J Hepatobiliary Pancreat Sci (2010) 17:24–36
DOI 10.1007/s00534-009-0214-3

New diagnostic criteria of acute pancreatitis

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Received: 1 August 2009 / Accepted: 1 September 2009 / Published online: 11 December 2009
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Abstract Practical guidelines for the diagnosis of acute pancreatitis are presented so that a rapid and adequate diagnosis can be made. When acute pancreatitis is suspected in patients with acute onset of abdominal pain and tenderness mainly in the upper abdomen, the diagnosis of acute pancreatitis is made on the basis of elevated levels of pancreatic enzymes in the blood and/or urine. Furthermore, other acute abdominal diseases are ruled out if local findings associated with pancreatitis are confirmed by diagnostic imaging. According to the diagnostic criteria established in Japan, patients who present with two of the following three manifestations are diagnosed as having acute pancreatitis: characteristic upper abdominal pain, elevated levels of pancreatic enzymes, and findings of

This article is based on the studies first reported in the JPN guidelines for the management of acute pancreatitis. 3rd ed. JPN Guidelines 2010 (in Japanese). Tokyo: Kanehara; 2009.

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ultrasonography (US), CT or MRI suggesting acute pancreatitis. Detection of elevated levels of blood pancreatic enzymes is crucial in the diagnosis of acute pancreatitis. Measurement of blood lipase is recommended, because it is reported to be superior to all other pancreatic enzymes in terms of sensitivity and specificity. For measurements of the blood amylase level widely used in Japan, it should be cautioned that, because of its low specificity, abnormal high values are also often obtained in diseases other than pancreatitis. The cut-off level of blood pancreatic enzymes for the diagnosis of acute pancreatitis is not able to be set because of lack of sufficient evidence and consensus to date. CT study is the most appropriate procedure to confirm image findings of acute pancreatitis. Elucidation of the etiology of acute pancreatitis should be continued after a diagnosis of acute pancreatitis. In the process of the etiologic elucidation of acute pancreatitis, judgment whether it is gallstone-induced or not is most urgent and crucial for deciding treatment policy including the assessment of whether endoscopic papillary treatment should be conducted or not. The diagnosis of gallstone-induced acute pancreatitis can be made by combining detection of elevated levels of bilirubin, transamylase (ALT, AST) and ALP detected by hematological examination and the visualization of gallstones by US.

Keywords Acute pancreatitis · Guidelines · Diagnostic criteria · Etiology · Gallstone pancreatitis · Clinical indicators

Introduction

The diagnosis of acute pancreatitis is determined on the basis of acute onset of abdominal pain and tenderness mainly in the upper abdomen, elevated levels of pancreatic enzymes in the blood and/or urine and findings of pancreatitis detected by diagnostic imaging such as ultrasonography (US) and CT. Other abdominal diseases should be ruled out. After the diagnosis of acute pancreatitis has been made, its etiology should be made clear to decide treatment policy of acute pancreatitis or to prevent the recurrence of pancreatitis.

The diagnostic criteria established by the Japanese Ministry of Health, Labour, and Welfare were revised in part in 2008. The present article shows a detailed description of the new diagnostic criteria. Based on up-to-date evidence, also reviewed are hematological examination, urinalysis, and various types of diagnostic imaging in the diagnosis of acute pancreatitis, and the clinical significance of etiological search.

Diagnostic criteria

CQ1 What are the diagnostic criteria for acute pancreatitis?

1. Acute abdominal pain and tenderness in the upper abdomen.
2. Elevated levels of pancreatic enzymes in the blood or urine.
3. Abnormal findings of acute pancreatitis detected by US, CT or MRI.

Patients who present with at least two of the above three manifestations and in whom other pancreatic diseases and acute abdomen have been ruled out are diagnosed as having acute pancreatitis. However, acute aggravation in chronic pancreatitis should be included as the category of acute pancreatitis.

Note: Measurement of pancreatic enzymes (such as pancreatic amylase and lipase) with high specificity for the pancreas is desirable.

In the diagnostic criteria of acute pancreatitis established by the Japanese Ministry of Health, Labour, and Welfare 2008 [1], a diagnosis of acute pancreatitis is made if the patient presents with at least two of the following three manifestations: acute attack of abdominal pain and tenderness in the upper abdomen, elevated levels of pancreatic leaking enzymes and findings of the pancreas detected by US, CT or MRI. Because individual cut-off levels differ depending upon reports (Table 1) [2–13], at present there is neither sufficient evidence nor consensus to support the setting of cut-off levels.

Indicated for differential diagnosis is acute abdomen causing abdominal pain, which also arises from perforation of the alimentary tract, acute cholecystitis, ileus, mesenteric artery occlusion and acute aortic dissection.

Clinical symptoms and signs

CQ2 What are clinical symptoms and signs in patients in whom acute pancreatitis is suspected?

Acute pancreatitis should be differentiated from other abdominal diseases in patients who have acute onset of abdominal pain and tenderness mainly in the upper abdomen (Recommendation A)

It is reported that more than 90% of patients with acute pancreatitis complain of abdominal pain (Table 2) (Level 3b–5) [14–16]. Clinical symptoms and signs most characteristic of acute pancreatitis are acute attack of abdominal pain and tenderness in the upper abdomen. It is reported that abdominal pain occurs most frequently in the upper abdomen followed by the whole abdomen, while tenderness occurs most frequently in the whole abdomen followed by the upper abdomen and the right

Table 1 Diagnostic ability of pancreatic enzyme measurements for acute pancreatitis

Author	Year	*n* (AP)	Methodology	Upper limit of normal	Cut-off values	Sensitivity	Specificity	PPV	NPV	PLR	NLR	AUC
Lipase												
Steinberg et al. [2]	1985	163 (39)	Turidimetric	72	75	86.5	99.0	97.0	95.1	86.50	0.14	
Ventrucci et al. [3]	1986	189 (12)	ELISA	62	62	91.7	84.7	42.3	98.9	5.99	0.10	
Thomson et al. [4]	1987	168 (×)	Seragen-lipase	68	68	100.0	96.0	85.0	100.0	25.00	0.00	
Jang et al. [5]	2007	193 (17)	Turbidimetric	100.0	300.0	53.0	99.0			9.00	0.05	
Petrov et al. [6]	2007	178 (64)	Turbidimetric	60.0	180.0	92.0	94.0	89.0	95.0			0.960
Sáezet al. [7]	2005	72 (50)	Turbidimetric	60.0	180.0	84.0	85.7	93.4	72.0	5.87	0.19	
Chenet al. [8]	2005	165 (98)	Turbidimetric	190.0	570.0	94.0	92.9	90.0	95.8	13.24	0.06	
Kylänpää-Bäck et al. [9]	2002	237 (29)	Turbidimetric	200.0	200.0	79.0	88.0	49.0	97.0	6.58	0.24	
					600.0	55.0	99.0	84.0	94.0	55.00	0.45	
Wilson et al. [10]	2005	188 (29)	Turbidimetric	190.0	570.0	100.0	99.0	97.0	100.0	100.00	0.00	
Amylase												
Steinberg et al. [2]	1985	163 (39)	Phadebas	326	326	94.9	86.0	75.5	97.4	6.78	0.06	
					600	92.3	100.0	100.0	96.6	ND	0.08	
Pace et al. [11]	1985	121 (21)	Phadebas	300	300	100.0	71.6	15.6	100.0	3.52	0.00	
Ventrucci et al. [3]	1986	189 (12)	Phadebas	377	377	91.7	77.8	35.5	98.6	4.13	0.11	
Thomson et al. [4]	1987	168 (×)	Phadebas	316	316	95.6	97.6	91.7	98.8	39.83	0.05	
					1000	60.9	100.0	100.0	90.4	∞	0.39	
Jang et al. [5]	2007	192 (17)	Turbidimetric	100.0	570.0	100.0	99.0	97.0	100.0	100.00	0.00	
Raty et al. [12]	2007	51 (13)	Turbidimetric		300.0	41.0	95.0			1.40	0.09	0.731
					×2 (Upper limit of normal)							0.654
Petrov et al. [6]	2007	177 (64)	Turbidimetric	100.0	×3 (Upper limit of normal)							0.910
Sáez et al. [7]	2005	72 (50)	Turbidimetric	100.0	300.0	77.0	95.0	89.0	87.0			
Chen et al. [8]	2005	165 (98)	Turbidimetric	190.0	330.0	74.0	86.4	92.5	59.3	5.44	0.30	
Wilson et al. [10]	2005	188 (29)	Turbidimetric	108.0	570.0	94.9	91.4	86.9	88.5	11.03	0.06	
p-Amylase												
Koehler et al. [13]	1982	37 (×)	Cellulose Electropheresis	52	324.0	63.0	99.0	95.0	93.0	63.00	0.37	
Steinberg et al. [2]	1985	163 (39)	Wheat Protein Inhibitor	181	181	92.3	85.1	73.5	96.1	6.19	0.09	
					375	84.0	96.5	91.7	93.3	24.00	0.17	
Pace et al. [11]	1985	121 (21)	Cellulose Electropheresis	120	225	100.0	48.9	17.9	100.0	1.96	0.00	
Ventrucci et al. [3]	1986	189 (12)	Phadebas	220	220	100.0	84.4	46.2	100.0	6.41	0.00	
Elastase-1												
Wilson et al. [10]	2005	188 (29)	ELISA	3.5	3.5	80.0	96.0	80.0	96.0	20.00	0.21	

