# American College of Radiology
# ACR Appropriateness Criteria®

**Clinical Condition:** **Acute Pancreatitis**

**Variant 1:** **Etiology unknown, first episode of pancreatitis.**

| Radiologic Procedure | Rating | Comments | RRL* |
|---|---|---|---|
| US abdomen | 8 | | None |
| CT abdomen | 6 | With or without contrast. | Med |
| MRI abdomen with contrast | 6 | | None |
| MRI abdomen with MRCP | 6 | | None |
| US abdomen endoscopic | 5 | | None |
| **Rating Scale:** 1=Least appropriate, 9=Most appropriate | | | *Relative Radiation Level |

**Variant 2:** **Severe abdominal pain, elevated amylase lipase, no fever or evidence of fluid loss at admission; clinical score pending.**

| Radiologic Procedure | Rating | Comments | RRL* |
|---|---|---|---|
| US abdomen | 8 | | None |
| MRI abdomen with MRCP | 7 | | None |
| CT abdomen | 7 | With or without contrast. | Med |
| MRI abdomen with contrast | 6 | | None |
| **Rating Scale:** 1=Least appropriate, 9=Most appropriate | | | *Relative Radiation Level |

**Variant 3:** **Severe abdominal pain, elevated amylase lipase, 48 hours later assuming no improvement or degradation (assume no prior imaging).**

| Radiologic Procedure | Rating | Comments | RRL* |
|---|---|---|---|
| CT abdomen | 8 | With or without contrast. | Med |
| US abdomen | 7 | | None |
| MRI abdomen with contrast | 7 | | None |
| MRI abdomen with MRCP | 7 | | None |
| **Rating Scale:** 1=Least appropriate, 9=Most appropriate | | | *Relative Radiation Level |

**Clinical Condition:** **Acute Pancreatitis**

**Variant 4:** **Severe abdominal pain, elevated amylase lipase, fever and elevated white blood cell count.**

| Radiologic Procedure | Rating | Comments | RRL* |
|---|---|---|---|
| CT abdomen | 9 | With or without contrast. | Med |
| MRI abdomen with MRCP | 7 | | None |
| MRI abdomen with contrast | 7 | | None |
| US abdomen | 7 | | None |
| **Rating Scale:** 1=Least appropriate, 9=Most appropriate | | | *Relative Radiation Level |

**Variant 5:** **Severe abdominal pain, elevated amylase lipase, hemoconcentration, oliguria, tachycardia.**

| Radiologic Procedure | Rating | Comments | RRL* |
|---|---|---|---|
| CT abdomen | 9 | With or without contrast. | Med |
| US abdomen | 7 | | None |
| MRI abdomen with MRCP | 7 | | None |
| MRI abdomen with contrast | 7 | | None |
| **Rating Scale:** 1=Least appropriate, 9=Most appropriate | | | *Relative Radiation Level |

# ACUTE PANCREATITIS

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## Summary of Literature Review

This document focuses on the diagnosis and initial evaluation of patients with suspected or known acute pancreatitis. It does not address interventional procedures or documentation of complications such as abscess, pseudocyst, or pseudoaneurysm.

Interstitial edematous pancreatitis and necrotizing pancreatitis are the most frequent clinical manifestations of acute pancreatitis. Fluid collections associated with acute pancreatitis usually resolve spontaneously. Pancreatic pseudocysts are fluid collections that persist for 6 weeks or more. Pancreatic abscess is usually a complication of necrotizing pancreatitis, typically developing after 3 to 5 weeks. Determinants of the natural course of acute pancreatitis are pancreatic parenchymal necrosis, extrapancreatic retroperitoneal fatty tissue necrosis, biologically active compounds in pancreatic ascites, and infection of necrosis [1]. Early in the course of acute pancreatitis, multiple organ failure is the consequence of various inflammatory mediators that are released from the inflammatory process and from activated leukocytes attracted by pancreatic injury. Late in the course, starting the second week, local and systemic septic complications are dominant. Around 80% of deaths in acute pancreatitis are caused by septic complications.

The infection of pancreatic necrosis occurs in 8%-12% of acute pancreatitis patients and in 30%-40% of patients with necrotizing pancreatitis. Pancreatic inflammation may result in enlargement of the gland, peripancreatic inflammation with or without fluid, solitary or loculated fluid collections, necrosis of pancreatic parenchyma, and subsequent infection in any of the above sites of inflammation. Distant organ complications can lead to organ failure, protracted course, and death [2]. Prediction of which patients will develop these complications is achieved through clinical scoring systems and imaging findings. Choice of scoring system is beyond the scope of these recommendations.

