Clinical Practice Guideline

Acute and Chronic Pancreatitis

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Summary

Background: Acute pancreatitis (AP) is among the commonest non-malignant admission diagnoses in gastroenterology. Its incidence in Germany lies between 13 and 43 per 100 000 inhabitants and is increasing. In 2017, 24 per 100 000 inhabitants were hospitalized for chronic pancreatitis.

Methods: From October 2018 to January 2019, we systematically searched the literature for original articles, meta-analyses, and evidence-based guidelines that were published in German or English between 1960 and 2018.

Results: 30–50% of cases of acute pancreatitis are caused by gallstone disease, and another 30–50% are due to alcohol abuse. The diagnosis is made when at least two of the following three criteria are met: typical abdominal pain, elevation of serum lipase, and characteristic imaging findings. If those criteria are ambiguous, transabdominal sonography is indicated. The early initiation of food intake lowers the rate of infected pancreatic necrosis, organ failure, or death (odds ratio 0.44; 95% confidence interval [0.2; 0.96]). In AP, Ringer's lactate solution should be preferred for fluid resuscitation, at 200–250 mL/hr for 24 hours. Severe pain should be treated with opiates.

Conclusion: The current German clinical practice guideline reflects the developments in the diagnosis and treatment of pancreatitis that have taken place over the past few years. The long-term care and monitoring of patients with complication-free pancreatitis is the responsibility of primary care physicians and gastroenterologists.

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Acute pancreatitis (AP) is one of the most common admission diagnoses among patients hospitalized for nonmalignant gastrointestinal conditions. In Germany, the incidence of acute pancreatitis ranges from 13 to 43 cases per 100 000 population, with an upward trend. (1). In 2017, 24 patients per 100 000 population were hospitalized for chronic pancreatitis in Germany (2).

The S3-level clinical practice guideline on pancreatitis is presented below. The full text of the guideline has been published in the Zeitschrift für Gastroenterologie and is posted on the websites of the Association of the Scientific Medical Societies in Germany (AWMF, Arbeitsgemeinschaft der Wissenschaftlichen Medizinischen Fachgesellschaften) and the German Society of Gastroengerology, Digestive and Metabolic Diseases (DGVS, Deutschen Gesellschaft für Gastroenterologie, Verdauungs- und Stoffwechselkrankheiten) (3). All recommendation of this guideline are listed in *eTable 1*.

Methods

The methodological approach is described in the guideline report (e1). Further details are provided in *eTable 2*.

Acute pancreatitis

Definition and etiology

Gallstone disease and alcohol abuse are the most common established risk factors for AP (level of evidence: 3, strong consensus). They each account for 30% to 50% of cases. Further common risk factors include hypertriglyceridemia (approximately 10% of cases), endoscopic retrograde cholangiopancreatography (ERCP) (relative risk of approximately 3–5%) and smoking. A meta-analysis of 12 observational studies found an odds ratio (OR) of 1.71 (95% confidence interval: [1.37; 2.14]) for smokers and 1.21 [1.02; 1.43]

TABLE 1

Definition of severity according to the Revised Atlanta Classification (6) (RAC) with corresponding mortality rates according to (40)

RAC	Mild	Moderate	Severe
Organ failure (OF) *1	No	Transient (<48 h) OF	Persistent (>48 h) single or multiple OF
		and/or	
Local or systemic complications*2	No	Present	Typically present
Mortality (%)	0.1	2.1	52.2

*1 Defined according to the modified Marshall scoring system for organ failure
*2 Example of local complications: infected necrosis, portal vein thrombosis
Examples of systemic complications: exacerbated chronic obstructive pulmonary disease, decompensated heart failure, delirium with known dementia

for ex-smokers (4, 5).Thus, tobacco smoke ought to be considered the likely most important risk factor (level of evidence: 2, strong consensus).

AP can be diagnosed if at least two of the following three criteria are met:

- typical abdominal pain (acute onset of persistent upper abdominal pain, often radiating to the back)
- serum lipase activity at least three times greater than the upper limit of normal
- characteristic morphology findings in imaging (strong consensus).

Given the poorer specificity of serum amylase, only the measurement of serum lipase is recommended.

Severity classification

The severity of AP is determined retrospectively based on the presence of organ failure and complications, and is classified according to the revised Atlanta classification (RAC) (6) as mild, moderate or severe (level of evidence: 3, strong consensus) (*Table 1*). Persistent organ failure (>48 h) is considered the best predictor of mortality.

Risk factors (age, comorbidity) as well as clinical (criteria for systemic inflammatory response syndrome [SIRS]) and laboratory parameters—determined on admission and after 48 hours—ought to be used to predict severity. From this, prognostic scoring systems can be derived (level of evidence: 3, consensus).The presence of SIRS on the day of hospital admission has a high sensitivity (85–100%) in predicting a severe disease course. By contrast, the absence of SIRS has a high negative predictive value (98–100%). In addition, it was shown that persistent SIRS (>48 h) is associated with a complicated disease course (7). Monitoring and re-evaluation of patients with acute pancreatitis ought to be performed daily and based on clinical findings, SIRS criteria and the presence of organ failure, as well as laboratory testing of parameters such as C-reactive protein (level of evidence: 3, strong consensus). The degree of lipase elevation does not correlate with severity and is only used to confirm the diagnosis (8).

Imaging

The initial aim of imaging in acute pancreatitis is to establish the diagnosis if signs, symptoms and laboratory criteria are inconclusive. If this is the case, first transabdominal ultrasound ought to be performed. To evaluate the extent of necrosis, computed tomography (CT) is indicated but ought not to be performed within the first three days after symptom onset (level of evidence: 4, strong consensus). Several studies have shown that a CT scan performed within six days after hospitalization does not lead to a change in therapeutic approach, but significantly prolongs the hospital stay (9). In a prospective case series with 128 patients with idiopathic acute pancreatitis, it was possible to revise the diagnosis "idiopathic" in 50% of cases, using a combination of endoscopic ultrasound (EUS) and magnetic resonance cholangiopancreatography (MRCP) (10).

Fluid therapy, pain management and intensive care management

Fluid therapy

Several randomized controlled trials (RCTs) have confirmed a correlation between fluid resuscitation and clinical course of pancreatitis. Thus, controlled fluid therapy is recommended (level of evidence: 1, strong consensus). Initially, fluid should be replaced at a rate of 200–250 mL/h. A retrospective study analyzed the outcome of 1097 patients in relation to fluid replacement, taking into account the initial disease severity (11). After adjustment for various risk factors, it was shown that fluid replacement of at least 6000 mL/24 h (250 mL/h) during the first 24 hours was associated with a reduction in mortality (OR 0.58; 95% confidence intervals [0.34; 0.98]). Fluid replacement at a rate of less than 150 mL/h on day 1 was found to be disadvantageous; however, fluid replacement at a rate of more than 250 mL/h also showed no benefit in any study (12). Initial fluid therapy should primarily be performed with Ringer's lactate solution (level of evidence: 1, strong consensus).

Pain management

Opioids should be used for pain management in patients with severe pain. Epidural anesthesia may be used in intensive care units (level of evidence: 2, strong consensus). None of the available data indicates that the active substances piritramide, morphine, fentanyl, and sufentanil, which are widely used in Germany, worsen the prognosis.

Transfer to intensive care unit and/or specialized center

Patients should be transferred to an intensive care unit if markers for an unfavorable prognosis are present (level of evidence: 2, strong consensus). Furthermore, if a severe clinical course and/or organ failure is to be expected, patient transfer to a specialized center ought to be considered (level of evidence: 2, consensus). A specialized pancreatic center ought to be equipped 24/7

with an intensive care unit (>15 ICU patients treated for pancreatitis annually), a department of diagnostic and interventional radiology, a diagnostic and interventional endoscopy unit, and a surgical department, each of them with expertise in the management of acute pancreatitis (>115 hospital admissions for AP annually [13, 14]) (level of evidence: 5, strong consensus).

Antibiotics and nutrition

Antibiotic therapy

Antibiotic prophylaxis to prevent infectious complications in patients with predicted severe pancreatitis cannot be generally recommended (level of evidence: 2, strong consensus). A recently published meta-analysis of 11 RCTs with a total of 747 patients found no significant difference in mortality (OR 0.71 [0.44; 1.15]; p = 0.16); however, the occurrence of infected pancreatic necrosis was significantly reduced in the group receiving antibiotics (OR 0.59 [0.42; 0.84]; p = 0.004) (15).

Nutrition

Enteral nutrition prevents severe catabolism and infectious complications by preserving mucosal integrity. Patients with mild pancreatitis should be offered oral nutrition within the first day after hospital admission (level of evidence: 1, strong consensus). A meta-analysis of data of 165 patients from 8 randomized studies showed for early nutrition a reduction in the composite endpoint (mortality, infected pancreatic necrosis and organ failure) from 45% to 19% (OR 0.44 [0.2; 0.96]) (16). If oral food intake is impossible, enteral nutrition should be started as early as possible, even in patients with severe or predicted severe disease course (level of evidence: 2, consensus). Two meta-analyses showed that early enteral nutrition initiated within 24 or 48 hours of admission to hospital was associated with reduced infection rates (OR between 0.44 and 0.38) (17, 18). Feeding via nasojejunal or nasogastric tube are to be considered equivalent (level of evidence: 1, strong consensus).

Acute biliary pancreatitis and management of biliary complications

In patients with suspected biliary pancreatitis, transabdominal ultrasound ought to be performed as the primary imaging modality to diagnose gall bladder stones. In pancreatitis of unclear etiology, endoscopic ultrasound (EUS) can identify a biliary origin with a high degree of accuracy and was found to be superior to magnetic resonance imaging (MRI)/MRCP in a meta-analysis (34% versus 9%) (19). A >3-fold increase in alanine aminotransferase (ALT) levels within the first 48 h after symptom onset has a positive predictive value >85% for biliary etiology (1).