PLR = Sensitivity/(100 − Specificity)

NLR = (100 − Sens)/Spec

AP Acute pancreatitis, *PPV* positive predictive value, *NPV* negative predictive value, *PLR* positive likelihood ratio, *NLR* negative likelihood ratio, *AUC* area under the curve, *ND* not determined

upper abdomen (Table 3) (Level 4) [17]. There are cases in which acute pancreatitis is not accompanied by abdominal pain, although this occurs very rarely (Level 2b) [18]. Of all the patients with abdominal pain, the rate of acute pancreatitis is reported to be 0.9% ($n = 1000$) (Level 2b) [19] and that the rate of acute pancreatitis is 1.6% ($n = 6317$) when there is abdominal pain of acute onset in patients under 50 years of age and 7.3% ($n = 2406$) in patients over 50 years of age (Level 4) [20]. On the other hand, it is reported that the rate is 2–3% in the case of acute abdomen (Level 2b, 5) [21, 22]. Except for abdominal pain, symptoms and signs observed frequently include pain radiating to the back, anorexia, fever, nausea and vomiting, and decreased bowel sound (Table VI-1, 2) (Level 3b–5) [14–16].

Hematological examination and urinalysis

CQ3 Which pancreatic enzyme measurements are important in making a diagnosis of acute pancreatitis?

Measurement of blood lipase is most useful for the diagnosis of acute pancreatitis (Recommendation A)

Table 2 Clinical symptoms and findings of acute pancreatitis

Symptoms[a]	Frequency of occurrence frequency (%)	Symptoms[b]	Frequency (%)
Abdominal pain	90	Abdominal pain	95
Muscular defense	80	Radiating pain to the back	
Nausea, vomiting	70	Anorexia	85
Meteorism	60	Nausea, vomiting	75
Ileus	55	Decreased sound of intestinal peristalsis	60
Jaundice	30	Fever	60
Shock	20	Muscular defense	50
Neurological findings	10	Shock	15
		Jaundice	15
		Hematemesis	10

[a] From Ref. [14]

[b] From Ref. [15] with partial alterations

When the measurement of blood lipase is difficult, blood amylase (pancreatic amylase) should be measured (Recommendation A)

Detection of elevated levels of blood pancreatic enzymes is crucial in the diagnosis of acute pancreatitis. The diagnostic ability of measurements of various types of pancreatic enzymes is listed in Table 1.

Among several pancreatic enzymes, blood amylase is used most widely because it can be measured rapidly. However, blood lipase is reported to be superior to blood amylase in terms of sensitivity and specificity [23–27] (Level 3b, 5). By comparison of values of various pancreatic enzymes for diagnosing acute pancreatitis (Level 2a) [28, 29], blood lipase has similar sensitivity to blood amylase but with superior specificity (Tables 4, 5), so measurement of blood lipase is recommended rather than blood amylase for the diagnosis of acute pancreatitis. Addition of measurements of blood amylase to blood lipase resulted in no improvement in the diagnostic ability of acute pancreatitis [30].

Blood lipase

The sensitivity and specificity of blood lipase in a diagnosis of acute pancreatitis are reported to be 85–100 and 84.7–99.0%, respectively (Level 2a) [28] and blood lipase is shown to be more sensitive than blood amylase (Level 2b–3b) [4, 31, 32] (Table 1). Abnormal values of blood lipase last longer than those of blood amylase (Level 2b) [33], so blood lipase is useful in a diagnosis of acute pancreatitis when blood amylase level is normal. Also, blood lipase has almost equal diagnostic value as that of blood *p*-amylase (Level 2b) [32]. Blood lipase is also reported to be useful because of its high sensitivity in a diagnosis of alcohol-induced acute pancreatitis (Level 2b) [34].

Blood amylase (total blood amylase)

The sensitivity and specificity of blood amylase in a diagnosis of acute pancreatitis are not constant because of the difference in the diagnostic grounds of acute pancreatitis and the cutoff level that has been set. When the cutoff blood amylase level is set at the upper limit of normal, its sensitivity and specificity are 91.7–100 and 71.6–97.6%,

Table 3 Abdominal pain, site of tenderness (%)

	RUQ	LUQ	RLQ	LLQ	Upper half	Lower half	Right half	Left half	Central	General
Site of abdominal pain	6		2	2	38	6		2	14	29
Site of tenderness	14	6			16		8	4		48

From Ref. [18]

Table 4 Diagnostic ability of measurements of blood amylase, *p*-amylase and lipase in acute pancreatitis

Lipase	Total amylase	Pancreatic amylase
Sensitivity		
Very good	Very good	Good
90–100%	95–100%	84–100%
Specificity		
Very good	Low	Good
99%	70%	40–97%
At upper limit of normal	Influenced by "cut-off level"	Influenced by "cut-off level"
Positive predictive value (PPV)		
Very good	Very low	50–96%
90%	15–72%	
Negative predictive value (NPV)		
95–100%	97–100%	70–100%
Reliability		
Good	Good	Poor

From Ref. [28] with partial alterations

Table 5 Sensitivity and specificity of main blood pancreatic enzymes

	Sensitivity (%)	Specificity (%)
Lipase	82–100	82–100
Total amylase	67–100	85–98
Pancreatic amylase	67–100	83–98
Trypsin	89–100	79–83
Elastase 1	97–100	79–96

From Ref. [29] with partial alterations

respectively. On the other hand, when the cutoff level is set higher, specificity improves but sensitivity decreases. It is shown that at the cutoff level of 1000 IU/L, specificity rises up to 100% while sensitivity goes down to 60.9% (Level 2a–3b) [2, 3, 8–11, 13, 25, 28, 35] (Table 1).