Acute pancreatitis is suspected in patients presenting with epigastric upper abdominal pain that is acute in onset, rapidly increasing in severity, and persistent without relief. The intensity of the pain almost always results in the patient seeking medical attention. Differential diagnosis includes mesenteric ischemia, perforated ulcer, intestinal obstruction, biliary colic, and myocardial infarction. Serum amylase and/or lipase levels can be considered diagnostic when the reported value(s) is ≥3 times normal. Lipase levels are more specific for acute pancreatitis, as hyperamylasemia may be present in a variety of conditions. Of note is that serum enzyme levels do not correlate with the severity of the disease [2]. Consequently, clinical scoring systems and imaging tests have been advocated to classify individual patients. Furthermore, the diagnosis may be overlooked in the absence of typical enzyme elevation [3]. In some patients, acute pancreatitis may be present in the absence of enzyme abnormalities [4].

Imaging tests available for the diagnosis of acute pancreatitis include transabdominal ultrasound (US), endoscopic ultrasound (EUS), computed tomography (CT) scanning, magnetic resonance imaging (MRI), and magnetic resonance cholangiopancreatography (MRCP) [5-7]. Imaging tests are performed for various reasons, including detection of gallstones, detection of biliary obstruction, diagnosis of pancreatitis when the clinical situation is unclear, identification of patients with high-risk pancreatitis, and detection of complications of pancreatitis.

US to detect gallbladder stones should be performed in every patient with acute pancreatitis, even alcoholics [8]. US is also effective in diagnosing biliary obstruction, which, when present, often prompts endoscopic retrograde cholangiopancreatography (ERCP) to relieve the cause of obstruction [9]. US is less successful in diagnosing choledocholithiasis [10] and has limited applications in the early staging of the disease. Visualization of the pancreas is often impaired because of overlying bowel gas, and the detection of intraparenchymal and retroperitoneal fluid collections correlates poorly with pancreatic necrosis [8]. US with color Doppler is useful to detect venous complications of acute pancreatitis [11]. In patients with suspected acute gallstone pancreatitis or with repeating acute pancreatitis, ERCP is used to reach a definite diagnosis and to investigate the etiology. EUS is useful, when needed

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clinically, to detect common duct stones when initial studies are negative. It can often determine an etiology (usually biliary) in patients initially diagnosed with idiopathic acute pancreatitis [12-15].

CT is an insensitive detector of biliary calculi, but is superb in delineating the pancreas and acute pancreatitis-associated abnormalities. CT scanning provides clear images of the pancreas and adjacent structures [16] and allows for the differentiation of acute pancreatitis from other abdominal diseases. CT findings helpful for diagnosing acute pancreatitis include pancreatic enlargement, peripancreatic inflammatory changes, fluid collections, and uneven density of pancreatic parenchyma.

MRI demonstrates pancreatic enlargement and the inflammatory changes around the pancreas [7]. It has the advantage of no x-ray exposure. Nevertheless, it takes a much longer time to scan the pancreas in comparison with CT. MRCP has a high accuracy in detecting bile duct stones [17].

Physiologically based scoring systems such as the APACHE II and Ranson's criteria are designed to identify early prognostic signs that predict severity of clinical course in an individual patient. In 1985, Balthazar et al [10] showed that although clinical scoring systems were highly correlated with increasing CT severity, disease severity was sometimes underestimated by clinical scoring alone. The key criterion for identifying patients at higher risk for fatal pancreatitis is the presence of pancreatic necrosis [4,8,18-21]. Balthazar et al [22] revised their scoring system in 1990 to account for the significance of pancreatic necrosis and created the CT severity index. The utility of the Ranson's criteria compared with that of the Balthazar CT severity index for predicting the necessity for admission to an ICU in patients with acute pancreatitis was analyzed in a recent study. The Balthazar CT severity index correlated highly with the overall occurrence of complications ($r^2$=0.96), the occurrence of sepsis ($r^2$=0.99), and death ($r^2$=0.99), and it was a better prognostic indicator than the Ranson criteria for complications and mortality [23]. A modified CT severity index, which simplifies the evaluation of pancreatic necrosis, inflammatory changes, and extrapancreatic complications, has also been proposed [24]. There are isolated reports of clinical scoring systems yielding equivalent or superior results to imaging tests [25,26]. However, it also should be remembered that most clinical systems require a second assessment within 48 hours to monitor progression or stability, as opposed to relatively instantaneous evaluation at imaging.