Patients with acute biliary pancreatitis should undergo endoscopic retrograde cholangiopancreatography (ERCP) with sphincterotomy in the presence of concomitant cholangitis, confirmed choledocholithiasis and/or biliary obstruction (level of evidence: 1/2, strong consensus). A prospective randomized trial from the Netherlands, the APEC trial, did not find a statistically significant advantage of early ERCP (<24 h) in patients with predicted severe pancreatitis without cholangitis (relative risk [RR] 0.87 [0.64; 1.18]; p = 0.37) (20).

In mild biliary pancreatitis, cholecystectomy should be performed during the initial hospital stay (level of evidence: 1, consensus). The largest randomized controlled trial, the PONCHO trial (21), compared early (within 3 days) and late (within 25–30 days) cholecystectomy in 266 patients with mild biliary pancreatitis. It showed a statistically significant advantage for early cholecystectomy during the initial hospital stay (RR 0.28 [0.12; 0.66]; p = 0.002) (21).

Indication, timing and treatment methods in infected necrosis

Superinfection of (peri-) pancreatic necrosis occurs in about 20% to 40% of patients with severe acute pancreatitis. In a systematic review and meta-analysis of a total of 6970 patients, patients with infected necrosis and organ failure had a mortality of 35.2%. In patients with infected necrosis without organ failure,

BOX 1

Causes of pancreatic exocrine insufficiency

Loss of pancreatic parenchyma

- Chronic pancreatitis
- Severe acute pancreatitis
- Pancreatic cancer
- Status post pancreatic resection
- Cystic fibrosis (mucoviscidosis)

Other common causes

- Pancreatic duct obstruction
- Reduced endogenous stimulation
  - Gluten-sensitive enteropathy (celiac disease)
  - Diabetes mellitus, with insulin deficiency (paracrine insulin action stimulates pancreatic enzyme synthesis)
  - Status post gastric surgery (gastric resection of the Billroth I or II type, gastrectomy, gastrojejunal bypass); these surgical procedures are followed by pancreaticocibal asynchrony the release of cholecystokinin(CCK) and secretin does not occur in synchrony with the necessary secretion of digestive enzymes into the jejunum

Rare causes

- Protein deficiency
- Shwachman-Diamond syndrome
- Johanson-Blizzard syndrome
- Hereditary enzyme defects (deficiency of trypsinogen, enterokinase, other proteases or alpha-1 antitrypsin)
- Lack of amylase, lipase
- Early inactivation of enzymes (hyperchlorhydria); pancreatic lipase is particularly sensitive to acid destruction.

TABLE 2

Comparison of imaging modalities employed to diagnose chronic pancreatitis (27)

Imaging	Sensitivity % [95% CI]	Specificity % [95% CI]
CT	75 [66; 83]	91 [81; 96]
MRI	78 [69; 85]	96 [90; 98]
EUS	81 [70; 89]	90 [82; 95]
ERCP	82 [76; 87]	94 [87; 98]
US	67 [53; 78]	98 [89; 100]

CT, computed tomography; EUS, endoscopic ultrasound; ERCP, endoscopic retrograde cholangiopancreatography ; CI, confidence interval; MRI, magnetic resonance imaging; US, ultrasound

TABLE 3

Chronic Pancreatitis Prognosis Score (COPPS), adapted from (28)

1 point	2 points	3 points
NRS (0–10), most severe pain intensity in the last 7 days		
0–2	3–6	7–10
HbA1c (%)		
> 6.0	5.5–6.0	< 5.5
CRP (mg/L)		
< 3.1	3.1–20	> 20
BMI (kg/m²)		
> 25	18–25	< 18
Platelets (Gpt/L)		
150–400	100–150	< 100, > 400
COPPS A = 5–6 points	**COPPS B = 7–9 points**	**COPPS C = 10–15 points**

BMI, body mass index; CRP, C-reactive protein; NRS, numerical rating scale

mortality was 1.4% (22). Not every necrosis requires treatment, as spontaneous remission is seen in up to 39% of cases (e2). In patients with infected pancreatic necrosis, an intervention should be performed if clinical necessary (e.g., in case of a septic disease course) (level of evidence: 2, strong consensus). The endoscopic approach is as effective as the percutaneous approach, but it less frequently results in fistula formation and it reduces the length of the hospital stay. Therefore, primarily an endoscopic approach ought to be pursued (level of evidence: 2, consensus) (23, 24). Given the side effects associated with each approach, the least invasive method should be the primary choice and only if it fails, escalation to a more invasive approach should follow (step-up approach) (level of evidence: 1, strong consensus).

Follow-up

After a first episode of acute pancreatitis, a structured follow-up ought to be recommended to the following patients (level of evidence: 2, consensus):

- non-mild severity, irrespective of etiology
- alcohol-induced pancreatitis, any severity
- any severity in case of unclear etiology and age >40 years
- no complete resolution of symptoms after discharge.

Several population-based cohort studies with a follow-up period of up to 10 years after initial diagnosis of acute pancreatitis have described an increased risk of a first diagnosis of pancreatic cancer. The adjusted hazard ratio (HR) is up to 19.28 [14.62; 25.41] compared to the respective controls. However, the more time passes after pancreatitis, the lower the risk. After 5–10 years it returns to the risk of the general population (25). Patient age above 40 years and diabetes mellitus further increase the risk. Thus, symptom-free patients with a first episode of acute pancreatitis of unknown etiology and age >40 years ought to undergo contrast-enhanced cross-sectional imaging or endoscopic ultrasound no later than three months after the resolution of an attack of acute pancreatitis to rule out pancreatic cancer. Another imaging follow-up after 12–24 months may be performed (level of evidence: 2, strong consensus) (*Box 2*).

Chronic pancreatitis

Definition, epidemiology and etiology

Chronic pancreatitis (CP) is a disease of the pancreas in which recurrent inflammatory episodes result in the replacement of pancreatic parenchyma by fibrous connective tissue. Alcohol consumption is the most common confirmed cause of chronic pancreatitis. Further causes include tobacco use, autoimmune diseases, malignancies, genetic alterations, and primary hyperparathyroidism, among others. In some cases, the cause of the disease cannot be identified (level of evidence: 4, strong consensus). In epidemiological studies, the risk from tobacco smoking is greater than that from alcohol consumption. In one study, the OR for current smoking as an independent risk factor was 1.99 [1.01; 3.91] (26). In patients with suspected genetically determined chronic pancreatitis, initially the genes PRSS1 (exons 2 and 3), SPINK1 (exon 3) and CPA1 (exons 7, 8 and 10) ought to be assessed in compliance with the German Genetic Diagnostics Act. Given the advances in sequencing technology, it would seem useful to investigate all CP-associated variants in the future (level of evidence: 5, strong consensus).

Pancreatic exocrine insufficiency

Pancreatic exocrine insufficiency (PEI) is defined as reduced secretion of pancreatic enzymes and/or bicarbonate into the duodenum. The main causes are summarized in *Box 1*.

Development and clinical signs and symptoms

There are no clinical signs and symptoms that allow to specifically diagnose pancreatic exocrine insufficiency. A noninvasive pancreatic function test should be performed to detect pancreatic exocrine insufficiency.

BOX 2

Key recommendations at a glance

- The severity of acute pancreatitis is determined by organ failure and complications along the disease course.
- To determine the extent of pancreatic necrosis, a computed tomography (CT) scan ought not to be obtained within the first three days after symptom onset.
- Goal-directed fluid therapy (GDFT) in patients with severe acute pancreatitis ought to be guided by serum urea and hematocrit levels as well as parameters of advanced hemodynamic monitoring, among others. The central venous pressure (CVP) ought to be used to guide goal–directed fluid therapy.
- Initial fluid resuscitation should primarily be performed with Ringer's lactate solution.
- If a severe clinical course and/or organ failure is to be expected, patient transfer to a specialized center ought to be considered.
- Antibiotic prophylaxis to prevent infectious complications in patients with predicted severe acute pancreatitis cannot be generally recommended.
- Enteral nutrition ought to be started as early as possible in patients with severe or predicted severe disease course.
- Endoscopic retrograde cholangiopancreatography (ERCP) should not be performed in patients with mild biliary pancreatitis without cholangitis and/or in the absence of evidence of choledocholithiasis or biliary obstruction.
- ERCP should not be performed in patients with predicted severe and complicated biliary pancreatitis without cholangitis and/or in the absence of evidence of choledocholithiasis or biliary obstruction.
- In mild biliary pancreatitis, cholecystectomy should be performed during the initial hospital stay.
- Given the side effects associated with each approach, the least invasive method should be the primary choice in treating infected walled-off pancreatic necrosis, and only if this fails, escalation to a more invasive approach should follow (step-up approach).
- In the absence of a clear biliary or obstructive mechanical etiology, patients with a single episode of acute pancreatitis ought to be recommended to abstain from nicotine and alcohol.
- Alcohol consumption is the most common confirmed cause of chronic pancreatitis. Further causes include tobacco use, autoimmune diseases, malignancies, genetic alterations, and primary hyperparathyroidism, among others. In at least 15% of cases, the etiology of the disease cannot be identified.
- The morphological changes in chronic pancreatitis ought to be classified using the Cambridge classification, adapted to the respective imaging modality..
- The Chronic Pancreatitis Prognosis Score (COPPS) ought to be used to estimate the severity of the short- and medium-term course of chronic pancreatitis.
- Pain management in chronic pancreatitis may be provided based on the WHO analgesic ladder. Pregabalin may be used to supplement pain medication.
- Patients with chronic pancreatitis and overt pancreatic exocrine insufficiency (weight loss, malnutrition) ought to receive pancreatic enzyme replacement therapy along with a personalized nutritional intervention to prevent or stop deterioration of nutritional status in a goal-directed manner.
- Painful chronic pancreatitis should not be treated with opioids for more than six months before surgical treatment is recommended in patients with a clearly dilated pancreatic duct. If an alternative endoscopic therapy over a period of no more than three months does not achieve a pain-free state, a surgical procedure should be recommended.
- Patients with hereditary pancreatitis ought to undergo annual clinical, imaging and laboratory follow-up examinations from age 40 years onward or 20 years after symptom onset in the pancreatic center.

