecific, the patient would be a reasonable candidate for outpatient follow-up and ongoing evaluation. The major problem with the "minimalist" or "middle-ground" approaches would be patient follow-up, since in many instances, the alcoholic patient is unreliable in this respect.

Questions do arise as to how one should approach such a patient should the CT scan prove negative and the additional laboratory testing is normal or nonspecific. Since clinicians had already decided that the epigastric discomfort and tenderness was impressive enough to merit hospitalization, the prudent course is always to observe the patient for an additional limited period of time, repeating laboratory testing and seeking a surgical consultation. It would also be appropriate to advance his diet and be certain he can tolerate a regular diet before discharge. It is likewise important to take advantage of any hospitalization of a chronic alcoholic patient to arrange for evaluation by alcohol treatment programs. In many instances, detoxification programs will take patients once medically cleared, directly from an inpatient status. Furthermore, the additional time in hospitalization while observing the patient may be helpful in arranging for short-term housing and/or support services for these medically and socially disenfranchised patients.

Recommended Management Strategy

Given the young age of the patient, the relatively short duration of mid-epigastric pain, associated nausea and vomiting and the generally benign physical examination and laboratory values, I would recommend a serum lipase determination and a CT scan of the abdomen and pelvis with oral and intravenous contrast. An initial consultation with a surgeon would also be helpful and can provide important insights early during the course of evaluation and treatment. Why the lipase determination in the face of an initial normal serum amylase? Why the CT scan at such a seemingly low threshold rather than a series of plain abdominal plain radiographs? We will examine these issues in turn.

Biochemical Tests in Patients With Suspected Pancreatitis

No biochemical test has been established as the true "gold standard" for making or excluding the diagnosis of acute pancreatitis, determining the etiology of acute pancreatitis, or predicting the severity of the illness.[1–8] Serum amylase determination was first recommended by Elman et al. in 1929 and for 75 years was considered the universal laboratory test to establish the diagnosis of acute pancreatitis. Amylase levels usually rise within 2 to 12 hours after the onset of symptoms, peak within the first 48 hours of the disease and return to normal within 5 days.[5] When abdominal CT scans are used to ascertain clinical diagnosis, serum amylase has a sensitivity of approximately 85% to 95%.[3] The major advantages of serum amylase determination over other biochemical tests in patients with suspected pancreatitis are its technical simplicity, its low costs, its ready availability, and its relatively high sensitivity. The main disadvantage of serum amylase determination in making the diagnosis of acute pancreatitis is a relatively low specificity, particularly in patients who have hyperlipidemia or acute alcoholism and in patients presenting late in the course of pancreatitis.[3]

Approximately 20% to 30% of patients with CT-diagnosed pancreatitis will have a normal serum amylase.[3] Moreover, many conditions other than pancreatitis are associated with elevated serum amylase levels, including alcoholism without pancreatitis, intra-abdominal inflammatory processes, parotiditis, macroamylasemia, and renal insufficiency. Acute alcoholics without pancreatitis have an elevated serum amylase level in up to 30% of instances. In patients with pancreatitis due to hypertriglyceridemia, serum amylase levels may be normal in 50%. Some circulatory inhibitor of amylase, not triglyceride itself, appears to interfere with certain amylase determinations. Serial dilutions of the serum and recalculation of the enzyme level is needed in patients with lactescent serum. As a general clinical recommendation, a normal serum amylase level in a patient with abdominal pain likely excludes the diagnosis of acute pancreatitis with the exception of possible hyperlipemic pancreatitis, acute exacerbations of chronic pancreatitis, and assessment late in the course of the disease.[3,5]

