Forecasting aids patients in the realization process by preventing disorientation. In contrast, stalling and being blunt are extremely disorienting to a patient.

UNDERSTANDING THE PATIENT'S EXPECTATIONS

Technique alone does not suffice when bringing bad news to a patient. The job requires sensitivity, tact, and an understanding of the patient's expectations, which can hamper the patient's understanding of the news. The effectiveness of a strategy depends as much on the patient's expectation and state of mind as on the strategy. The physician should keep in mind the patient's level of understanding, expectations, and culture.

Many patients do not want to know about a bad diagnosis, for cultural reasons or perhaps because it is the only way they can cope. As much as the physician feels that the truth must be faced, he or she must be sensitive to what the patient feels is adequate information.

For instance, in Ethiopia, bad news is not given in the afternoon, as it is thought to induce a restless night. In Japan, physicians traditionally tell the patient nothing that might cause him or her to lose heart, but they do inform the patient's family. The process is an elaborate but superficial effort at concealment, a dance around rather than a concealment of the truth.

In this country, attitudes about giving bad news have reversed. A 1961 survey found that 90% of physicians would not reveal the finding of cancer to a patient; by 1979, a similar survey found that 97% of physicians would reveal the diagnosis.

A physician must be sensitive to the context of the patient and family, and how much understanding they have about what is happening. The amount of forecasting needed will be very different for the family who has coped for months with a family member's terminal illness, as opposed to a patient or family facing the outcome of a sudden accident. ■

SUGGESTED READING

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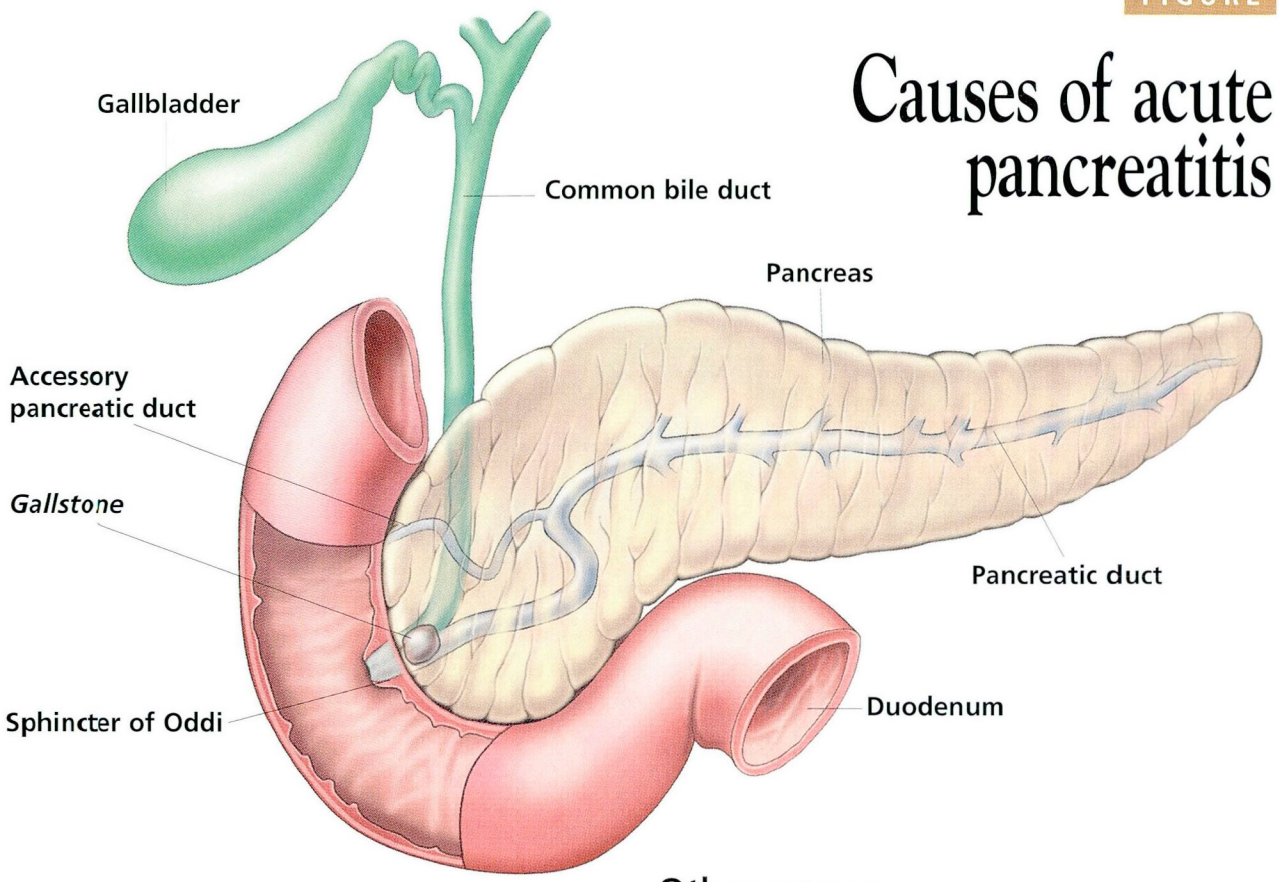
Diagnosis and management of acute pancreatitis