Suitable analyses include testing for fecal elastase 1 (with specific antibodies) and a breath test with 13C-marked lipids (level of evidence: 1, strong consensus). Exocrine pancreatic function assessment ought to be performed at the time of diagnosis of PEI and followed up over the course of the disease depending on clinical symptoms.

Imaging and staging

Various imaging studies are available which help to diagnose chronic pancreatitis. EUS, CT and MRI are the imaging modalities with the highest sensitivity and specificity for confirmation of the diagnosis of chronic pancreatitis (*Table 2*) (27). For the morphological evaluation of chronic pancreatitis by EUS, parenchymal and ductal signs have been identified and weighted as diagnostic criteria (Rosemont classification, *eTable* 3) (e3).

With the aim of standardizing findings, the morphological changes seen with chronic pancreatitis should be classified according to the Cambridge classification—modified for the corresponding imaging modalities (clinical consensus point, strong consensus). The Chronic Pancreatitis Prognosis Score (COPPS) ought to be used to estimate the severity of the short- and medium-term clinical course of chronic

pancreatitis (level of evidence: 2, consensus). The COPPS model is based on the model for classifying the prognosis of liver cirrhosis (Turcotte-Child-Pugh score). The COPPS model (*Table 3*) uses the number of hospital admissions and in-hospital days within one year as surrogate markers for severity (28).

Pharmacotherapy

The majority of patients with chronic pancreatitis are symptomatic and require long-term specialized outpatient care. Complementary to pain management, a treatment attempt with pregabalin can be considered (level of evidence: 2, strong consensus) (RCT: mean reduction of pain score 12% [−21.82; −2.18]) (29).

Interventional therapy

With regard to interventional therapy, a prerequisite for deciding on the best treatment strategy is the availability of an interdisciplinary treatment team. For pancreatic surgery, it has been shown that centers with high case load achieve better patient outcomes and significantly lower mortality compared to centers with lower case load, regardless of the underlying primary disease (30, 31).

Pain requiring long-term analgesic therapy can be treated efficiently with both endoscopic and surgical procedures (32). A recent study reported that patients with pancreatic duct dilatation >5mm, who underwent surgery within few months after initiation of opioid therapy, showed superior pain control compared to patients treated according to a step-up approach (pharmacological pain management, endoscopy and extracorporeal shock wave lithotripsy, surgery) (33).

Prospective non-randomized studies have shown long-term success (>2 years) for the treatment of biliary strictures with fully covered self-expandable metal stents (fcSEMS) or with multiple plastic stents (up to six 10 Fr stents) in 50% to 90% of cases (34–38). Comparative studies of the surgical treatment with fcSEMS are not available. The available average long-term follow-up data (up to five years) suggest that both approaches are equally effective.

Pancreatic pseudocyst complications include: pain, gastric of duodenal stenosis, biliary stenosis, pancreatic duct stenosis, infections, hemorrhage, pancreaticopleural fistula, and recurrent episodes of chronic pancreatitis. As the first-line treatment, endoscopic drainage of pancreatic pseudocysts ought to be performed; in case of pseudocyst recurrence, surgical treatment may be considered (level of evidence: 3, consensus).

Monitoring and follow-up

Follow-up care of patients with uncomplicated chronic pancreatitis ought to be provided by specialists in internal medicine, general practitioners and gastroenterologists involved in primary care.

In patients with hereditary pancreatitis, the risk of developing pancreatic cancer is increased 9– to 70-fold (e4). Patients with hereditary pancreatitis ought to undergo annual clinical, imaging and laboratory follow-up examinations from age 40 years onward or 20 years after symptom onset in a pancreatic center (level of evidence: 3, strong consensus).The recommendation made here is in line with the consensus recommendation of the International Cancer of the Pancreas Screening (CAPS) consortium (39).

Conflict of interest statement

Dr. Beyer received fees for continuing medical education events from Falk Foundation e. V. and Akcea. He received fees for court-ordered expert opinions with content related to the manuscript. In addition, he is the spokesperson for the DGVS Working Group "Pancreas", coordinator of the S3-level clinical practice guideline Pancreatitis of the DGVS and a Member of the Board of the German Pancreas Club.

Mrs. Lorenz is a spokeswoman of the DGVS.

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Prof. Lerch is the chairman of the German Society of Internal Medicine (DGIM).

Prof. Mayerle received fees for continuing medical education events from Falk Foundation e. V. She is Chair of the Scientific Committee UEG as well as Member of the Advisory Board of the DGVS and Member of the Advisory Board of the Working Group of Patients After Pancreatectomy.

Prof. Hoffmeister declares that no conflict of interest exists.

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Clinical guidelines are not peer-reviewed in the Deutsche Ärzteblatt, as well as in many other journals, because clinical (S3) guidelines are texts which have already been repeatedly evaluated, discussed and broadly consented by experts (peers).

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► **Supplementary material**

eReferences, eTables, eBoxes:
www.aerzteblatt-international.de/m2022.0223

Questions on the article in issue 29–30/2022:

Clinical Practice Guideline: Acute and Chronic Pancreatitis

cme plus

The submission deadline is 24 July 2023. Only one answer is possible per question. Please select the answer that is most appropriate.

Question 1

What are the most common established risk factors for acute pancreatitis?

a) Diabetes and hypertension
b) Hypertension and gastritis
c) Gastric ulcer and hypertriglyceridemia
d) Mediterranean diet and genetic factors
e) Gallstones and alcohol abuse

Question 2

What does the abbreviation SIRS stand for in the text?

a) Systemic infectious reaction syndrome
b) Superinfection response syndrome
c) Severe infection response syndrome
d) Systemic inflammatory response syndrome
e) Severe inflammatory reaction symptoms

Question 3

What imaging modality should first be performed in patients admitted for suspected biliary pancreatitis?

a) Abdominal CT scan
b) Transabdominal ultrasound
c) Abdominal MRI with contrast
d) Abdominal MRI without contrast
e) Abdominal x-ray

Question 4

More or less, what is the mortality rate in patients with infected (peri-)pancreatic necrosis and organ failure?

a) Up to 0.5%
b) Up to 5%
c) Up to 10%
d) Up to 35%
e) Up to 55%

Question 5

What solution ought to be used for initial fluid resuscitation in patients with acute pancreatitis?

a) Ringer's lactate solution
b) Ringer's acetate solution
c) 0.9% saline solution
d) Ringer's acetate malate solution
e) Glucose solution 5%

Question 6

Which of the following laboratory parameters is a useful pancreatic function test for detecting pancreatic exocrine insufficiency?

a) Urinary elastase 1
b) Fecal calprotectin
c) Fecal elastase 1
d) Fecal lactoferrin
e) Urinary pH value

Question 7

What endpoints (for the period of one year) were used in the development of the COPPS model that determines the severity of chronic pancreatitis?

a) Number and duration of hospital treatments
b) Number and duration of antibiotic administrations
c) Number of outpatient visits and drug treatments
d) Number and duration of pain episodes
e) Number of imaging modalities used and drug prescriptions

Question 8

In the management of acute pancreatitis, what volume per unit of time ought to be used for controlled fluid therapy?

a) 20–50 mL/h
b) 50–100 mL/h
c) 150–200 mL/h
d) 200–250 mL/h
e) 300–350 mL/h

Question 9

What substances ought to be used for the pharmacological management of severe pain in patients with pancreatitis?

a) Nonsteroidal anti-inflammatory drugs
b) Opioids
c) Nitrous oxide (laughing gas)
d) Cyclooxygenase inhibitors
e) Antipyretic analgesics

Question 10

Which of the following conditions is one of the rare causes of pancreatic exocrine insufficiency?

a) Pancreatic cancer
b) Cystic fibrosis
c) Chronic pancreatitis
d) Celiac disease
e) Inherited enzyme defects

Supplementary material to:

Acute and Chronic Pancreatitis

by Georg Beyer[*1], Albrecht Hoffmeister[*1], Pia Lorenz, Petra Lynen, Markus M. Lerch[*2], and Julia Mayerle[*2]

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eBOX

Representativeness of the Guideline Development Group

- **Participating medical societies (*collaborators)**
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eTABLE 1

Recommendations of the DGVS S3-level Clinical Practice Guideline for Pancreatitis