Serum lipase levels rise within 4 to 8 hours after the onset of symptoms, peak after 24 hours, and normalize within 8 to 14 days from the onset of acute symptoms.[5] Lipase determination is more complicated than serum amylase and is more costly to perform. Most patients with acute pancreatitis have an elevated serum amylase level and the benefits of substituting or adding a serum lipase is not clear. The sensitivity of serum lipase determination in patients with acute pancreatitis diagnosed by CT scans is about the same as for amylase, between 85% and 95%. Serum lipase levels do have increased sensitivity when compared with amylase, particularly in patients with alcoholic pancreatitis. Serum lipase is also a helpful diagnostic test in patients who present many days into the course of the illness. Serum lipase levels remain elevated longer than do amylase levels. In a study

Table 2. Pancreatic Enzyme Levels in Pancreatitis

Enzyme determination	Pancreatitis patients (U/L) 51 patients	Control patients (U/L) 35 patients	Diagnostic threshold (U/L) for pancreatitis	Sensitivity (at diagnostic threshold)	Specificity (at diagnostic threshold)
Day 1 serum lipase	677 (139–3592)[a]	71 (26–129)	208	67[b]	97
Day 1 serum total amylase	118 (64–699)[a]	63 (33–101)	176	45[b]	97
Day 1 serum pancreatic isoamylase	100 (34–703)[a]	34 (16–43)	94	51	94

NOTE. Values for pancreatitis and control patients expressed as medians and interquartile ranges.
[a]$P < 0.001$ vs. disease controls.
[b]$P < 0.05$ vs. serum lipase.
Adapted from Tracy J, Williams A, Bais R, Willson K, et al. Evaluation of amylase and lipase in the diagnosis of acute ancreatitis. ANZ J Surg 2001;71:577–582.

by Treacy et al., enzyme levels in patients with acute pancreatitis were analyzed (Table 2).[1] On the day of admission, serum lipase had the highest diagnostic accuracy ($P = 0.041$ vs. serum amylase). Using a threshold level of 208 IU/L, lipase gave a sensitivity of 67% and a specificity of 97%. Other diagnostic thresholds at admission were as follows: serum amylase, 176 IU/L (sensitivity 45%, specificity 97%) and serum pancreatic isoamylase, 41 IU/L (sensitivity 63% specificity 85%).[1] At 3 days into the admission, no enzyme determination was superior to lipase. The authors also reported that the sensitivity of urine amylase determinations and urine amylase/creatinine ratios were similar to those of the serum amylase thus supporting the contention that urinary amylase measurements are not particularly helpful in this clinical setting.[1] As with serum amylase determinations, the serum lipase has virtually no value in assessing the severity or the likely etiology of acute pancreatitis.

A variety of other tests have been recommended to enhance the clinical accuracy in making the diagnosis of acute pancreatitis.[1–8] The determination of the pancreatic isoamylase by electrophoresis is certainly more sensitive than total serum amylase but is generally not available. For that matter, the determination of immunoreactive trypsin and elastase have likewise been suggested as being more sensitive and specific in making the diagnosis of acute pancreatitis. These tests are also not generally available in most clinical laboratories.

More recently, a urine trypsinogen-2 test strip (Actim Pancreatitis Test Strip, Medix Biochemica, Helsinki, Finland) has been suggested to have a sensitivity of 93% and a specificity of 92% compared to a serum lipase sensitivity of only 79% and specificity of 88%.[5–7] While these additional laboratory tests have been proffered to enhance the accuracy of diagnosing acute pancreatitis they are not generally available and have not gained widespread acceptance, particularly in the United States, when compared with serum amylase and lipase. This is particularly relevant when we consider that for the vast majority of patients with new onset of abdominal pain, some form of abdominal imaging, principally a computed tomography scan of the abdomen is usually used.

Imaging Studies in Patients With Abdominal Pain

The evaluation of patients with abdominal pain has benefited greatly by developments in cross-sectional imaging, principally multidetector row helical computed tomography (CT) scans of the abdomen. These advances have engendered controversy as well as enthusiasm. One initial question to be answered is what, if any, is the place of plain radiographs of the abdomen in the face of the near universal availability and accessibility of CT scans?