There are the following factors contributing to decreased blood amylase sensitivity. Blood amylase levels do not increase in many cases of alcohol-induced acute pancreatitis, especially when chronic pancreatitis is present in the background (Level 2b) [31, 36]. Compared with other pancreatic enzymes, blood amylase levels decrease soon after the onset of the disease and an abnormal high level lasts for only a short time. Therefore, if the passage of time from the onset of the disease to the hospital visit is long, the level may return to normal (Level 3b) [37, 38]. There is also a report showing that blood amylase levels seldom rise in the case of acute pancreatitis caused by hyperlipidemia (Level 3b) [39].

Table 6 Condition causing hyperamylasemia

Pancreatic diseases	Neoplastic lesion other than pancreatitis
Pancreatitis	Ovary prostate lung esophagus solid tumor of the thymus
Complications in pancreatitis (pancreatic pseudocysts, pancreatic abscess)	Multiple osteoma
	Pheochromocytoma
Trauma (surgery and ERCP included)	Others
Pancreatic obstruction	Renal failure
Pancreatic tumor	Renal transplantation
Cystic fibrosis	Macroamylasemia
Salivary disease	Burns
Infection (mumps)	Acidosis (ketotic non-ketotic)
Trauma (surgery included)	Pregnancy
X-rays irradiation	Head injury
Ductal stenosis	Drug-induced (morphine diuretic steroid)
Gastrointestinal disease	Acute aortic dissection
Penetration or perforation in gastrointestinal ulcer	Postoperative (except for trauma)
Intestinal penetration or perforation	Anorexia nervosa
Obstruction of mesenteric artery	Atopic
Appendicitis	
Liver disease(hepatitis cirrhosis)	
Gynecological disorder	
Rupture of ectopic pregnancy	
Ovarian cystoma	
Pelvic infection	

From Ref. [40]

A hole regarding blood amylase levels in a diagnosis of acute pancreatitis is that an abnormal high level is often detected in diseases other than pancreatic diseases (Table 6) and that blood amylase has poor specificity for diagnosis (Level 2a) [30].

p-Amylase (amylase isozyme)

There is a study reporting that, by measuring the blood *p*-amylase level, a differential diagnosis of hyperamylasemia not associated with pancreatic diseases was made in 83% (19/23 cases) of patients with hyperamylasemia (Level 4) [40]. On the other hand, there are reports showing that the differential ability of measurements of blood *p*-amylase was 20–44% (Level 3b) [11, 13]. According to another report, no improvement was observed in sensitivity and specificity compared with blood amylase and other blood pancreatic enzymes (Level 2b) [2] (Table 1). The usefulness of blood *p*-amylase in making a diagnosis of acute pancreatitis is therefore not certain.

Urine amylase

Urine amylase has shown high sensitivity in a diagnosis of acute pancreatitis in the past (Level 2b) [41]. However, comparison with blood amylase and other blood pancreatic enzymes found that, at present, measurements of urine amylase have no superiority in diagnostic ability to measurements of other blood pancreatic enzymes (Level 2b–3b) [42, 43].

Blood esterase 1

Esterase 1 is characterized by keeping an abnormal high level longer than any other pancreatic enzymes (Level 2b–3b) [44, 45], so its measurement is considered useful when medical examination is conducted after a long time has passed since the onset of the disease. A study reports that blood esterase 1 has no additional value in the diagnosis of acute pancreatitis and severity assessment (Level 2b) [46]. However, it is also reported that esterase 1 is as suitable as amylase and lipase in terms of clinical usefulness including sensitivity and specificity because rapid and simple measurement has recently become possible (Table 1) [10].

Other blood pancreatic enzymes

Trypsin is a key enzyme involved in the onset of acute pancreatitis and is inactivated rapidly by protease inhibitors in the blood, so determination of its enzymatic activity is difficult. However, it is determined as an antigen quantity by an immunological method. Measurement of the blood trypsin level in acute pancreatitis shows that it has high sensitivity for acute pancreatitis (Level 2b–3b) [39, 47]. Furthermore, there are reports that blood phospholipase A2 (PLA2) increases remarkably in acute pancreatitis and its level is correlated with the severity of the disease (Level 3b) [48, 49]. However, determination of both enzymes depends upon the immunological method, which makes rapid determination difficult. Therefore, their measurement is not suitable for making a diagnosis of acute pancreatitis in clinical settings.

Other urine pancreatic enzymes

Trypsinogen-2, one of the precursors of trypsin, belongs to the group of pancreatic enzymes and is excreted into the urine in the early phase of acute pancreatitis. Recently, there are several studies that reported a method that uses a stick resembling test paper to examine the presence or absence of an elevated level of urine trypsinogen-2 within 5 min or so (Level 2b) [5, 7–9, 35, 50, 51]. Clinical value of this method including its sensitivity and specificity is as high as that of amylase and lipase (Table 7). Particularly, recent studies report that sensitivity and negative predictive value (NPV) are both 100% [5, 12, 50]. This is a rapid and simple method, so its measurement is also a promising procedure for general clinicians.

Diagnostic imaging

Plain chest-abdominal roentgenography

CQ4 Is plain chest-abdominal roentgenography necessary for the diagnosis of acute pancreatitis?

Plain chest-abdominal roentgenography is necessary when acute pancreatitis is suspected (Recommendation A)

Because both plain chest and abdominal roentgenographic findings associated with acute pancreatitis are not specific, it is impossible to make a diagnosis of acute pancreatitis using this method (Level 4) [52]. However, plain chest and abdominal roentgenography is a crucial test in patients with acute pancreatitis for the differential diagnosis from other diseases such as alimentary tract perforation as well as for assessing the clinical course.

Findings in acute pancreatitis detected by plain X-ray examinations include images of ileus, colon cut-off signs, images of localized sentinel loop signs in the left upper abdomen, images of dilated duodenal loops and gas collection, and images of retroperitoneal gas collection. Colon cut-off signs are reported to be a result that the narrowing of the inner spaces of colon by the spread of inflammation arises from the extension of fluid collection and fat necrosis as far as the transverse mesocolon, phrenicocolic ligaments and the left or right anterior paranephric cavities, causes the dilatation of the mouth side of the colon [53–55] (Level 4). Most of the colon cut-off signs are observed in the splenic flexura or descending colon, followed by the transverse colon. Findings detected by plain chest and abdominal X-ray examinations include images suggesting the presence of such conditions as collection of pleural effusion, acute respiratory distress syndrome (ARDS) and pneumonia.

Ultrasonography

CQ5 Is ultrasonography necessary for the diagnosis of acute pancreatitis?

When acute pancreatitis is suspected, ultrasonography is necessary (Recommendation A)

Ultrasonography is one of the tests to be performed at first in every patient in whom acute pancreatitis is suspected.

Ultrasonography, which enables visualization of findings associated with acute pancreatitis such as pancreatic enlargement, inflammatory changes around the pancreas and ascites, is useful in making a diagnosis of acute pancreatitis. It is reported that the visualization rate of the pancreas by US is 62–90% and that of inflammatory changes around the pancreas are 62–90% for the anterior paraphrenic cavity, 90% for the lesser momentum, and 65% for the mesentery, respectively (Level 1b–2b) [56, 57]. Visualization of the pancreas and parapancreatic tissues may be poor in severe cases under the influence of images of intra-intestinal retention of gas bubbles (Level 1b–2b) [56, 57]. US is also effective in detecting biliary lithiasis responsible for acute pancreatitis and differentiating acute pancreatitis from other abdominal diseases.