Recommendation	Level of evidence, consensus
Definition and etiology of acute pancreatitis	
Pathophysiologically, acute pancreatitis is defined as a primarily sterile inflammation of the pancreas characterized by abnormal enzyme activation, causing an inflammatory reaction with edema, vascular damage and cell death.	-, Consensus
Recurrent acute pancreatitis is defined as two or more attacks of acute pancreatitis, irrespective of its etiology, accompanied by symptom-free intervals, without evidence of chronic pancreatitis.	-, Strong consensus
Asymptomatic hyperlipasemia is defined as a serum lipase activity at least three times greater than the upper limit of normal in the absence of clinical symptoms and imaging morphology criteria for pancreatitis.	-, Consensus
Gallstone disease and alcohol abuse are the most common established risk factors for acute pancreatitis.	Level of evidence: 3, strong consensus
Hypertriglyceridemia is associated with an increased risk of acute pancreatitis.	Level of evidence: 1, strong consensus
Endoscopic retrograde cholangiopancreatography (ERCP) and balloon enteroscopy are associated with an increased risk of post-interventional pancreatitis.	Level of evidence: 4, consensus
Tobacco smoking is associated with an increased risk of non-biliary acute pancreatitis.	Level of evidence: 2, strong consensus
Type 2 diabetes mellitus is associated with an increased risk of acute pancreatitis.	Level of evidence: 2, strong consensus
Numerous drugs can trigger acute pancreatitis. In most cases, the diagnosis is based on the exclusion of other causes. In order to conclusively prove a causal relationship, a re-exposure test would be necessary; however, this is often not ethically justifiable.	-, strong consensus
There is insufficient evidence to support an increased risk of pancreatitis from the use of the GLP-1 receptor agonists exenatide and liraglutide.	Level of evidence: 1, strong consensus
The use of DPP4 inhibitors is associated with an increased risk of lipasemia; however, the increase in risk of clinically symptomatic pancreatitis is unknown.	Level of evidence: 1, consensus
The population-based incidence of acute pancreatitis shows a high regional variability of 13–100/100 000 population in the western world. In Germany, the incidence varies between 13 and 43/100 000 population.	Level of evidence: 1, strong consensus
The incidence of alcohol-induced acute pancreatitis peaks in men between age 35 and 44 years and in women between age 25 and 34 years. The incidence of biliary acute pancreatitis significantly increases with increasing age from age 55 years onwards and peaks in patients aged >75 years.	Level of evidence: 1, strong consensus
Acute pancreatitis can be diagnosed if at least two of the following three criteria are met: (1) typical abdominal pain (acute onset of persistent upper abdominal pain, often radiating to the back); (2) serum lipase activity at least three times greater than the upper limit of normal; and (3) characteristic morphology findings in imaging.	Strong consensus
In order to confirm/rule out biliary etiology, laboratory testing for cholestasis parameters and aminotransferases as well as an upper abdominal ultrasound scan ought to be performed at the time of admission. In order to rule out hypertriglyceridemia/hypercalcemia-induced pancreatitis, serum levels of triglycerides and albumin-adjusted calcium ought to be obtained.	Level of evidence: 5, strong consensus
In case of unclear etiology, endoscopic ultrasound ought to be performed to confirm/rule out biliary etiology or pancreatic mass or pancreas divisum. Alternatively or additionally in case of inconclusive findings, MRI/MRCP ought to be performed to assess the biliary and pancreatic duct system and to rule out solid or cystic masses. With regard to the work-up for suspected autoimmune etiology, please refer to the corresponding guideline chapter (WG-8 CP).	Level of evidence: 1, strong consensus
In patients with recurrent episodes of acute pancreatitis, genetic predisposition testing (see chapter 1 – chronic pancreatitis) may be performed in addition to the examinations mentioned under 1.6.3.	Level of evidence: 5, strong consensus

Recommendation	Level of evidence, consensus
Classification of the severity of acute pancreatitis	
The severity of acute pancreatitis is determined by the presence of organ failure and complications and can be classified according to the Revised Atlanta Classification (RAC; mild, moderate, severe) or the Determinants-based Classification (DBC; mild, moderate, severe, critical).	Level of evidence: 3, strong consensus
Risk factors (age, comorbidity) as well as clinical (SIRS criteria) and laboratory parameters—determined on admission and after 48 hours—ought to be used to predict severity. Prognostic scoring systems can be developed from this.	Level of evidence: 3, consensus
Acute pancreatitis ought to be monitored and re-evaluated daily on the basis of clinical findings, SIRS criteria and organ failure as well as laboratory testing of parameters such as C-reactive protein. Scoring systems, such as the SOFA score as a composite of the named determinants, can help to better objectify the disease course.	Level of evidence: 3, strong consensus
Imaging in acute pancreatitis	
In acute pancreatitis, one goal of imaging is to obtain diagnostic criteria for acute pancreatitis, if symptoms and laboratory criteria are inconclusive. In addition, imaging should reveal complications of the disease in the course of acute pancreatitis, describe the extent of the disease and support treatment planning. Imaging can help classify the severity of the disease and rule out differential diagnoses.	Level of evidence: 5, strong consensus
In the presence of characteristic signs and symptoms and significantly elevated serum lipase levels (>3x ULN), no CT scan should be obtained to confirm the diagnosis of acute pancreatitis.	Level of evidence: 4, consensus
If acute pancreatitis is suspected, but the clinical and laboratory findings (serum lipase levels <3x ULN) are inconclusive, transabdominal ultrasound ought to be performed as the imaging modality of choice. If ultrasound findings are inconclusive, a contrast-enhanced CT scan ought to be performed.	Level of evidence: 5, consensus
If complications of acute pancreatic are clinically suspected, transabdominal ultrasound ought to be performed (level of evidence: 1). In case of inconclusive findings, contrast-enhanced CT should be the next step in the diagnostic work-up.	Level of evidence: 4, consensus
A CT scan to confirm a diagnosis of necrosis ought not to be performed within the first three days after symptom onset.	Level of evidence: 4, strong consensus
The modified CT Severity Index (MCTSI) may be used to assess the severity of acute pancreatitis based on CT findings.	Level of evidence: 4, strong consensus
As a general rule, the CT scan ought to be performed biphasically (arterial and portal-venous phase imaging). In the arterial contrast phase, only the upper abdomen ought to be scanned. By contrast, in the portal-venous phase the whole abdomen is scanned. With this approach it is possible to (i) obtain an adequate diagnosis; (ii) accurately determine pancreatic necrosis; (iii) comprehensively visualize fluid collections; and (iv) diagnose/rule out vascular complications.	Clinical consensus point, consensus
For contrast-enhanced CT scans, contrast media with iodine concentrations of 300 mg/mL or more ought to be used. The contrast medium ought to be administered intravenously with a flow rate of more than 3 mL/s. The amount of contrast medium administered ought to be between 1 and 1.5 mL/kg/KG (max. 150 mL), depending on the iodine concentration used and the device technology.	Clinical consensus point, consensus
In case of absolute contraindications to contrast-enhanced CT (e.g., known relevant contrast allergy with cardiovascular reaction or airway edema), non-contrast abdominal CT and supplementary contrast-enhanced upper abdominal MRI If a therapeutic consequences is to be expected, renal failure is not a contraindication to contrast-enhanced CT.	Level of evidence: 5, consensus
After an episode of acute idiopathic pancreatitis, endoscopic ultrasound (or MRI with MRCP) ought to be performed in the interval to rule out choledocholithiasis or a space-occupying lesion.	Level of evidence: 4, consensus
Fluid therapy, pain management and intensive care management in acute pancreatitis	
In acute pancreatitis, controlled fluid therapy should be performed.	Level of evidence: 1, strong consensus
Immediately after confirmation of the diagnosis, controlled fluid therapy ought to be started.	Level of evidence: 2 to 5, consensus

Recommendation	Level of evidence, consensus
As long as goal-directed fluid therapy (GDFT) cannot be started, initially 200–250 mL/h of fluid ought to be given. Based on the assumption of an already existing fluid deficit, administration of an initial bolus may be considered useful.	Level of evidence: 2, consensus
Instead of inflexible standard therapy, goal-directed fluid therapy ought to be administered.	Level of evidence: 2, consensus
Goal-directed fluid therapy in patients with severe acute pancreatitis ought to be guided by BUN (serum urea) and hematocrit levels as well as parameters of advanced hemodynamic monitoring, among others. The central venous pressure (CVP) ought not to be used as the goal of goal–directed fluid therapy.	Level of evidence: 2, consensus
Persistence or resolution of organ failure as well as SIRS criteria ought to be used for evaluating the response to initial fluid resuscitation.	Level of evidence: 2, strong consensus
Initial fluid resuscitation should primarily be performed with Ringer's lactate solution.	Level of evidence: 1, strong consensus
Opioids (preferably pentazocine, buprenorphine and pethidine) ought to be used for pain management in patients with severe pain. Epidural anesthesia may be used in intensive care units.	Level of evidence: 2, strong consensus
In patients treated with opioids, special attention ought to be paid to intestinal paralysis and respiratory depression. Epidural anesthesia ought to be accompanied by cardiovascular monitoring.	-, Strong consensus
Combinations of different analgesics may be used for pain management.	Level of evidence: 3, consensus
Intra-abdominal pressure ought to be determined by measuring the urinary bladder pressure.	Level of evidence: 2, strong consensus
Intraabdominal hypertension and compartment syndrome ought to be defined according to the World Society of Abdominal Compartment (WSACS) expert commission.	Level of evidence: 5, strong consensus
Patients with markers for an unfavorable prognosis (BISAP score ≥ 3, APACHE II score ≥ 8, Ranson score ≥ 3, elevated hematocrit [≥ 44% in men, ≥40 in women], BUN ≥ 25 mg/dL; increase in SOFA scores by ≥ 2 points, among others) ought to be transferred to an intensive care unit.	Level of evidence: 2, strong consensus
If a severe clinical course and/or organ failure is to be expected, patient transfer to a specialized center ought to be considered.	Level of evidence: 2, consensus
A center specialized in the management of acute pancreatitis ought to have an intensive care unit, a department of diagnostic and interventional radiology, a diagnostic and interventional endoscopy unit, and a surgical department continuously available, each of them with expertise in the management of acute pancreatitis.	Level of evidence: 5, strong consensus
Antibiotics, probiotics and nutrition in acute pancreatitis (prevention and management of infectious complications)	
Antibiotic prophylaxis ought not to be administered in patients with mild and predicted mild pancreatitis.	Level of evidence: 2, strong consensus
Antibiotic prophylaxis to prevent infectious complications in patients with predicted severe pancreatitis cannot be generally recommended.	Level of evidence: 2, strong consensus
If in septic patients with suspected infected (peri)pancreatic necrosis (based on clinical, laboratory and imaging findings) an anti-infectious treatment is started, a carbapenem antibiotic may be used until the results of antibiotic susceptibility testing have become available.	Level of evidence: 2, consensus
Antibiotic prophylaxis to prevent cholangitis can currently not be recommended.	Level of evidence: 5, consensus
Selective bowel decontamination cannot be generally recommended based on the limited data on acute pancreatitis currently available.	Level of evidence: 3, consensus
Probiotics should not be given to prevent infectious complications.	Level of evidence: 1, consensus
Patients with mild pancreatitis should be offered oral nutrition within the first day after hospital admission.	Level of evidence: 1, strong consensus
Patients with mild/predicted mild pancreatitis should not receive total parenteral nutrition.	Level of evidence: 1, strong consensus
Enteral nutrition ought to be started as early as possible in patients with severe or predicted severe disease course.	Level of evidence: 2, consensus