Contrast CT and/or gadolinium-enhanced MRI [20,27-31] can both be used to assess pancreatic necrosis and evaluate peripancreatic inflammation and fluid collections. MRI is particularly useful in patients who cannot receive iodinated contrast material due to prior adverse contrast reaction or renal insufficiency. Furthermore, the integrity of the pancreatic duct can be assessed by means of MRCP in an MRI study; this is important, since in previous studies pancreatic duct rupture was reported in about 30% patients with acute pancreatitis [32]. In both CT and MRI studies of the pancreas, pancreatic necrosis can be diagnosed when segments of pancreatic parenchyma do not enhance on images obtained following intravenous contrast administration. These unenhanced areas have been proved to represent necrotic regions when correlated with findings at pancreatic debridement [33]. While some have suggested that the site of necrosis within the pancreas may further predict outcome [19], others have found no such correlation [4]. The presence of peripancreatic fluid collections is usually associated with severe disease [10,25,29]. Echo-enhanced US has been recently reported as a new initial imaging approach [34]; it can be used as an alternative in patients in whom both CT and MRI are contraindicated.

Controversy has emerged because of the observation that intravenous contrast impairs the microcirculation of the pancreas in rats with acute necrotizing pancreatitis and may increase the severity of the disease [35,36]. These results could not be reproduced in the opossum [37]. No prospective human trials have been published to date. Most experts believe the benefits of detecting necrosis outweigh any potential risk.

No objective clinical selection criteria exist that can determine which patients should have CT to assess the risk of severe pancreatitis. Imaging is clearly indicated when the cause of abdominal pain is unclear. In patients with known acute pancreatitis, however, CT is reserved for patients with clinical, biochemical, or physiologic indications of severe disease [2]. There is no information suggesting that routine CT in patients with milder disease (low APACHE II or Ranson scores) would result in upstaging a significant number of patients.

## References


1. Beger HG, Rau B, Mayer J, Pralle U. Natural course of acute pancreatitis. *World J Surg* 1997; 21(2):130-135.
2. Banks PA. Practice guidelines in acute pancreatitis. *Am J Gastroenterol* 1997; 92(3):377-386.
3. Lankisch PG, Schirren CA, Kunze E. Undetected fatal acute pancreatitis: why is the disease so frequently overlooked? *Am J Gastroenterol* 1991; 86(3):322-326.
4. Lankisch PG, Struckmann K, Lehnick D. Presence and extent of extrapancreatic fluid collections are indicators of severe acute pancreatitis. *Int J Pancreatol* 1999; 26(3):131-136.
5. Lecesne R, Taourel P, Bret PM, Atri M, Reinhold C. Acute pancreatitis: interobserver agreement and correlation of CT and MR cholangiopancreatography with outcome. *Radiology* 1999; 211(3):727-735.