CT

CQ6 Is CT useful in making a diagnosis of acute pancreatitis?

CT is useful when acute pancreatitis is suspected (Recommendation A)

CT should be performed aggressively when a definitive diagnosis of acute pancreatitis on the basis of clinical manifestations, hematological examination, urinalysis and US is impossible. CT enables visualization of objective local images of the pancreas free from the influence of gas bubbles in the alimentary tract and fatty tissues in the abdominal wall and cavity (Level 1b) [56, 58, 59], so it is the most useful imaging examination for making a diagnosis of acute pancreatitis. It is also useful in a differential diagnosis from other intra-abdominal diseases such as perforation associated with gastroduodenal ulcer.

CT findings useful in a diagnosis of acute pancreatitis include the enlargement of the pancreas, increased concentrations of adipose tissue in the parapancreatic and retroperitoneal cavities (mainly in an anterior pararenal space) and mesocolon and mesenteriolum, fluid collection, pseudocyst formation, uneven density of the pancreatic parenchyma, pancreatic necrosis, fatty necrosis in the retroperitoneal space and mesentery, hematoma, images of pancreatic fissure associated with trauma [60] (Figs. 1, 2, 3). Gas images in and around the pancreas are often caused by fistula formation between the intestinal tract and infections with gas-forming bacteria (Level 1c) (Fig. 4) [61].

CT also helps in assessing the severity of acute pancreatitis because a diagnosis that is important in deciding treatment policy has been made possible concerning complications accompanying pancreatitis and comorbidities in the intra-abdominal organs.

Table 7 Diagnostic ability of urine trypsinogen-2 measurement in acute pancreatitis

Author	Year	*n* (AP)	Methodology	Upper limit of normal	Unit	Cut-off value	Sensitivity	Specificity	PPV	NPV	PLR	NLR	AUC
Trypsinogen-2 (urinary)													
Jang et al. [5]	2007	191 (17)	Urine dipstick (immunochromatography)	50.0	μg/l		100.0	96.0			2.43	0.00	
Sankaralingam et al. [50]	2007	30 (5)	Urine dipstick (immunochromatography)	50.0	μg/l	(After 1 h)	100.0	91.0	66.0	100.0			
						(After 4 h)	100.0	96.0	80.0	100.0			
Raty et al. [12]	2007	50 (13)	Urine dipstick (immunochromatography)	50.0	μg/l		100.0	92.0	81.0	100.0			0.959
Sáez et al. [7]	2005	72 (50)	Urine dipstick (immunochromatography)	50.0	μg/l		68.0	86.4	91.9	54.3	5.00	0.37	
Chen et al. [8]	2005	165 (98)	Urine dipstick (immunochromatography)	50.0	μg/l		89.6	85.7	81.1	92.3	6.27	0.12	
Kylänpää-Bäck et al. [9]	2002	237 (29)	Urine dipstick (immunochromatography)	50.0	μg/l		93.0	92.0	63.0	99.0	11.63	0.08	
Kylänpää-Bäck et al. [35]	2000	525 (45)	Urine dipstick (immunochromatography)	50.0	μg/l		96.0	92.0	54.0	99.6	12.00	0.04	

AP Acute pancreatitis, *PPV* positive predictive value, *NPV* negative predictive value, *PLR* positive likelihood ratio, *NLR* negative likelihood ratio, *AUC* area under the curve

MRI

CQ7 In which cases is MRI useful in making a diagnosis of acute pancreatitis?

MRI is useful in making a diagnosis of biliary stones causing pancreatitis and hemorrhagic pancreatic necrosis (Recommendation B)

A diagnosis of edematous pancreatitis by CT is difficult when it is not accompanied by enlargement of the pancreas but T2-enhanced MRI imaging enables visualization of the pancreas clearly in accordance with the severity of edema. Also, MRI has diagnostic ability similar to that of CT in making a diagnosis of parapancreatic fluid collection and hypertrophy of the anterior renal fascia [62, 63] (Fig. 5). Although differentiation by CT of parapancreatic fatty necrosis from fluid collection may be difficult in some cases, MRI enables clear differentiation of fatty necrosis from fluid according to signal strength (compared with fluid, fatty necrosis presents higher signals in T1-enhanced imaging and mildly low signals in T2-enhanced imaging) [62, 64, 65]. Hemorrhagic fatty necrosis that presents a high signal particularly in fat-saturation T1-enhanced imaging can be diagnosed relatively easily (Fig. 6). Gd-DTPA dynamic MRI imaging is able to depict foci of pancreatic necrosis as a hyperchromatic area [66, 67].

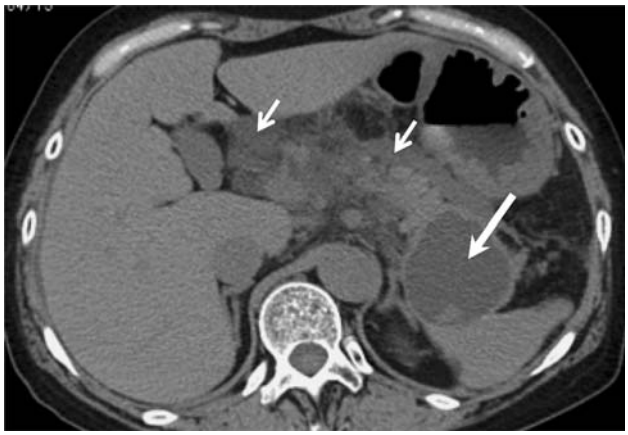

Fig. 1 Plain CT shows fluid in the parapancreatic cavities (*small arrows*) and pseudocyst formation in the pancreatic tail (*large arrow*)

Endoscopic retrograde cholangiopancreatography (ERCP)

CQ8 Is ERCP necessary for the diagnosis of acute pancreatitis?

ERCP is not used for the purpose of making a diagnosis of acute pancreatitis itself (Recommendation D) Note: As far as a disease such as gallstone-induced pancreatitis is

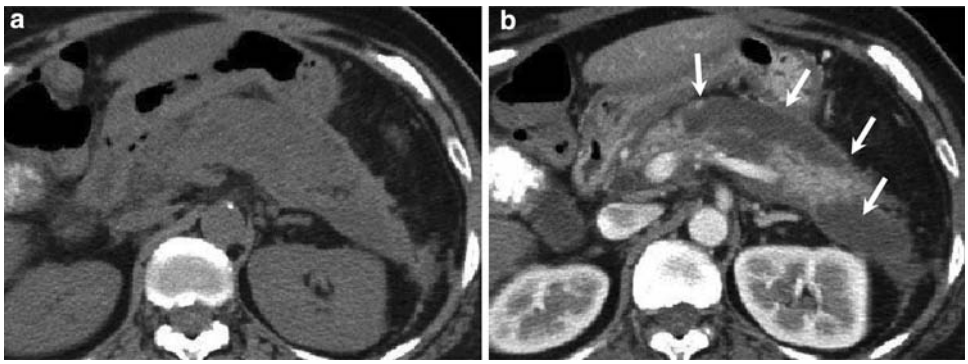

Fig. 2 Plain CT shows enlargement of the pancreatic body (**a**). Contrast-enhanced CT shows pancreatic necrosis as unenhanced area (*arrows*) (**b**)