Recommendation	Level of evidence, consensus
For enteral nutrition, a high-molecular-weight tube feed should be used.	Level of evidence: 2, strong consensus
Feeding via nasojejunal or nasogastric tube are considered equivalent.	Level of evidence: 1, strong consensus
Acute biliary pancreatitis and management of biliary complications	
The diagnosis of biliary pancreatitis ought not to be established on the basis of only one clinical or laboratory parameter. Biliary pancreatitis is likely, if a combination of medical history (known gallstones disease, family history), laboratory parameters (elevated aminotransferase levels and cholestasis parameters) and imaging (ultrasound, EUS, MRI/MRCP, CT) is suggestive of biliary etiology.	Level of evidence: 2, strong consensus
Often, a clinical diagnosis of acute cholangitis cannot be made with sufficient reliability solely based on Charcot's triad (right-sided upper abdominal pain, elevated bilirubin levels and fever >38.4 °C) . Diagnostic accuracy increases in combination with biliary duct dilation and elevated cholestasis and inflammatory parameters. However, systemic inflammatory parameters, upper abdominal pain and elevated bilirubin levels can also occur in the absence of cholangitis in patients with acute biliary pancreatitis, making it significantly more challenging to rule out acute cholangitis.	Level of evidence: 5, consensus
ERCP should not be performed in patients with mild biliary pancreatitis without cholangitis and/or in the absence of evidence of choledocholithiasis or biliary obstruction.	Level of evidence: 2, strong consensus
ERCP ought not to be performed in patients with predicted severe and complicated biliary pancreatitis without cholangitis and/or in the absence of evidence of choledocholithiasis or biliary obstruction.	Level of evidence: 2, consensus
Patients with acute biliary pancreatitis and concomitant cholangitis should undergo ERCP with sphincterotomy.	Level of evidence: 1, strong consensus
Patients with biliary pancreatitis and confirmed choledocholithiasis and/or biliary obstruction should undergo ERCP with sphincterotomy.	Level of evidence: 2, strong consensus
Evidence from currently available studies does not allow to conclusively determine the optimum timing of ERCP with sphincterotomy in patients with acute biliary pancreatitis and confirmed choledocholithiasis and/or biliary obstruction without cholangitis.	Level of evidence: 2, strong consensus
The primary imaging modality used to detect biliary obstruction in patients with acute biliary pancreatitis should be transabdominal ultrasound with visualization of the intra- and extrahepatic biliary system as well as potential outflow obstructions (e.g. choledocholithiasis).	Level of evidence: 2, consensus
In patients with suspected acute biliary pancreatitis and no evidence of biliary dilation or mechanical outflow obstruction in transabdominal ultrasound, EUS or MRCP should be performed—ERCP intended entirely for diagnostic purposes is considered obsolete.	Level of evidence: 2, strong consensus
After an episode of acute biliary pancreatitis, a cholecystectomy ought to be performed.	Level of evidence: 2, strong consensus
In mild biliary pancreatitis, cholecystectomy should be performed during the initial hospital stay.	Level of evidence: 1, consensus
The timing of cholecystectomy in patients with severe (necrotizing) biliary pancreatitis is decided on a case-by-case basis and determined based on the clinical course of the severe acute pancreatitis.	Level of evidence: 5, strong consensus
If it is not possible to perform cholecystectomy after an episode of biliary pancreatitis, ERCP with sphincterotomy can be performed to lower the risk of recurrence.	Level of evidence: 3, strong consensus
Indication, timing and treatment methods in infected necrosis	
If an infected necrosis is clinically suspected, an endoscopic ultrasound (EUS) and contrast-enhanced cross-sectional imaging (CT or MRI) should be performed for further differential diagnostic work-up prior to a planned intervention.	Level of evidence: 2, consensus
In acute necrotizing pancreatitis, ultrasound, CT or endosonography-guided fine-needle aspiration ought to only be performed if imaging and/or clinical and laboratory findings over the disease course do not unequivocally support an intervention, while at the same time an infection of the necrosis is suspected.	Level of evidence: 2, consensus

Recommendation	Level of evidence, consensus
Prophylactic antibiotic/antifungal treatment without evidence of an infected (peri-)pancreatic necrosis (clinical, laboratory and imaging findings) should not be performed.	Level of evidence: 2, consensus
Patients with signs of infected pancreatic necrosis ought to be treated with antibiotics in parallel to the intervention (see below).	Level of evidence: 3, strong consensus
Patients with severe pancreatitis and confirmed bacteremia should be treated with antibiotics. If at the same time an invasive fungal infection is detected, antifungal treatment should be performed.	Level of evidence: 3, strong consensus
In presumably sterile necrotizing pancreatitis, patients with compression symptoms and/or early multi-organ failure should undergo an intervention.	Level of evidence: 2, strong consensus
In patients with infected pancreatic necrosis, an intervention should be performed if clinical necessary (e.g., in case of a severe/septic disease course).	Level of evidence: 2, strong consensus
The endoscopic approach (transgastric or transduodenal) is as effective as the percutaneous approach, but it is associated with a lower rate of fistula formation and reduced length of hospital stay. Therefore, primarily an endoscopic approach ought to be pursued.	Level of evidence: 2, consensus
In case of a difficult endoscopic approach, primarily a percutaneous drainage ought to be placed, which can be combined with a flexible endoscopic or inflexible minimally invasive surgical technique.	Level of evidence: 2, strong consensus
The type of intervention (drainage only, rinsing, necrosectomy) should be decided based on the presentation of the infected pancreatic fluid collection (extent, location, necrosis?).	Level of evidence: 2, strong consensus
Given the side effects associated with each approach, the least invasive method should be the primary choice and only if this fails, escalation to a more invasive approach should follow (step-up approach).	Level of evidence: 1, strong consensus
An open surgical procedure (laparotomy) should only be performed as the first-line treatment in exceptional cases.	Level of evidence: 3, consensus
Plastic stents or lumen-apposing metal stents (LAMS) ought to be used for endoscopy-guided drainage of infected (peri-)pancreatic necrosis.	Level of evidence: 2, strong consensus
If drainage is required for more than four weeks, plastic stents ought to be used.	Level of evidence: 2, strong consensus
Follow-up after acute pancreatitis	
After a first episode of acute pancreatitis, a structured follow-up ought to be recommended to the following patients: - non-mild severity, irrespective of etiology - alcohol-induced pancreatitis, any severity - any severity in case of unclear etiology and age >40 years - in the absence of complete resolution of symptoms after discharge	Level of evidence: 2, consensus
In symptom-free patients after the first episode of non-mild acute pancreatitis, an annual clinical follow-up examination and testing for exocrine and endocrine insufficiency (fecal elastase and HbA1c/fasting glucose) ought to be performed.	Level of evidence: 2, consensus
During the first two years after an episode of alcohol-induced acute pancreatitis, symptom-free patients ought to be recommended to undergo six-monthly clinical follow-up examinations and testing for exocrine and endocrine insufficiency by the family physician, community-based internist or gastroenterologist and a behavioral intervention. The goal is complete abstinence from alcohol.	Level of evidence: 2, consensus
Symptom-free patients with a first episode of acute pancreatitis of unknown etiology and age >40 years ought to undergo contrast-enhanced cross-sectional imaging or endoscopic ultrasound no later than three months after resolution of an attack of acute pancreatitis to rule out pancreatic cancer. Another imaging follow-up may be performed after 12–24 months.	Level of evidence: 2, strong consensus
In symptom-free diabetic patients with a one-time episode of acute pancreatitis of unknown etiology who are over age 40 years and in whom no signs of pancreatic neoplasia have been found over the course of the follow-up, extending the follow-up period to 5 years with annual imaging flow-ups may be considered.	Level of evidence: 3, strong consensus
In symptom-free patients with a first episode of non-alcohol-induced, mild acute pancreatitis of known and ideally treated etiology, a structured follow-up may be omitted.	Level of evidence: 2, consensus