The course of acute pancreatitis is mild and self-limited in most patients, but complications occur in 25%, and the overall mortality rate is 5% to 10%.[1] Timely recognition and management of factors that indicate severe disease may prevent catastrophic outcomes. Treatment is still mostly supportive, but endoscopic retrograde cholangiopancreatography (ERCP; for pancreatitis caused by gallstones) and empiric antibiotic therapy are under study and may be reasonable in certain situations. Studies of other therapies are underway as well.

FIGURE

Causes of acute pancreatitis



Obstructive causes

Gallstones (approximately 45% of all cases)
Tumors
Worms or foreign bodies
Pancreas divisum
Choledochocele
Periampullary duodenal diverticula
Hypertensive sphincter of Oddi

Other causes

Toxins or drugs
  Alcoholism (approximately 35% of all cases)
Trauma
Metabolic abnormalities
  Hypertriglyceridemia, hypercalcemia
Inherited conditions
Infections
Vascular abnormalities
Idiopathic causes (approximately 10% of all cases)

POTENTIAL COMPLICATIONS

Early complications

In the first 7 days of illness, several factors can lead to failure of one or more organs, admission to an intensive care unit (ICU), or death:

Shock due to extravasation of plasma or blood or both into the retroperitoneum.

Pulmonary failure, perhaps caused by released enzymes or cytokines, factors that affect pulmonary capillaries or alveoli or both.

Renal failure due to hypotension.

Multiorgan failure accounts for most deaths during the first week.

Late complications

After 7 days, potential complications include:

- Pancreatic pseudocyst, abscess, or infection of necrotic tissue.
- Hemorrhage from pancreatic vessels (pseudoaneurysm formation).
- Perforation, obstruction, and fistulization of the colon.

Necrotic pancreatic tissue becomes infected in 40% to 60% of cases, and such infections account for most deaths after the first week of acute pancreatitis. Fine-needle aspiration biopsy of the pancreas or collected pancreatic fluid can confirm the diagnosis.[2]

TABLE

FACTORS THAT PREDICT SEVERITY OF DISEASE IN ACUTE PANCREATITIS*

Factor	Non–gallstone-related pancreatitis	Gallstone-related pancreatitis
On admission		
Age	> 55 yr	> 70 yr
White blood cell count	> 16 000/mm^3	> 18 000/mm^3
Serum glucose level	> 200 mg/dL	> 220 mg/dL
Lactic dehydrogenase level	> 350 U/L	> 400 U/L
Aspartate aminotransferase level	> 250 U/L	> 250 U/L
Within 48 hours of admission		
Decrease in hematocrit	> 10%	> 10%
Increase in blood urea nitrogen	> 5 mg/dL	> 2 mg/dL
Calcium level	< 8 mg/dL	< 8 mg/dL
Partial pressure of oxygen (Po_2)	< 60 mm Hg	...
Base deficit	> 4 mmol/L	> 5 mmol/L
Fluid deficit	> 6 L	> 4 L