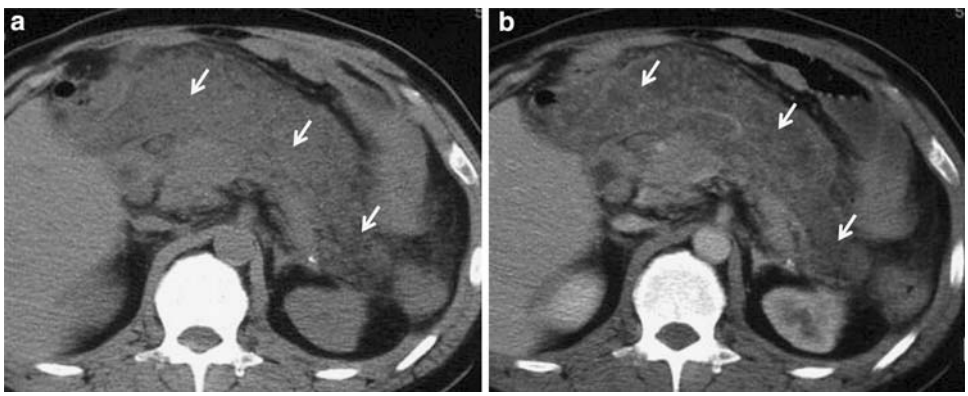

Fig. 3 Plain CT (**a**) and contrast-enhanced CT (**b**) show fatty necrosis (*arrows*) in mesentery

concerned, ERCP is often performed on the assumption that endoscopic treatment is to be delivered.

Adverse events associated with endoscopic retrograde cholangiopancreatography (ERCP) are reported, so ERCP is not used for the diagnosis of acute pancreatitis itself (Level 2b) [68, 69].

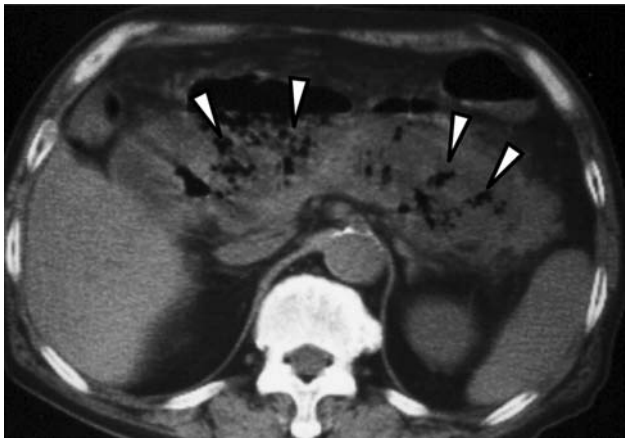

Fig. 4 Plain CT shows gas image caused by infection with gas-forming bacteria, in and around the pancreas

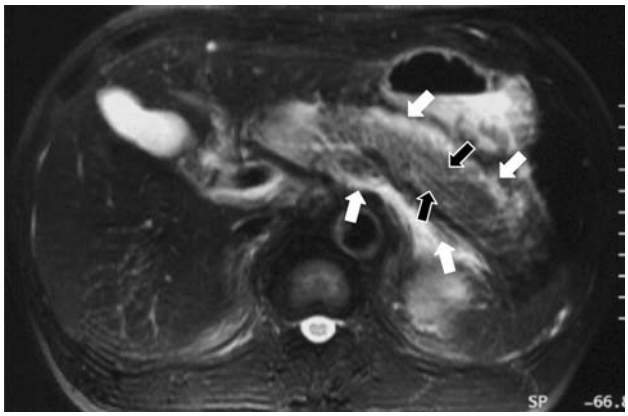

Fig. 5 T2-enhanced MRI imaging shows a mildly high signal of the pancreatic parenchyma (*black arrows*) and a high signal of parapancreatic fluid collection (*white arrows*), in edematous pancreatitis with mild enlargement of the pancreas

Etiologic diagnosis

Necessity and significance of etiologic diagnosis

CQ9 What is the purpose of etiologic diagnosis?

The purpose of etiologic diagnosis is deciding treatment policy in acute pancreatitis by elucidating causes of the disease condition. Treatments for these causes are also important in achieving resolution of acute pancreatitis and preventing recurrence of pancreatitis (Recommendation A)

As soon as a diagnosis of acute pancreatitis has been made, etiologic diagnosis should be made. Especially, the diagnosis of gallstone-induced pancreatitis should be given top priority as it is related closely to treatment policy including the assessment of whether endoscopic papillary treatment should be conducted or not. Etiologic diagnosis should be made immediately because treatment differs depending upon the causes of diseases including gallstone, hyperlipemia, trauma, incomplete fusion of the pancreatic duct, autoimmunity, hyperfunction of the parathyroid gland, and tumors of the pancreaticobiliary system. Pancreatic cancer and intrapancreatic papillary mucous tumor is likely to be associated with acute pancreatitis, so imaging examinations should be conducted.

CQ10 Which tests are necessary for the diagnosis of gallstone-induced acute pancreatitis?

Hematological examinations and ultrasonography should be performed in the first place (Recommendation A)

Presence of jaundice, elevated levels of ALP, γGTP and transamylase detected by blood tests and the presence of common bile duct stones and gallbladder stones visualized by extracorporeal US (EUS henceforth) lead to the suspicion of gallstone-induced acute pancreatitis. However, many of the stones in the common bile duct are small-sized 'passed stones' that induce acute pancreatitis and that have already been excreted from the papilla to the duodenum, so visualization of these stones by US may be difficult in

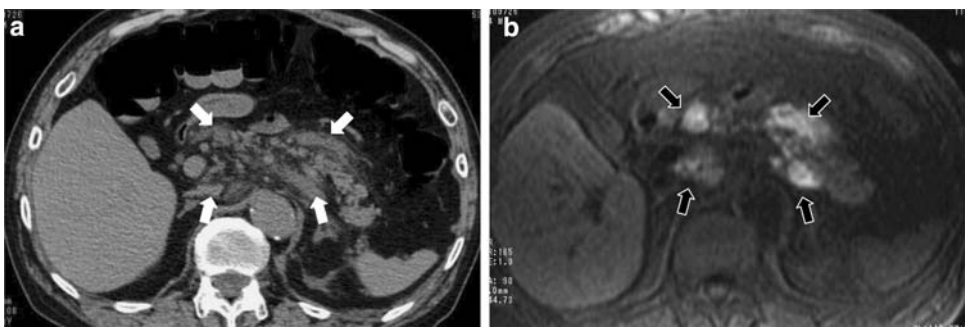

Fig. 6 Plain CT (**a**) and T1-enhanced MRI imaging (**b**). Parapancreatic fatty necrosis can be differentiated from fluid collection by fat-saturation T1-enhanced MRI imaging. Hemorrhagic fatty necrosis showed as fluid collection by plain CT (**a** *white arrows*) presents a high signal in fat-saturation T1-enhanced imaging (**b** *black arrows*)

some cases. This often makes difficult the diagnosis of gallstone-induced acute pancreatitis.

Combination of US and blood tests yields a sensitivity of 95–98%, specificity of 100%, positive likelihood ratio of ∞ and negative likelihood ratio of 20.0–50.0, which enables the etiologic diagnosis of gallstone-induced acute pancreatitis (Level 2b) [70–72]. Not all cases involved are necessarily visualized by US, so US should be conducted repeatedly or MRCP, EUS, or ERCP (on the assumption that endoscopic papillary treatment is to be provided) should be conducted.

Personal and family history taking

Checking is necessary for past history of alcohol consumption, gallstone and hyperlipemia, and the presence or absence of tests and procedures involved in the onset of pancreatitis including ERCP, endoscopic papillary treatment, surgery and use of drugs.