Recommendation	Level of evidence, consensus
In symptom-free non-diabetic patients with a single episode of acute pancreatitis of unknown etiology who are over age 40 years and in whom no signs of pancreatic neoplasia have been found over the course of the follow-up, it may be considered to end the follow-up after two years.	Level of evidence: 2, consensus
In the absence of a clear biliary or obstructive mechanical etiology, patients with a single episode of acute pancreatitis ought to be recommended to abstain from nicotine and alcohol.	Level of evidence: 2 alcohol / 3 tobacco, strong consensus
Definition, epidemiology and etiology of chronic pancreatitis	
Chronic pancreatitis is a disease of the pancreas in which recurrent episodes of inflammation result in replacement of pancreatic parenchyma by fibrous connective tissue. The fibrous transformation of the pancreas leads to a progressive loss of exocrine and endocrine pancreatic function. In addition, there are characteristic complications, such as pseudocysts, pancreatic duct stenosis, duodenal stenosis, vascular complications, compression of the biliary ducts, malnutrition, and pain syndrome. Pain is the cardinal symptom of patients with chronic pancreatitis. Chronic pancreatitis is a risk factor for pancreatic cancer. The quality of life and life expectancy of patients with chronic pancreatitis is significantly reduced by the disease.	strong consensus, adopted from the 2012 guideline, reviewed in 2021
Alcohol consumption is the most common confirmed cause of chronic pancreatitis. Further causes include tobacco use, autoimmune diseases, malignancies, genetic alterations, and primary hyperparathyroidism, among others. In some cases, the etiology of the disease cannot be determined.	Level of evidence: 4, strong consensus
In patients with suspected genetically determined chronic pancreatitis, initially the genes PRSS1 (exons 2 and 3), SPINK1 (exon 3) and CPA1 (exons 7, 8 and 10) ought to be assessed. Given the advances in sequencing technology, it would seem useful to investigate all CP-associated variants in the future.	Level of evidence: 5, strong consensus
Genetic testing may be indicated in patients with a positive family history (at least one first-grade or two second-grade affected relatives) or with early onset of the disease (before age 30 years), if no other obvious cause can be identified.	Level of evidence: 5, strong consensus
If a genetic variant predisposing to chronic pancreatitis is identified in a patient, the patient should receive counselling by specialists in human genetics or physicians with specialist, special interest or additional qualification in genetic counseling pursuant to the German Genetic Diagnostics Act.	Pursuant to the German Genetic Diagnostics Act of 31 July 2009 (BGBl. I pp. 2529, 3672), strong consensus
The causes of pain in chronic pancreatitis are multifactorial and their pathophysiology is not fully understood. Pain can be related to structural changes in the pancreas, but also neuropathic.	Level of evidence: 5, strong consensus
Diagnostic workup (function tests, imaging, classification) in chronic pancreatitis	
Exocrine pancreatic function and morphological signs of chronic pancreatitis are not necessarily correlated; thus, both morphology examination and function testing should be used to establish the diagnosis.	Level of evidence: 1, strong consensus
A noninvasive pancreatic function test should be performed to detect pancreatic exocrine insufficiency. Suitable analyses include testing for fecal elastase 1 (with specific antibodies) and a breath test with 13C-marked lipids.	Level of evidence: 1, strong consensus
The exocrine pancreatic function ought to be evaluated at the time of diagnosis of the disease. In addition, exocrine pancreatic function testing should be performed over the course of the disease, if, after initially normal pancreatic function tests, changes in clinical signs and symptoms are noted which could be associated with exocrine pancreatic insufficiency.	Level of evidence: 4, strong consensus
In patients with diabetes, a noninvasive test of pancreatic exocrine function ought to be performed, if a patient develops clinical signs and symptoms of pancreatic exocrine insufficiency, e.g. unexplained weight loss.	Level of evidence: 5, strong consensus
Endoscopic ultrasound (EUS), CT and MRI are the imaging modalities with the highest sensitivity and specificity for establishing the diagnosis of chronic pancreatitis. Given its non-invasive nature and widespread use, transabdominal ultrasound ought to be the basic examination modality to be initially used, despite its low accuracy. In case of inconclusive findings, an endoscopic ultrasound ought to be performed after the transabdominal ultrasound. CT and MRI provide additional information in case of inconclusive findings.	Level of evidence: 2, consensus

Recommendation	Level of evidence, consensus
Endoscopic ultrasound is the diagnostic method of choice to diagnose chronic pancreatitis in an early stage with high sensitivity. Over the course of chronic pancreatitis, endoscopic ultrasound should be used for the detection and classification of complications (necrosis, pseudocysts) and especially for the management of significant pancreatic pseudocysts or necrosis.	Level of evidence: 2, strong consensus
Morphological changes in chronic pancreatitis ought to be classified using the Cambridge classification, adapted to the respective imaging modality.	Clinical consensus point, strong consensus
The Chronic Pancreatitis Prognosis Score (COPPS) ought to be used to estimate the severity of the short- and medium-term clinical course of chronic pancreatitis.	Level of evidence: 2, consensus
Pharmacotherapy of chronic pancreatitis	
The same principles that apply to the treatment of acute pancreatitis also apply to the treatment of acute attacks in patients with chronic pancreatitis. Thus, for detailed recommendations please refer to the corresponding section of the Guideline on Acute Pancreatitis.	Strong consensus
Pain management in chronic pancreatitis may be provided based on the WHO analgesic ladder.	Level of evidence: 5, strong consensus
The decision on the duration of an attempt to pharmacologically control pain in chronic pancreatitis may be made on a case-by-case basis. However, if these attempts fail, regular re-evaluations ought to be performed and endoscopic or surgical procedures added to the pain management plan, if required.	Level of evidence: 5, strong consensus
Weaning from pain medications may be undertaken by following the steps of the WHO Pain Ladder in reverse order.	Level of evidence: 5, strong consensus
Octreotide should not be used to manage pain in chronic pancreatitis.	Level of evidence: 1b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Pregabalin may be used in addition to other pain medications on a trial basis.	Level of evidence: 2, strong consensus
In general, pancreatic enzymes ought not to be used for pain management in chronic pancreatitis.	Level of evidence: 1, consensus
The use of pancreatic enzymes on a trial basis may be useful if there is evidence that malabsorption is causing the abdominal pain.	Level of evidence: 1, strong consensus
Currently, no recommendation can be made with regard to the use of antioxidants for pain management in chronic pancreatitis.	Level of evidence: 2, strong consensus
Montelukast ought not to be used for pain management in chronic pancreatitis.	Level of evidence: 2, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Pancreatin should be supplemented in patients with or with suspected significant steatorrhea (diagnostic test: fecal fat > 15 g/d (if available) or abnormal fecal fat excretion or abnormal pancreatic function tests in combination with clinical signs of malabsorption).	Level of evidence: 1, consensus, adopted from the 2012 guideline, reviewed in 2021
Even in the presence of minor abnormal fecal fat excretion (7–15 g/d), pancreatin should be replaced if signs of malassimilation are noted (e.g., weight loss) or if the patient experiences abdominal symptoms attributable to maldigestion and malabsorption.	Level of evidence: 1, consensus, adopted from the 2012 guideline, reviewed in 2021
The goals of replacement therapy should be to reduce malabsorption with adequate oral nutrition (all major nutrient groups and vitamins) and effectively treat abdominal symptoms, if required. Complete normalization of nutrient digestion and absorption is rarely achievable.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
The success of pancreatic enzyme (pancreatin) replacement therapy ought to be primarily confirmed based clinical parameters (weight gain, long-term normalization of vitamin status, disappearance of abdominal symptoms).	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
In case of clinical doubts as to whether symptom persistence can be explained by a lack of effectiveness of enzyme replacement therapy, fecal fat excretion or pancreatic function tests ought to be used to measure nutrient digestion while the patient is on replacement therapy (e.g., breath tests with 13C-marked lipids).	Level of evidence: 2b, consensus, adopted from the 2012 guideline, reviewed in 2021

Recommendation	Level of evidence, consensus
Pancreatin should be taken with meals.	Level of evidence: 1b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Given the fact that pancreatic enzymes are acid sensitive, acid-protected preparations should be used in patients with intact gastric acid secretion.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Since mixing of chyme and pancreatin is required for optimum effectiveness, drugs containing acid-protected particles with diameters ≤ 2 mm ought to be chosen.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
The pancreatin dose given should offer sufficient enzymatic activity for digesting a meal.	Level of evidence: 1b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Dosing of pancreatin preparations is based on lipase activity. Per main meal, 40 000 to 50 000 units Ph. Eur. ought to be given as a starting dose, for digesting smaller snacks between meals about half of this dose.	Level of evidence: 1b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
In case of inadequate effectiveness, the enzyme dose ought to be doubled or, if required, tripled.	Level of evidence: 5, strong consensus
If effectiveness continues to be inadequate, pancreatin powder or granules ought to be combined with an acid inhibitor.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
If this strategy does not achieve the desired treatment success, another cause of the persisting symptoms ought to be searched.	Clinical consensus point, consensus, adopted from the 2012 guideline, reviewed in 2021
Almost all pancreatic enzyme preparations available in Germany contain porcine pancreatin. As a medicine, these can often be taken by patients who would otherwise refuse to consume preparations of porcine origin (for religious or ethical reasons).	Level of evidence: 5, strong consensus
When pancreatic enzyme preparations are given, attention ought to be paid to potential adverse reactions, such as abdominal symptoms (in <10% abdominal pain, altered bowel motion, nausea/vomiting) or allergic reactions (in <1% of patients).	Level of evidence: 3b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Very high enzyme doses (>10 000–20 000 units lipase per kg body weight per day) ought to be avoided, if possible.	Level of evidence: 5, strong consensus
In patients with diabetes mellitus and newly started or increased pancreatin treatment, blood glucose levels ought to be temporarily more closely monitored, because the enhanced digestion of nutrients may lead to changes in blood glucose regulation.	Level of evidence: 2, strong consensus
Pancreatic exocrine insufficiency is not the only reason for malnutrition in patients with chronic pancreatitis; pain-related reduction in food intake or continued alcohol consumption may also cause or aggravate malnutrition. In addition, the basal metabolic rate is increased in some patients.	Level of evidence: 3b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Patients with chronic pancreatitis and overt pancreatic exocrine insufficiency (weight loss, malnutrition) ought to receive pancreatic enzyme replacement therapy along with a personalized nutritional intervention by a nutritionist to prevent or stop deterioration of nutritional status in a targeted manner.	Level of evidence: 2, consensus
The nutritional intervention should be aimed at providing an adequate supply of nutrients, vitamins and trace elements as well as adequately meeting the individual daily energy requirements to avoid catabolism.	Level of evidence: 5, strong consensus, adopted from the 2012 guideline
As a rule, patients with chronic pancreatitis and overt pancreatic exocrine insufficiency ought to be treated with a standard isocaloric diet and adequate pancreatic enzyme replacement therapy. For a better response, dietary intake can be divided into 4–6 (proportionally smaller) meals.	Level of evidence: 2, strong consensus, adopted from the 2012 guideline, reviewed in 2021
A low-fat diet cannot (generally) be recommended. Only if with progressing pancreatic exocrine insufficiency patients develop bothersome clinical signs of fat maldigestion despite adequate oral enzyme replacement, the amount of fat consumed orally may be reduced as tolerated.	Level of evidence: 5, strong consensus, adopted from the 2012 guideline, reviewed in 2021