6. Robinson PJ, Sheridan MB. Pancreatitis: computed tomography and magnetic resonance imaging. *Eur Radiol* 2000; 10(3):401-408.
7. Piironen A, Kivisaari R, Kemppainen E, et al. Detection of severe acute pancreatitis by contrast-enhanced magnetic resonance imaging. *Eur Radiol* 2000; 10(2):354-361.
8. Balthazar EJ. Acute pancreatitis: assessment of severity with clinical and CT evaluation. *Radiology* 2002; 223(3):603-613.
9. Pezzilli R, Billi P, Barakat B, D'Imperio N, Miglio F. Ultrasonographic evaluation of the common bile duct in biliary acute pancreatitis patients: comparison with endoscopic retrograde cholangiopancreatography. *J Ultrasound Med* 1999; 18(6):391-394.
10. Balthazar EJ, Ranson JH, Naidich DP, Megibow AJ, Caccavale R, Cooper MM. Acute pancreatitis: prognostic value of CT. *Radiology* 1985; 156(3):767-772.
11. Dorffel T, Wruch T, Ruckert RI, Romaniuk P, Dorffel Q, Wermke W. Vascular complications in acute pancreatitis assessed by color duplex ultrasonography. *Pancreas* 2000; 21(2):126-133.
12. Chak A, Hawes RH, Cooper GS, et al. Prospective assessment of the utility of EUS in the evaluation of gallstone pancreatitis. *Gastrointest Endosc* 1999; 49(5):599-604.
13. Froussard JL, Sosa-Valencia L, Amouyal G, Marty O, Hadengue A, Amouyal P. Usefulness of endoscopic ultrasonography in patients with "idiopathic" acute pancreatitis. *Am J Med* 2000; 109(3):196-200.
14. Norton SA, Alderson D. Endoscopic ultrasonography in the evaluation of idiopathic acute pancreatitis. *Br J Surg* 2000; 87(12):1650-1655.
15. Lui CL, Lo CM, Chan JK, Poon RT, Fan ST. EUS for detection of occult cholelithiasis in patients with idiopathic pancreatitis. *Gastrointest Endosc* 2000; 51(1):28-32.
16. Silverstein W, Isikoff MB, Hill MC, Barkin J. Diagnostic imaging of acute pancreatitis: prospective study using CT and sonography. *AJR* 1981; 137(3):497-502.
17. Chan YL, Chan AC, Lam WW, et al. Choledocholithiasis: comparison of MR cholangiography and endoscopic retrograde cholangiography. *Radiology* 1996; 200(1):85-89.
18. Kivisaari L, Schroeder T, Sainio V, Somer K, Standertskjold-Nordenstam CG. CT evaluation of acute pancreatitis: 8 years clinical experience and experimental evidence. *Acta Radiol Suppl* 1991; 377:20-24.
19. London NJ, Neoptolemos JP, Lavelle J, Bailey I, James D. Contrast-enhanced abdominal computed tomography scanning and prediction of severity of acute pancreatitis: a prospective study. *Br J Surg* 1989; 76(3):268-272.
20. Dalzell DP, Scharling ES, Ott DJ, Wolfman NT. Acute pancreatitis: the role of diagnostic imaging. *Crit Rev Diag Imaging* 1998; 39(5):339-363.
21. Casas JD, Diaz R, Valderas G, Mariscal A, Cuadras P. Prognostic value of CT in the early assessment of patients with acute pancreatitis. *AJR* 2004; 182(3):569-574.
22. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JH. Acute pancreatitis: value of CT in establishing prognosis. *Radiology* 1990; 174(2):331-336.
23. Vriens PW, van de Linde P, Slotema ET, Warmerdam PE, Breslau PJ. Computed tomography severity index is an early prognostic tool for acute pancreatitis. *J Am Coll Surg* 2005; 201(4):497-502.
24. Mortele KJ, Wiesner W, Intriere L, et al. A modified CT severity index for evaluating acute pancreatitis: improved correlation with patient outcome. *AJR* 2004; 183(5):1261-1265.
25. Foitzik T, Bassi DG, Schmidt J, et al. Intravenous contrast medium accentuates the severity of acute necrotizing pancreatitis in the rat. *Gastroenterology* 1994; 106(1):207-214.
26. Meek K, de Virgilio C, Murrell Z, et al. Correlation between admission laboratory values, early abdominal computed tomography values, early abdominal computed tomography, and severe complications of gallstone pancreatitis. *Am J Surg* 2000; 180(6):556-560.
27. Ward J, Chalmers AG, Guthrie AJ, Larvin M, Robinson PJ. T2-weighted and dynamic enhanced MRI in acute pancreatitis: comparison with contrast enhanced CT. *Clin Radiol* 1997; 52(2):109-114.
28. Lucarotti ME, Virjee J, Alderson D. Patient selection and timing of dynamic computed tomography in acute pancreatitis. *Br J Surg* 1993; 80(11):1393-1395.
29. van den Biezenbos AR, Kruyt PM, Bosscha K, et al. Added value of CT criteria compared to the clinical SAP score in patients with acute pancreatitis. *Abdom Imaging* 1998; 23(6):622-626.
30. Simchuk EJ, Traverso LW, Nukui Y, Kozarek RA. Computed tomography severity index is a predictor of outcomes for severe pancreatitis. *Am J Surg* 2000; 179(5):352-355.
31. Arvanitakis M, Delhaye M, De Maertelaere V, et al. Computed tomography and magnetic resonance imaging in the assessment of acute pancreatitis. *Gastroenterology* 2004; 126(3):715-723.
32. Lau ST, Simchuk EJ, Kozarek RA, Traverso LW. A pancreatic ductal leak should be sought to direct treatment in patients with acute pancreatitis. *Am J Surg* 2001; 181(5):411-415.
33. Kemppainen E, Sainio V, Haapiainen R, Kivisaari L, Kivilaakso E, Puolakkainen P. Early localization of necrosis by contrast-enhanced computed tomography can predict outcome in severe acute pancreatitis. *Br J Surg* 1996; 83(7):924-929.
34. Rickes S, Uhle C, Kahl S, et al. Echo enhanced ultrasound: a new valid initial imaging approach for severe acute pancreatitis. *Gut* 2006; 55(1):74-78.
35. Foitzik T, Bassi DG, Fernandez-del Castillo C, Warshaw AL, Rattner DW. Intravenous contrast medium impairs oxygenation of the pancreas in acute necrotizing pancreatitis in the rat. *Arch Surg* 1994; 129(7):706-711.
36. Kaiser AM, Grady T, Gerdes D, Saluja M, Steer ML. Intravenous contrast medium does not increase the severity of acute necrotizing pancreatitis in the opossum. *Dig Dis Sci* 1995; 40(7):1547-1553.
37. Saifuddin A, Ward J, Ridgway J, Chalmers AG. Comparison of MR and CT scanning in severe acute pancreatitis: initial experiences. *Clin Radiol* 1993; 48(2):111-116.