Blood tests

Levels of bilirubin, transamylase (ALT, AST) and ALP should be measured in all cases to differentiate gallstone-induced acute pancreatitis from other acute pancreatitis [70]. There is a high possibility that gallstone-induced acute pancreatitis is present when blood ALT is over 150 IU/L (48–93% for sensitivity,34–96% for specificity, 1.4–12.0 for positive likelihood ratio, and 1.8–4.9 for negative likelihood ratio) (Level 1c–2b) [73, 74], or when abnormal values were detected by blood tests in more than three of the items including bilirubin, ALP, γGTP, ALT, ALT/AST (85% for sensitivity, 69% for specificity, 2.7 for positive likelihood ratio, and 4.6 for negative likelihood ratio) [71].

When the level of blood neutral fat exceeds 1000 mg/dL, there is a possibility that hyperlipemia is the cause of acute pancreatitis and when pancreatitis is accompanied by hypercalcemia, hyperfunction of the parathyroid gland is likely to be a cause [70].

Ultrasonography

Ultrasonography is useful in visualizing abnormal findings associated with the etiology of acute pancreatitis such as biliary stones and common bile duct dilatation. However, the ability of US to visualize the common bile duct decreases in acute pancreatitis due to intestinal gas imaging. The rate of US to visualize common bile duct stones differs from report to report (20–90%), so gallstone-induced pancreatitis should not be ruled out even if US has failed to detect biliary stones and bile duct dilatation (Level 1b–4) [75–77].

CT

CT is useful in the diagnosis of a pancreatic cancer and intrapancreatic papillary mucous tumor as a possible cause of acute pancreatitis along with acute worsening of chronic pancreatitis and traumatic pancreatitis. Because CT is not able to visualize biliary stones in many cases (40–53% for sensitivity), it is not suitable for diagnosing gallstone-induced acute pancreatitis (Level 1b) [71, 77].

MRI/MRCP

Compared with ERCP, MRCP enables visualization of, less invasively and without manipulation of the papilla and use of contrast media, the pancreatic duct and bile duct in a relatively early phase of the disease without carrying the risk of worsening the condition of acute pancreatitis. MRCP should be conducted aggressively when the presence of biliary stones is not certain according to US and CT (Level 3) [78–80]. The sensitivity to visualize common bile duct stones is 20% for CT, and 40% for MRCP, respectively, but it is 80% for MRI/MRCP. There is an opinion that recommends MRI/MRCP as a procedure for determining the indications for endoscopic papillary treatment (ERCP/ES) [69]. MRCP treated with MIP alone is likely to fail to detect small biliary stones, so the presence or absence of bile stones should be judged by all means, using as references original MRCP images and thin-sliced T2-enhanced images visualized from multiple directions. MRI/MRCP that visualizes an anomalous arrangement of the pancreaticobiliary tract and incomplete fusion of the pancreatic duct besides bile stones is useful in the etiologic diagnosis of acute pancreatitis (Level 4) [73, 81, 82].

EUS

EUS is superior to US in terms of the ability to visualize common bile duct stones (Level 1b–2b) [75, 83, 84]. EUS is indicated when extracorporeal US is not able to identify common bile duct stones after an attack has subsided. In cases where US has failed to elucidate the etiology, visualization of common bile duct stones is made possible by EUS in 59–78% of those cases (Level 1b–3b) [83, 85, 86]. There is a report showing that by performing EUS in cases where causes were not known by blood tests, US or CT, common bile duct stones were identified in 77.8% of those cases (Level 2b) [84]. Besides biliary stones, EUS is able to make a diagnosis of chronic pancreatitis, pancreatic cancer, intrapancreatic papillary mucous tumor, an anomalous arrangement of the pancreaticobiliary duct and incomplete fusion of the pancreatic duct. Therefore, this procedure is useful in making an etiologic diagnosis of acute pancreatitis (Level 1b–3b) [85, 86].

ERCP

ERCP performed at the time of an attack of acute pancreatitis carries a risk of worsening pancreatitis further. However, in gallstone-induced pancreatitis, when the presence of common bile duct stones is suspected along with jaundice and hepatic disorders, ERCP should be performed on the assumption of endoscopic treatment for biliary stones. When ERCP/ES is not available, patients should be transferred to a medical facility that is in a position to perform ERCP/ES. When gallstone-induced acute pancreatitis is suspected, elective ERCP should be conducted after recovery from pancreatitis because there is a possibility that common bile duct stones not visualized by other procedures are present (Level 3b) [87]. Besides bile stones, elective ERCP is also able to make an etiologic diagnosis of anatomic anomalies (an anomalous arrangement of the pancreaticobiliary duct, incomplete fusion of the pancreatic duct, obstruction of the accessory pancreatic duct, and long common channel [88]) and tumors.