Recommendation	Level of evidence, consensus
Medium-chain triglyceride do not require lipase to be absorbed and thus can improve fat absorption in patients with exocrine insufficiency who are not receiving enzyme replacement therapy. Medium-chain triglycerides ought not to be recommended to patients on enzyme replacement therapy.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Additional nutritional approaches (oral, enteral or parenteral) may be necessary in patients with advanced pancreatic exocrine insufficiency.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
As a rule, patients with chronic pancreatitis should refrain from alcohol consumption.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021, NKLM
Any deficiency in vitamins or trace elements should be replaced in a targeted manner.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021, NKLM
In adults, the indication for replacement of vitamins and trace elements ought to be established primarily based on clinical signs of deficiency. Additional measuring of serum concentrations ought to be performed on a case-by-case basis only.	Level of evidence: 5, strong consensus
In pediatric patients, the indication for replacement therapy ought to be established on a liberal basis and before clinical signs of deficiency occur.	Level of evidence: 5, strong consensus
Endoscopic and interventional therapy	
Painful chronic pancreatitis should not be treated with opioids for more than six months before primary surgical treatment is recommended to patients with a considerably dilated pancreatic duct. If an alternative endoscopic therapy of no more than three months does not result in the patient being pain-free, a surgical procedure should again be recommended.	Level of evidence: 1, strong consensus
If a resectable pancreatic cancer is suspected, the patient should undergo surgical treatment. An endoscopic ultrasound-guided aspiration biopsy may be obtained from atypical lesions.	Level of evidence: 2, strong consensus
The presence of pancreatic endocrine insufficiency or of a combination of endocrine and exocrine insufficiency is by itself not an indication for surgical or interventional treatment of chronic pancreatitis.	Levels of evidence: 1–4, strong consensus
If clinical symptoms of chronic pancreatitis-related gastric outlet or duodenal stenosis persist, the patient should undergo surgical or, in case of unresectability, interventional treatment.	Clinical consensus point, strong consensus
Chronic pancreatitis patients with benign biliary stenosis or biliary dilatation without pain ought to receive endoscopic or surgical treatment.	Level of evidence: 2, consensus
If a pancreatic pseudocyst causes complications, the patient ought to receive interventional or surgical treatment.	Level of evidence: 2, strong consensus
First-line treatment of symptomatic pancreatic pseudocysts ought to be endoscopic drainage of the pseudocysts; in case of pseudocyst recurrence, surgical treatment may be considered.	Level of evidence: 3, consensus
The decision whether to perform endoscopic or surgical pseudocyst drainage ought to be made taking into account the location of the pseudocyst and the type of additional pathomorphological changes.	Level of evidence: 3, strong consensus
Asymptomatic pancreatic pseudocysts that measure more than 5 cm in diameter and do not resolve within six weeks may be treated.	Level of evidence: 2, consensus, adopted from the 2012 guideline, reviewed in 2021
Prior to draining a pseudocyst, MRI with MRCP may be obtained to be able to assess the morphology of the pancreatic duct (connection to the cyst, rupture).	Level of evidence: 4, consensus
Endoscopic drainage of pseudocysts may be performed via a transgastric or transduodenal approach. In addition, there is the option of percutaneous drainage; however, this approach is associated with risk of external fistula formation and thus ought not to be chosen as the first-line treatment.	Level of evidence: 4, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Transpapillary drainage of pseudocysts may be used for cysts <5 cm in diameter that communicate with the pancreatic duct and are located in the pancreatic head/body.	Level of evidence: 4, consensus

Recommendation	Level of evidence, consensus
Transmural endoscopic needle aspiration ought to be performed under endosonographic guidance.	Level of evidence: 3, strong consensus
Double-pigtail drains ought to be removed not earlier than 6 weeks after resolution of the pseudocyst. Exclusion of a rupture of the pancreatic duct may be undertaken.	Level of evidence: 2, strong consensus
Diagnostic aspiration biopsy of a cystic pancreatic lesion in patients with chronic pancreatitis ought to be performed, if a pre-malignant cystic neoplasia is suspected and may be used to diagnose infected cyst content.	Level of evidence: 4, consensus
If a malignant cystic lesion is suspected, a surgical approach should be chosen.	Level of evidence: 4, strong consensus
In patients with chronic pancreatitis with advanced pancreatic duct changes, especially with pancreatolithiasis, a pseudocyst ought to be treated as a part of an overall therapeutic strategy.	Level of evidence: 2b, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Pancreatic duct stenosis may be treated in the presence of a pancreatic pseudocyst, prestenotic duct dilatation or fistula.	Level of evidence: 4, strong consensus, adopted from the 2012 guideline, reviewed in 2021
If pancreatic pseudocysts are suspected or represent the indication for performing a, ERCP, peri-interventional antibiotic therapy should be performed.	Level of evidence: 4, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Vascular pseudoaneurysms ought to be treated in patients with chronic pancreatitis.	strong consensus, Clinical consensus point
Bleeding pancreatic pseudoaneurysms ought to be treated with angiographic techniques.	Level of evidence: 3, strong consensus
Pancreatic duct stones causing pain by obstructing the outflow of pancreatic secretions, inducing recurrent attacks, maintaining a pseudocyst or fistula, or bringing about other complications, ought to be treated. In the long term, surgical treatment is superior to endoscopic treatment.	Level of evidence: 1, consensus
Pancreatic duct stenosis causing pain by obstructing the outflow of pancreatic secretions, inducing recurrent attacks, maintaining a pseudocyst or fistula, or bringing about other complications, ought to be treated. Endoscopic dilation and stenting may be performed as first-line treatment.. In the subgroup of patients with chronic pancreatitis and pain not previously treated with opioids and a pancreatic duct measuring >5 mm in diameter, first-line treatment ought to be surgical.	Level of evidence: 1, strong consensus
Endoscopic stenting of the pancreatic duct ought to be performed, if symptomatic pancreatic duct stones or a pancreatic duct stenosis close to the papilla of Vater are obstructing the outflow. No general recommendations can be made for the required duration of stent therapy after complete relief from pain has been achieved.	Level of evidence: 1, consensus
If surgical treatment is contraindicated, pain management may include the insertion of a fully covered metal stent into the pancreatic duct.	Level of evidence: 4, consensus
Isolated pancreatic duct stones causing pain by obstructing the outflow of pancreatic secretions, inducing recurrent attacks, maintaining a pseudocyst or fistula, or bringing about other complications, may be treated with ESWL, pancreatoscopy-guided electrohydraulic lithotripsy or laser lithotripsy.	Level of evidence: 2, consensus
In case of chronic pancreatitis-related distal biliary duct stenosis along with clinical signs of cholangitis, the stenosis ought to endoscopically drained within 24 hours.	Level of evidence: 4, strong consensus
In case of a chronic pancreatitis-related distal biliary duct stenosis with cholestasis or icterus for more than 4 weeks, endoscopic stenting or surgical treatment ought to be performed.	Level of evidence: 4, strong consensus
Endoscopic treatment of distal biliary duct stenosis ought to be performed by inserting several stents or one fcSEMS.	Level of evidence: 3, strong consensus
Endoscopic treatment of chronic pancreatitis-related distal biliary duct stenosis ought not to be performed for a period of more than 12 months. If plastic stents are used, the stents ought to be replaced at least every 6 months.	Level of evidence: 4, strong consensus
After a failed attempt of treating a chronic biliary duct stenosis endoscopically, the patient should receive surgical treatment.	Level of evidence: 1, strong consensus, adopted from the 2012 guideline