It is painfully obvious that plain films of the abdomen are rarely diagnostic when patients present with acute abdominal pain.[9–14] Possible significant positive findings on plain radiographs in patients with acute abdominal pain include pneumoperitoneum, pneumobilia, hepatic portovenous gas, small bowel obstruction, large bowel obstruction, toxic megacolon, volvulus, intramural gas, and calcifications in kidneys, ureters, appendix, liver, gallbladder, or pancreas.[11]

Plain radiographs of the abdomen, also termed incorrectly "KUB" or "flat plate of the abdomen," usually consist of a series of 3 images including an erect abdominal film, a supine abdominal film, and an erect chest x-ray centered on the diaphragm to look for the presence of subdiaphragmatic air. In a study by Anyamwu et al. of 224 patients referred to an emergency room for acute nontraumatic abdominal pain, only 13 of 125 patients (10.4%) had "diagnostic" findings on the plain abdominal radiographs.[10]

Certainly in patients with more impressive clinical findings, abdominal computed tomography scans should and have all but replaced plain abdominal radio-

graphs.[16–22] Nagurney et al. retrospectively reviewed 177 patients with nontraumatic abdominal pain who underwent abdominal CT scans.[16] Ninety-seven patients (55%) also received plain radiographs of the abdomen. Seventy-four patients were admitted to the hospital and their clinical course and/or invasive procedural findings were used as the gold standard for the diagnosis. The overall sensitivity of plain radiographs of the abdomen in this clinical setting was 43% while the overall specificity was 75%. The corresponding sensitivity and specificity of abdominal CT scans was significantly better at 91% and 94%. In patients in whom a CT scan is likely to be ordered, the plain radiographs of the abdomen provided no information over and above that provided by the CT scans.[16]

Indeed, plain radiography of the abdomen is more commonly used in the evaluation of patients who have low intensity nonspecific abdominal pain. Because of their relatively low cost and ready availability, plain abdominal radiographs are currently being used disproportionately as a "routine screening" test to exclude major significant findings, which in fact are hardly ever present in these patients (Table 3). Aside from the detection of gross free peritoneal air, foreign bodies, and marked gut distention, the "KUB" is virtually useless and should be abandoned in almost all instances in favor of the CT scan.

Roth et al. studied 164 sequential patients with nonspecific, nontraumatic abdominal pain who were evaluated in the emergency room by abdominal CT scans of the abdomen and pelvis.[17] One hundred patients (60.9%) had abnormal scans with the most common findings consisting of appendicitis (17 patients), diverticulitis (9 patients), abdominal fluid collections (5 patients), neoplasms and abdominal abscesses (3 patients each), and pancreatitis (2 patients). An additional 57 of the 164 patients who underwent CT scans of the abdomen for nonspecific abdominal pain had unrelated findings on CT. Positive findings for appendicitis in their study correlated best with a white blood cell count greater than 11,500, male sex, age younger than 25 years (all $P < 0.05$). A positive finding on abdominal CT correlated best with a white blood cell count greater than 11,500. (OR 7.7, 95% CI, 3.3–18).[17]

Table 3. Diagnostic Sensitivity in Patients With Abdominal Pain: Plain Films vs. CT

Clinical diagnosis	Abdominal plain films	CT scans
Foreign body	90%	–
Bowel obstruction	33%	75%
Appendicitis	0%	50%
Pyelonephritis	0%	40%
Pancreatitis	0%	60%
Diverticulitis	0%	25%
Urolithiasis	0%	68%

Adapted from Ahn SH, et al. Acute nontraumatic abdominal pain in adult patients: abdominal radiography compared with CT evaluation. Radiology 2002;225:159–164.

Clearly CT now plays the major imagining role in the evaluation of patients with suspected acute appendicitis, a diagnosis that must be seriously considered in all patients evaluated for abdominal pain except those who have already undergone an appendectomy. Hershko reviewed a cohort of 308 patients with suspected acute appendicitis of whom 198 were evaluated by CT scans.[18] CT scans had a sensitivity, specificity, and overall accuracy for the diagnosis of acute appendicitis of 91%, 92%, and 91% respectively. Similarly, in a study by Maluccio, CT scans were evaluated in 104 patients with suspected appendicitis with a reported sensitivity of 80%, and specificity and positive predictive value of 91% and 82% respectively.