References

1. Takeda K, Ohtuki K, Kitagawa M, et al. Final draft of the diagnostic criteria for and the criteria for severity assessment of acute pancreatitis; 2005 Report. Tokyo: The Intractable Pancreatic Disease Investigation and Research Group of the Japanese Ministry of Health, Labour and Welfare; 2006. p. 27–34.
2. Steinberg WM, Goldstein SS, Davis ND, Shamma'a J, Anderson K. Diagnostic assays in acute pancreatitis. A study of sensitivity and specificity. Ann Intern Med. 1985;102:576–80.
3. Ventrucci M, Pezzilli R, Naldoni P, Montone L, Gullo L. A rapid assay for serum immunoreactive lipase as a screening test for acute pancreatitis. Pancreas. 1986;1:320–3.
4. Thomson HJ, Obekpa PO, Smith AN, Brydon WG. Diagnosis of acute pancreatitis: a proposed sequence of biochemical investigations. Scand J Gastroenterol. 1987;22:719–24.
5. Jang T, Uzbielo A, Sineff S, Naunheim R, Scott MG, Lewis LM. Point-of-care urine trypsinogen testing for the diagnosis of pancreatitis. Acad Emerg Med. 2007;14:29–34.
6. Petrov MS, Gordetzov AS, Emelyanov NV. Usefulness of infrared spectroscopy in diagnosis of acute pancreatitis. ANZ J Surg. 2007;77(5):347–51.
7. Sáez J, Martínez J, Trigo C, Sánchez-Payá J, Compañy L, Laveda R, et al. Clinical value of rapid urine trypsinogen-2 test strip, urinary trypsinogen activation peptide, and serum and urinary activation peptide of carboxypeptidase B in acute pancreatitis. World J Gastroenterol. 2005;11:7261–5.
8. Chen YT, Chen CC, Wang SS, Chang FY, Lee SD. Rapid urinary trypsinogen-2 test strip in the diagnosis of acute pancreatitis. Pancreas. 2005;30:243–7.
9. Kylänpää-Bäck ML, Kemppainen E, Puolakkainen P, Hedström J, Haapiainen R, Korvuo A, et al. Comparison of urine trypsinogen-2 test strip with serum lipase in the diagnosis of acute pancreatitis. Hepatogastroenterology. 2002;49:1130–4.
10. Wilson RB, Warusavitarne J, Crameri DM, Alvaro F, Davies DJ, Merrett N. Serum elastase in the diagnosis of acute pancreatitis: a prospective study. ANZ J Surg. 2005;75:152–6.
11. Pace BW, Bank S, Wise L, Burson LC, Borrero E. Amylase isoenzymes in the acute abdomen: an adjunct in those patients with elevated total amylase. Am J Gastroenterol. 1985;80:898–901.
12. Raty S, Sand J, Nordback I. Detection of postoperative pancreatitis after pancreatic surgery by urine trypsinogen strip test. Br J Surg. 2007;94:64–9.
13. Koehler DF, Eckfeldt JH, Levitt MD. Diagnostic value of routine isoamylase assay of hyperamylasemic serum. Gastroenterology. 1982;82:887–90.
14. Malfertheiner P, Kemmer TP. Clinical picture and diagnosis of acute pancreatitis. Hepatogastroenterology. 1991;38:97–100.
15. Corsetti JP, Arvan DA. Acute pancreatitis. Diagnostic strategies for common medical problems. 2nd ed. Philadelphia: American College of Physician; 1999. p. 205.
16. Ohtsuki M, Kihara Y, Kikuchi K, et al. National epidemiological research of acute pancreatitis; 2004 Report. Tokyo: The Intractable Pancreatic Disease Investigation and Research Group of the Japanese Ministry of Health, Labour and Welfare; 2005. p. 56–63.
17. Staniland JR, Ditchburn J, De Dombal FT. Clinical presentation of acute abdomen: study of 600 patients. Br Med J. 1972;3(823):393–8.
18. Read G, Braganza JM, Howat HT. Pancreatitis: a retrospective study. Gut. 1976;17:945–52.
19. Brewer BJ, Golden GT, Hitch DC, Rudolf LE, Wangensteen SL. Abdominal pain. An analysis of 1,000 consecutive cases in a University Hospital emergency room. Am J Surg. 1976;131(2):219–23.
20. Telfer S, Fenyo G, Holt PR, de Dombal FT. Acute abdominal pain in patients over 50 years of age. Scand J Gastroenterol Suppl. 1988;144:47–50.
21. de Dombal F. Acute abdominal pain: an O.M.G.E. survey. Scand J Gastroenterol. 1979;56(suppl):29–43.
22. de Dombal FT. Diagnosis of acute abdominal pain. 2nd ed. London: Churchill Livingstone; 1991. p. 19–30.
23. UK Working Party on Acute Pancreatitis. UK guidelines for the management of acute pancreatitis. Gut. 2005;54:1–9.
24. Koizumi M, Takada T, Kawarada Y, Hirata K, Mayumi T, Yoshida M, et al. JPN Guidelines for the management of acute pancreatitis: diagnostic criteria for acute pancreatitis. J Hepatobiliary Pancreat Surg. 2006;13:25–32.
25. Sargent S. Pathophysiology, diagnosis and management of acute pancreatitis. Br J Nurs. 2006;15:999–1005.
26. Banks PA, Freeman ML, the Practice Parameters Committee of the American College of Gastroenterology. Practice guidelines in acute pancreatitis. Am J Gastroenterol. 2006;101:2379–400.
27. Smith RC, Southwell-Keely J, Chesher D. Should serum pancreatic lipase replace serum amylase as a biomarker of acute pancreatitis? ANZ J Surg. 2005;75:399–404.
28. Agarwal N, Pitchumoni CS, Sivaprasad AV. Evaluating tests for acute pancreatitis. Am J Gastroenterol. 1990;85:356–66.
29. Dervenis C, Johnson CD, Bassi C, Bradley E, Imrie CW, McMahon MJ, et al. Diagnosis, objective assessment of severity, and management of acute pancreatitis. Santorini consensus conference. Int J Pancreatol. 1999;25:195–210.
30. Vissers RJ, Abu-Laban RB, McHugh DF. Amylase and lipase in the emergency department evaluation of acute pancreatitis. J Emerg Med. 1999;17:1027–37.
31. Nordestgaard AG, Wilson SE, Williams RA. Correlation of serum amylase levels with pancreatic pathology and pancreatitis etiology. Pancreas. 1988;3:159–61.
32. Apple F, Benson P, Preese L, Eastep S, Bilodeau L, Heiler G. Lipase and pancreatic amylase activities in tissues and in patients with hyperamylasemia. Am J Clin Pathol. 1991;96:610–4.
33. Orebaugh SL. Normal amylase levels in the presentation of acute pancreatitis. Am J Emerg Med. 1994;12:21–4.