*According to Ranson and colleagues, reference 4; presence of three or more risk factors indicates severe disease

Every patient with a first attack of acute pancreatitis should have a triglyceride determination on admission

ETIOLOGY: GALLSTONES, ALCOHOLISM, OR SOMETHING ELSE?

Gallstones cause approximately 45% of cases of acute pancreatitis; alcohol abuse causes 35% (although alcohol abuse cases may outnumber gallstone cases in inner-city hospitals). Less-common causes include hypertriglyceridemia, drug reactions, pancreas divisum, and sphincter of Oddi dysfunction; 10% of cases are idiopathic.

Every patient with a first attack of acute pancreatitis should have a triglyceride determination on admission, as levels often return to normal within 2 to 3 days. A triglyceride level greater than 1000 mg/dL can be an initiating factor in acute pancreatitis, and reducing the level to less than 500 mg/dL helps prevent future attacks.

MARKERS OF SEVERITY

Serum amylase, lipase, and trypsinogen levels do *not* correlate with severity of disease. Attacks are more severe in obese patients.

Ranson and colleagues[3,4] (TABLE) and researchers in Glasgow[5] developed lists of risk factors commonly used to predict severity of disease, but these scoring systems have a disadvantage in clinical use in that some of the factors can be determined only by comparing the admission measurement with a repeat measurement within 48 hours of admission, delaying the assessment of disease severity and possibly the initiation of appropriate treatment. The APACHE II (*a*cute *p*hysiology *a*nd *c*hronic disease *e*valuation) score, in contrast, can be calculated at any time; scores greater than 9 predict a severe course. However, the positive predictive value of the Ranson, Glasgow, and APACHE II scores are only approximately 40% to 50%, but their negative predictive values are about 90% to 95%. Thus, they are better able to predict mild disease than severe disease.

Other tests that can help determine severity include the peritoneal tap ("prune juice" ascitic fluid indicates severe, necrotizing pancreatitis); levels of C-reactive protein (levels > 120 mg/L predict severe disease), trypsinogen-activation peptide, granulocytic elastase, and interleukin-6; and dynamic computed tomographic (CT) pancreatography.

IMAGING TESTS IN ACUTE PANCREATITIS

Sonography and computed tomography are the imaging tests used most frequently in diagnosing acute pancreatitis. The tests are complementary: the sonogram provides better images of the gallbladder and common bile duct, and the CT scan provides better images of the pancreas.

Looking for gallstones

Although sonography is the best test for gallstones, it is only 70% to 80% sensitive in acute pancreatitis because overlying gas caused by paralytic ileus can obscure the pancreas. If the initial sonogram reveals no gall-

stones, a repeat sonogram after the acute attack remits and air has cleared the bowel may reveal them.

The liver enzyme profile on admission to the hospital may indicate gallstones even if the sonogram is negative. The alanine aminotransferase (ALT) level is the best single marker of biliary tract disease in acute pancreatitis—the higher the ALT level on admission, the greater the probability that gallstones caused the attack. An ALT level three times higher than normal (approximately 150 U/L) on admission has a 95% positive predictive value for gallstone pancreatitis. However, the sensitivity of the admission ALT level for gallstones is only 50%; therefore, a normal ALT level does not rule out gallstones.