It does appear, therefore, that in the vast majority of patients presenting to the emergency room with nonspecific nontraumatic acute abdominal pain, an oral and intravenous contrast enhanced CT scan of the abdomen is distinctly helpful and should be ordered.

Evolution of the Case

Over the first 24 hours of admission, the symptoms and physical findings were unchanged. Urinalysis, serum lipase, and repeat blood count and liver biochemical tests were negative or normal, and a chest radiograph was normal. Computed tomography examination of the abdomen and pelvis was performed with oral and intravenous contrast administration (Figure 1). The pancreas was edematous with focal pancreatic calcifications. The pancreatic duct measured 6 mm in the head. The common bile duct measured 6 mm just cephalad to the head of the pancreas. The intrahepatic ducts and the gallbladder were normal. The patient responded to conservative treatment and by 72 hours began on clear liquids. By the 4th day of hospitalization, he was tolerating solid foods and the physician was asked for recommendations concerning further diagnostic studies, specifically ERCP.

Subsequent Management

Endoscopic Retrograde Cholangiopancreatography (ERCP) in Chronic Pancreatitis

The gastroenterologist is often asked to evaluate patients such as ours with the diagnosis of "chronic

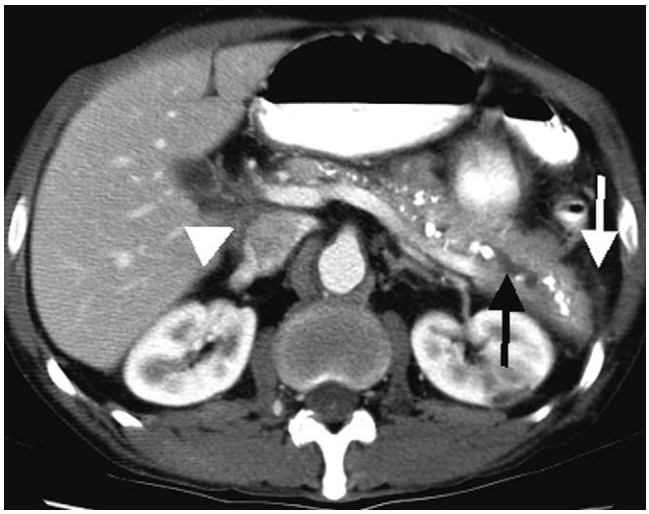

Figure 1. Abdominal commuted tomography scan in patient presented. The pancreatic duct is dilated (*black arrow*) and contains multiple calcified stones diagnostic of chronic pancreatitis. Acute pancreatitis is demonstrated based on fat stranding (*white arrow*) and peripancreatic fluid (*arrowhead*) next to the portal vein.

pancreatitis" suggested usually by clinical course and/or noninvasive abdominal imaging techniques. In a patient with diffuse pancreatic calcification who has an initial bout of clinical acute pancreatitis, the indications for ERCP should be relatively limited. With a long history of alcohol consumption, and the first episode of acute pancreatitis beginning at age 32, other causes of calcific pancreatitis such as familial pancreatitis, tropical pancreatitis, or cystic fibrosis would not be seriously entertained. While a single calcification in the head of the pancreas may suggest the diagnosis of acute gallstone pancreatitis, the multiplicity of calcifications throughout the gland, principally within the pancreatic ductal system, overwhelmingly supports the diagnosis of chronic calcific alcoholic pancreatitis. The common bile duct measuring 6 mm in diameters is at the upper limits of normal, suggesting that there may well be initial fibrotic stricturing of the bile duct in its retropancreatic course. Thus, there is little rational for considering a purely diagnostic ERCP.