34. Gumaste V, Dave P, Sereny G. Serum lipase: a better test to diagnose acute alcoholic pancreatitis. Am J Med. 1992;92:239–42.
35. Kylänpää-Bäck ML, Kemppainen E, Puolakkainen P, Hedström J, Haapiainen R, Perhoniemi V, et al. Reliable screening for acute pancreatitis with rapid urine trypsinogen-2 test strip. Br J Surg. 2000;87:49–52.
36. Clavien PA, Robert J, Meyer P, Borst F, Hauser H, Herrmann F, et al. Acute pancreatitis and normoamylasemia. Not an uncommon combination. Ann Surg. 1989;210:614–20.
37. Eckfeldt JH, Kolars JC, Elson MK, Shafer RB, Levitt MD. Serum tests for pancreatitis in patients with abdominal pain. Arch Pathol Lab Med. 1985;109:316–9.
38. Ventrucci M, Pezzilli R, Naldoni P, Plate L, Baldoni F, Gullo L, et al. Serum pancreatic enzyme behavior during the course of acute pancreatitis. Pancreas. 1987;2:506–9.
39. Toskes PP. Hyperlipidemic pancreatitis. Gastroenterol Clin N Am. 1990;19:783–91.
40. Swensson EE, King ME, Malekpour A, Maull KI. Serum amylase isoenzyme alterations in acute abdominal conditions. Ann Emerg Med. 1985;14:421–3.
41. Holdsworth PJ, Mayer AD, Wilson DH, Flowers MW, McMahon MJ. A simple screening test for acute pancreatitis. Br J Surg. 1984;71:958–9.
42. Gunn IR, Faye S, Clayton MG. Prospective evaluation of urinary amylase test strip. Lancet. 1986;1:1161.
43. Werner M, Steinberg WM, Pauley C. Strategic use of individual and combined enzyme indicators for acute pancreatitis analyzed by receiver-operator characteristics. Clin Chem. 1989;35:967–71.
44. Buchler M, Malfertheiner P, Uhl W, Beger HG. Diagnostic and prognostic value of serum elastase 1 in acute pancreatitis. Klin Wochenschr. 1986;64:1186–91.
45. Flamion B, Delhaye M, Horanyi Z, Delange A, Demanet H, Quenon M, et al. Comparison of elastase-1 with amylase, lipase, and trypsin-like immunoreactivity in the diagnosis of acute pancreatitis. Am J Gastroenterol. 1987;82:532–5.
46. Millson CE, Charles K, Poon P, Macfie J, Mitchell CJ. A prospective study of serum pancreatic elastase-1 in the diagnosis and assessment of acute pancreatitis. Scand J Gastroenterol. 1998;33:664–8.
47. Ventrucci M, Pezzilli R, Gullo L, Plate L, Sprovieri G, Barbara L. Role of serum pancreatic enzyme assays in diagnosis of pancreatic disease. Dig Dis Sci. 1989;34:39–45.
48. Schroder T, Lempinen M, Kivilaakso E, Nikki P. Serum phospholipase A2 and pulmonary changes in acute fulminant pancreatitis. Resuscitation. 1982;10:79–87.
49. Nishijima J, Okamoto M, Ogawa M, Kosaki G, Yamano T. Purification and characterization of human pancreatic phospholipase A2 and development of a radioimmunoassay. J Biochem. 1983;94:137–47.
50. Sankaralingam S, Wesen C, Barawi M, Galera R, Lloyd L. Use of the urinary trypsinogen-2 dipstick test in early diagnosis of pancreatitis after endoscopic retrograde cholangiopancreatography. Surg Endosc. 2007;21:1312–5.
51. Warshaw AL, Fuller AF Jr. Specificity of increased renal clearance of amylase in diagnosis of acute pancreatitis. N Engl J Med. 1975;292:325–8.
52. Stein GN, Kalser MH, Sarian NN, Finkelstein A. An evaluation of the roentgen changes in acute pancreatitis: correlation with clinical findings. Gastroenterology. 1959;36:354–61.
53. Brascho DJ, Reynolds TN, Tanca P. The radiographic "colon cutoff sign" in acute pancreatitis. Radiology. 1962;79:763–8.
54. Meyers MA, Evans JA. Effect of pancreatitis on the small bowel and colon: spread along mesenteric planes. AJR. 1973;199:151–65.
55. Pickhardt PJ. The colon cutoff sign. Radiology. 2000;215:387–9.
56. Silverstein W, Isikoff MB, Hill MC, Barkin J. Diagnostic imaging of acute pancreatitis: prospective study using CT and sonography. AJR. 1981;137:497–502.
57. Jeffrey RB Jr, Laing FC, Wing VW. Extrapancreatic spread of acute pancreatitis: new observations with real-time US. Radiology. 1986;159:707–11.
58. Bollen TL, van Santvoort HC, Besselink MGH, et al. Update on acute pancreatitis: ultrasound, computed tomography, and magnetic resonance imaging features. Semin Ultrasound CT MRI. 2007;28:371–83.
59. Ichikawa K, Idoguachi K, Tanaka H, et al. Classification of acute pancreatitis based on retroperitoneal extension: application of the concept of interfascial planes. Eur J Radiol. 2006;20:445–52.
60. Balthazar EJ, Freeny PC, van Sonnenberg E. Imaging and intervention in acute pancreatitis. Radiology. 1994;193:297–306.
61. Alexander ES, Clark RA, Federle MP. Pancreatic gas: indication of pancreatic fistula. AJR. 1982;139:1089–93.
62. Miller FH, Keppke AL, Dalal K, Ly JN, Kamler VA, Sica GT. MRI of pancreatitis and its complications: part 1. Acute pancreatitis. AJR. 2004;183:1637–44.
63. Kim YK, Ko SW, Kim CS, Hwang SB. Effectiveness of MR imaging for diagnosing mild forms of acute pancreatitis: comparison with MDCT. J Magn Reson Imaging. 2006;24:1324–49.
64. Morgan DE, Baron TH, Smith JK, Robbin ML, Kenney PJ. Pancreatic fluid collection prior to intervention: evaluation with MR imaging compared with CT and US. Radiology. 1997;203:773–8.
65. Hirota M, Kimura Y, Ishiko T, Beppu T, Yamashita Y, Ogawa M. Visualization of the heterogeneous internal structure of so-called "pancreatic necrosis" by magnetic resonance imaging in acute necrotizing pancreatitis. Pancreas. 2002;25:63–7.
66. Ward J, Chalmers AG, Guthrie AJ, Larvin M, Robinson PJ. T2-weighted and dynamic MRI in acute pancreatitis: comparison with contrast enhanced CT. Clin Radiol. 1997;52:109–14.
67. Piironen A, Kivisaari R, Kemppainen E, Laippala P, Koivisto AM, Poutanen VP, et al. Detection of severe acute pancreatitis by contrast-enhanced magnetic resonance imaging. Eur Radiol. 2000;10:354–61.
68. Loperfido S, Angelini G, Benedetti G, Chilovi F, Costan F, De Berardinis F, et al. Major early complications from diagnostic and therapeutic ERCP: a prospective multicenter study. Gastrointest Endosc. 1998;48:1–10.
69. British Society of Gastroenterology. United Kingdom guidelines for the management of acute pancreatitis. Gut. 1998;42(suppl 2):S1–13.
70. Pezzilli R, Uomo G, Zerbi A, et al. Diagnosis and treatment of acute pancreatitis: the position statement of the Italian Association for the study of the pancreas. Dig Liver Dis. 2008;40:803–8.
71. Wang SS, Lin XZ, Tsai YT, et al. Clinical significance of ultrasonography, computed tomography, and biochemical tests in the rapid diagnosis of gallstone-related pancreatitis: a prospective study. Pancreas. 1988;3:153–8.
72. Ammori BJ, Boreham B, Lewis P, et al. The biochemical detection of biliary etiology of acute pancreatitis on admission: a revisit in the modern era of biliary imaging. Pancreas. 2003;26:e32–5.
73. Liu CL, Fan ST, Lo CM, et al. Clinico-biochemical prediction of biliary cause of acute pancreatitis in the era of endoscopic ultrasonography. Aliment Pharmacol Ther. 2005;22:423–31.
74. Tenner S, Dubner H, Steinberg W. Predicting gallstone pancreatitis with laboratory parameters: a meta-analysis. Am J Gastroenterol. 1984;89:1863–6.
75. Liu CL, Lo CM, Chan HKF, et al. Detection of choledocholithiasis by EUS in acute pancreatitis: a prospective evaluation in 100 consecutive patients. Gastrointest Endosc. 2001;54:325–30.

76. Fogel EL, Sherman S. Acute biliary pancreatitis: when should the endoscopist intervene? Gastroenterology. 2003;125:229–35.
77. Moon JH, Cho YD, Cha SW, et al. The detection of bile duct stones in suspected biliary pancreatitis: comparison of MRCP, ERCP, and intraductal US. Am J Gastroenterol. 2005;100:1051–7.
78. Saifuddin A, Ward J, Radgway J, Chalmers AG. Comparison of MR and CT scanning in severe acute pancreatitis: initial experiences. Clin Radiol. 1993;48:111–6.
79. Hallal AH, Amortegui JD, Jeroukhimov IM, et al. Magnetic resonance cholangiopancreatography accurately detects common bile duct stones in resolving gallstone pancreatitis. J Am Coll Surg. 2005;200(6):869–75.
80. Makary MA, Duncan MD, Harmon JW, et al. The role of magnetic resonance cholangiography in the management of patients with gallstone pancreatitis. Ann Surg. 2005;241(1):119–24.
81. Lomas DJ, Bearcroft PWP, Gimsa AE. MR cholangiopancreatography: prospective comparison of a breath-hold 2D projection technique with diagnostic ERCP. Eur Radiol. 1999;9:1411–7.
82. Hirohashi S, Hirohashi R, Uchida H, et al. Pancreatitis: evaluation with MR cholangiopancreatography in children. Radiology. 1997;203:411–5.
83. Chak A, Hawes RH, Cooper GS, et al. Prospective assessment of the utility of EUS in the evaluation of gallstone pancreatitis. Gastrointest Endosc. 1999;49:599–604.
84. Liu CL, Lo CM, Chan JKF, et al. EUS for detection of occult cholelithiasis in patients with idiopathic pancreatitis. Gastrointest Endosc. 2000;51:28–32.
85. Norton SA, Alderson D. Endoscopic ultrasonography in the evaluation of idiopathic acute pancreatitis. Br J Surg. 2000;87:1650–5.
86. Frossard JL, Sosa-Valencia L, Amouyal G, et al. Usefulness of endoscopic ultrasonography in patients with "idiopathic" acute pancreatitis. Am J Med. 2000;109:196–200.
87. Kamisawa T, Egawa N, Matsumoto G, et al. Pancreatographic findings in idiopathic acute pancreatitis. J Hepatobiliary Pancreat Surg. 2005;12:99–102.
88. Kamisawa T, Tu Y, Nakajima H, et al. Acute pancreatitis and a long common channel. Abdom Imaging. 2007;32:365–9.