CT scanning to check the pancreas

A CT scan should be obtained in every patient presenting with symptoms of a first attack of acute pancreatitis. The CT scan is the best single test to rule out causes other than acute pancreatitis (ie, perforated viscus, obstructed bowel, cystic or solid masses) in patients with severe abdominal pain. A CT scan may occasionally reveal gallstones missed by sonography, or rarely a tumor or cystic mass that may have caused the attack. A normal CT scan rules out severe disease.

A dynamic CT scan should be obtained to differentiate edematous (interstitial) from necrotizing pancreatitis. Edematous pancreatitis is consistent with mild attacks, although this relationship is not 100%. Necrotizing pancreatitis is consistent with severe attacks.

Concern has been raised that dynamic CT scanning may injure the pancreas by decreasing the microcirculation to the organ. A recent study showed that patients with acute pancreatitis who underwent an early CT scan had longer episodes of clinical pancreatitis than those who did not.[6] This study, however, was retrospective; prospective data are needed to answer the question.

CRITERIA FOR ICU ADMISSION

Early in the attack, I would send a patient to the ICU immediately if he or she has either:

Pulmonary insufficiency, marked by tachypnea, which warrants a blood-gas determination immediately. If the P_{O_2} is low, consider admitting the patient to an ICU or step-down unit.

Vascular fluid problems such as low blood pressure, low urine output, and high fluid requirements. These patients are at risk of vascular collapse and are best managed in an ICU.

All patients admitted to the ICU should be monitored for pancreatic infection. An initial fine-needle aspiration biopsy should be considered 7 to 14 days after ICU admission if unexplained fever or an elevated white blood count persists, with a follow-up aspiration biopsy as needed.

TREATMENT

Mild, self-limited acute pancreatitis is treated by withholding food and oral fluids and giving intravenous fluids and analgesics. A nasogastric tube is needed only if significant ileus or nausea or vomiting are present.

Patients with severe acute pancreatitis need total parenteral nutrition and treatment for complications, such as massive fluid replacement for shock, endotracheal intubation and respiratory support for pulmonary insufficiency, peritoneal dialysis for renal failure, and intravenous calcium, glucose, and magnesium.

Endoscopic retrograde cholangiopancreatography may reduce complications if gallstones are suspected of causing an attack, although its effect on the mortality rate in severe pancreatitis is controversial. Of four randomized, controlled studies of emergency ERCP to remove gallstones,[7–10] two showed decreases in complications and one showed a lower mortality rate, but one showed an increase in pulmonary failure. At present, it appears reasonable to consider emergency ERCP if a gallstone is believed to be impacted in the common duct (with increasing jaundice) and the patient has severe disease as manifested by pulmonary insufficiency, vascular problems, renal insufficiency, or cholangitis.

Mortality is due to multiorgan failure or pancreatic infection

Enzyme inhibitors have produced mixed results. Aprotinin was studied in five trials, one of which showed a decrease in mortality.[11] A recent meta-analysis of five randomized trials of gabexate mesilate (a new enzyme inhibitor) revealed a significant reduction in complications.[12] An ERCP study demonstrated that gabexate can prevent ERCP pancreatitis more effectively than placebo.[13]

Cytokine inhibitors are being tested. In a promising initial randomized, placebo-controlled study, lexipafant, a potent receptor antagonist of platelet-activating factor, reduced the incidence of early multiorgan fail-

ure in acute pancreatitis.[14] A follow-up study from England showed a halving of the mortality rate.

Antibiotics are now a consideration for patients with severe pancreatitis without documented infection. In three studies of broad-spectrum antibiotic treatment,[15–17] treated patients had a lower overall mortality rate in one study, a lower pancreatic infection rate in two studies, and a lower nonpancreatic infection rate in two studies compared with patients who did not receive antibiotics. Broad-spectrum antibiotics are therefore reasonable to add to the regimen, particularly for patients with severe disease (ie, necrosis on the CT scan, those admitted to the ICU). The antibiotics found to be helpful are intravenous imipenem, cefuroxime, and oral and rectal nonabsorbable antibiotics. ■

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