Is there any role for therapeutic ERCP, however, in the treatment of a patient who has had a single episode of clinical acute pancreatitis but has extensive pancreatic calcifications supportive of the diagnosis of chronic pancreatitis? Principal to this point would be whether a therapeutic ERCP could prevent recurrent bouts of acute pancreatitis and/or prevent progressive pancreatic dysfunction such as exocrine and/or endocrine insufficiency. It is tempting to postulate that the dilated pancreatic duct together with pancreatic calculi would inevitably lead to future bouts of acute pancreatitis and/or progressive acinar cell and islet cell loss with chronic pancreatitis. Clearly an ERCP will visualize the pancreatic duct and be highly likely to demonstrate irregular ectasia (i.e., "chain of lakes") of the pancreatic duct together with intraductal calculi. There may also be a predominant stricture of the pancreatic duct in the head of the gland not suggested by CT scans. Controlled clinical trials demonstrating the long-term efficacy of ERCP therapy for patients with chronic pancreatitis are, however, lacking. The therapeutic maneuvers undertaken in these patients include pancreatic sphincterotomy, pancreatic stone extraction, and short-term or long-term pancreatic duct stenting.[23–35] These therapeutic strategies are performed under the clinical supposition that pancreatic duct obstruction by stricture or calculi is inevitable, playing a significant role in determining the course of the disease. There is even more controversy as to whether pancreatic duct decompression plays any role in forestalling the progression of exocrine and/or endocrine insufficiency in established patients with chronic pancreatitis.

In 1995, Ponchon et al. reported a decrease in recurrent pain following pancreatic stenting (from 14/14 patients with recurrent pain before drainage to only 8/14 following drainage) and reduced analgesic usage (from 23/23 patients prior to ERCP to 6/23 following drainage).[23] Smits et al. likewise reported favorable results of endoscopic treatment of pancreatic stones.[24] Employing sphincterotomy, pancreatic stenting, mechanical lithotripsy, or extra-corporeal shockwave lithotripsy, they reported early relief of symptoms in 90% of patients who underwent complete stone clearance.[24] Recurrent symptoms appeared in one-third of patients at a mean of 33 months. When analyzing prognostic factors for success of stone extraction therapy, they noted improved pain relief in nonalcoholic (76%) vs. alcoholic patients (67%) ($P = 0.066$). Other features such as the number of stones, the location of calculi, or presence of strictures were not helpful at predicting therapeutic ERCP success.

In the largest retrospective study to date, Rosch et al. followed-up 1000 patients with painful chronic pancreatitis who underwent endoscopic treatment for pancreatic duct strictures and/or stones.[35] At a mean of 4.9 years (range, 2–12 years), long-term success of endoscopic treatment (i.e., significant improvement in pain) was reported in 85% of the entire group but in only 65% in the intention-to-treat analysis. Pancreatic function

was not positively affected by endoscopic therapy. Diabetes was present in 23% initially and in 37% at follow-up. In fact, diabetes improved in only 8% of patient in their study but worsened or developed in 21% of patients.[35] Clinically significant diarrhea, likely related to exocrine insufficiency, was also noted in 37% of patients before ERCP therapy and in 28% following treatment.

For several reasons, the author recommends that no ERCP evaluation be performed on this patient. First, there is virtually no doubt as to the diagnosis in this patient. Secondly, no reliable therapeutic technique can be performed in this patient, especially given that this is the patient's first episode of acute pancreatitis and he has no history of chronic abdominal pain. Surgical and/or endoscopic treatment of the dilatation of the common bile duct is not warranted at present in this patient with normal liver tests.

Conclusion

The approach to the patient with abdominal pain has changed considerably over the past several decades. The advent of sophisticated noninvasive imaging techniques and newer biochemical tests has enhanced the clinician's ability to rapidly assess patients and initiate therapy. The chronic alcoholic patient with abdominal pain presents a considerable challenge to all but one that can be met with directed diagnostic focus, expedited evaluation, appropriate therapy and compassionate follow-up.

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