Recommendation	Level of evidence, consensus
If first-line surgical treatment of chronic pancreatitis with cholestatic jaundice is indicated, preoperative endoscopic stenting of the biliary duct ought to be performed only if (1) surgery cannot be performed immediately or (2) in the presence of cholangitis.	Level of evidence: 2, strong consensus, adopted from the 2012 guideline, reviewed in 2021
The more important it is to achieve a lasting treatment outcome, the longer the statistical and individual life expectancy of the patient, the better their general condition, and the lower the expected morbidity and mortality of the pancreas-resecting procedure, the more likely a surgical treatment approach ought to be chosen. The lower the statistical and individual life expectancy of the patient, the greater the comorbidity and the more challenging the expected technical difficulty of the surgical procedure (e.g., extensive collateral circulation due to portal hypertension), the more likely endoscopic treatment should be preferred over a pancreas-resecting procedure.	Clinical consensus point, strong consensus
Indication and surgical management in chronic pancreatitis	
Patients with pain requiring continuous analgesic treatment should undergo first-line surgical, or, in case of unresectability, interventional treatment.	Level of evidence: 1, strong consensus
In presence of preoperative cholestasis (imaging and/or laboratory findings), intraoperative internal drainage of the intrapancreatic biliary duct ought to be performed.	Level of evidence: 2, strong consensus
While all surgical options, including duodenum-preserving procedures, may be used in patients with chronic pancreatitis, first-line oncological pancreatic resection should be performed—subject to tumor location as a Kausch-Whipple procedure (classic or pylorus-preserving), a pancreatic left resection, or a total pancreatectomy.	Clinical consensus point, strong consensus
Patients with chronic pancreatitis should undergo surgery early, within a few months (<2–6 months) after initiation of opioid treatment for pain management.	Level of evidence: 2, consensus
In patients with portal hypertension and formation of venous collateral circulation due to long sections of occlusion of the portal-superior mesenteric veins, preference ought to be given to resection of the pancreatic head by means of a variant of the duodenum-preserving pancreatic head resection procedure, because of the great complexity of a Kausch-Whipple procedure in this situation.	Clinical consensus point, strong consensus
If an organ-preserving procedure is performed (e.g., a variant of the duodenum-preserving pancreatic head resection, DPPHR), intraoperative frozen-section analysis of the surgical resection specimen ought to be performed to rule out malignancy.	Level of evidence: 4, strong consensus
Surgical specimens of patients with chronic pancreatitis ought to undergo intensive pathological analysis (intensified tissue collection). The aim of this strategy is (1) to definitely rule out malignancy and (2) to obtain insights into the etiology.	Clinical consensus point, strong consensus
In case of cholestasis with duct dilation due to compression of the common hepatic duct (CHD), primary endoscopy and stenting of the biliary duct ought to be performed in the absence of other surgical indications (suspected malignancy, pain controllable with opioids, etc.). However, if symptoms or cholestasis persist after temporary endoscopic treatment (maximum duration: 12 months), surgical resection ought to be performed. In patients with calcifying pancreatitis, first-line surgical treatment is indicated.	Level of evidence: 2, strong consensus, NKLM
Resection of the pancreatic head is the standard surgical treatment for chronic pancreatitis with inflammatory pseudotumor of the pancreatic head. One of the variants of duodenum-preserving pancreatic head resection techniques (technique described by Beger, technique according to Frey or Berner modification) or the Kausch-Whipple procedure (in the classic or pylorus-preserving variant) should be performed.	Level of evidence: 1, strong consensus
In the absence of an inflammatory pancreatic head tumor and congested pancreatic duct, a modification of the duodenum-preserving pancreatic head resections or drainage surgery may be performed.	Level of evidence: 3, strong consensus
Treatment of chronic pancreatitis ought to be performed at centers with high procedure numbers.	Clinical consensus point, consensus
Monitoring and follow-up	
Once a patient is diagnosed with chronic pancreatitis, monitoring/follow-up ought to be recommended, since morbidity and mortality are found increased in patients with chronic pancreatitis compared to an age-adjusted control group.	Level of evidence: 1, consensus

Recommendation	Level of evidence, consensus
If signs of impaired bone metabolism or reduced nutritional status are noted, the exocrine function should be re-assessed.	Level of evidence: 2, consensus
Follow-up endocrine function testing should be performed as a parameter to monitor disease progression. For this purpose, HbA1c/fasting blood glucose should be measured.	Level of evidence: 1, strong consensus
If a patient with chronic pancreatitis experiences unintended weight loss or develops B symptoms, icterus or diabetes mellitus, imaging studies should be obtained to rule out pancreatic cancer. An attack of acute pancreatitis should also be investigated with diagnostic imaging to exclude complications that would require treatment.	Level of evidence: 1, strong consensus
After a surgical or interventional treatment, a one-time follow-up to evaluate treatment success and exclude complications ought to be performed.	Level of evidence: 1, consensus
In patients with chronic pancreatitis, endocrine pancreatic function should be followed-up annually by measuring HbA1c/ fasting glucose levels, because the long-term prognosis of patients who develop diabetes is poorer due to an increased comorbidity risk.	Level of evidence: 1, strong consensus
Follow-up care for patient with uncomplicated chronic pancreatitis ought to be provided by primary-care providing specialists in internal medicine, general practitioners and gastroenterologists. In case of complications, specialists in visceral medicine (gastroenterologists and visceral surgeons) ought to get involved in the management of the patient. Depending on the disease-related comorbidities, other specialties (psychologists, endocrinologists, (medical) nutritionists, pain therapists, radiologists) ought to get involved as well. Surgical or interventional procedures ought to be preceded by a discussion of the case in a broad interdisciplinary board, ideally in a pancreatic center.	Level of evidence: 2, strong consensus
Patients developing complications of chronic pancreatitis which require surgical or interventional treatment ought to be referred to a pancreatic center.	Level of evidence: 2, consensus
A behavioral intervention ought to be recommended to patients with chronic pancreatitis having problems with addiction (tobacco, alcohol, other drugs).	Level of evidence: 5, consensus
Prophylactic total pancreatectomy as a measure of cancer prevention in high-risk groups ought not to be performed in patients with chronic pancreatitis.	Level of evidence: 2, strong consensus
Patients with hereditary pancreatitis ought to undergo annual clinical, imaging and laboratory follow-up examinations from age 40 years onward or 20 years after symptom onset in the pancreatic center.	Level of evidence: 3, strong consensus
Diagnosis and management of chronic pancreatitis in pediatric patients	
Diagnosis and management of chronic pancreatitis in children and adolescents ought to be provided under the direction of a pediatric gastroenterologist in collaboration with an experienced pediatric surgeon or visceral surgeon, pediatric radiologist, and, if required, interventional endoscopist.	Level of evidence: 4, consensus
In children and adolescents, diffuse and persistent upper abdominal pain that responds poorly to standard analgesic therapy ought to raise differential diagnostic considerations about the presence of pancreatitis.	Level of evidence: 3, strong consensus
The causes of pancreatitis in children and adolescents should be fully investigated.	Level of evidence: 2, strong consensus
Complications of acute and chronic pancreatitis in children and adolescents are the same as in adults. Children and adolescents rarely develop exocrine or endocrine pancreatic insufficiency before they reach adulthood.	Level of evidence: 2, strong consensus
In children and adolescents with suspected pancreatitis, serum lipase levels should be measured.	Level of evidence: 2, strong consensus
Abdominal ultrasound should be used as the initial imaging modality in children and adolescents with suspected pancreatitis.	Level of evidence: 2, strong consensus
In children and adolescents with an acute attack of pancreatitis, abdominal computed tomography should only be obtained in case of progressive clinical deterioration.	Level of evidence: 3, strong consensus
In children, MRCP ought to be used as the imaging modality of choice to visualize the bile ducts and the pancreatic duct.	Level of evidence: 3, grade of recommendation A, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Endoscopic ultrasound may be performed in pediatric patients.	Level of evidence: 4, strong consensus

Recommendation	Level of evidence, consensus
Pursuant to the German Genetic Diagnostics Act (GenDG), genetic testing may only be performed after documented informed consent discussion and obtaining a written consent.Clinical testing of minor relatives with no signs of the disease (predictive genetic testing) is prohibited pursuant to the Genetic Diagnostics Act. Patients with a positive family history of pancreatitis and patients with idiopathic chronic pancreatitis or two or more attacks of idiopathic acute pancreatitis should undergo genetic testing.	Level of evidence: 5, strong consensus
In pediatric patients, the differential diagnostic work-up of chronic pancreatitis should include sweat testing to rule out cystic fibrosis.	Level of evidence: 1, strong consensus, adopted from the 2012 guideline, reviewed in 2021
Intravenous fluid replacement and analgesia should be initiated as a supportive measure.	Level of evidence: 2, strong consensus
The indications for ERCP in pancreatitis are the same for children, adolescents and adults.	Level of evidence: 3, strong consensus
Children and adolescents with chronic pancreatitis may be treated surgically if conservative and/or endoscopic treatment over several months has been unsuccessful, e.g. persistent pain and/or progressive organ destruction.	Level of evidence: 3, strong consensus
The management of complications of acute pancreatitis ought to be performed using the same criteria as in adults.	Clinical consensus point, strong consensus
Studies on specific pain management in pediatric patients with chronic pancreatitis are not available. As in adults, pain management ought to be provided in accordance with the WHO Pain Ladder.	Level of evidence: 5, strong consensus
In children with an acute mild episode of pancreatitis, early initiation of oral-enteral nutrition (< 48 h) ought to be pursued.	Level of evidence: 3, strong consensus
In children and adolescents with an acute severe episode of pancreatitis, initiation of enteral nutrition < 72 h may be pursued.	Level of evidence: 4, strong consensus
The general use of antibiotics in children and adolescents with an acute episode of pancreatitis cannot be recommended.	Level of evidence: 5, strong consensus
Children and adolescents with chronic pancreatitis and pancreatic exocrine insufficiency should receive pancreatic enzyme replacement therapy.	Level of evidence: 2, strong consensus
In children and adolescents with chronic pancreatitis, it should be followed up on a routine basis whether weight and length development are in line with the respective percentiles.	Level of evidence: 4, strong consensus
In children and adolescents with chronic pancreatitis and pancreatic exocrine insufficiency, serum levels of the fat-soluble vitamins A, E and D as well as INR ought to checked annually.	Level of evidence: 3, strong consensus
In children and adolescents with chronic pancreatitis and evidence of pancreatic exocrine insufficiency, annual checks of serum zinc and selenium levels may be recommended.	Level of evidence: 4, strong consensus
Autoimmune pancreatitis	
Autoimmune pancreatitis (AiP) is defined as “a distinct form of pancreatitis characterized by imaging morphology and histological features of the pancreas, associated with extra-pancreatic manifestations, which are part of an IgG4-associated disease or other diseases, and by the fact that the signs and symptoms of AiP almost always resolve with steroid medication”.	strong consensus
Histologically, two types of AiP are distinguished based on histological and clinical criteria (type 1, lymphoplasmacytic sclerosing pancreatitis [LPSP]; type 2, idiopathic duct-centric pancreatitis [IDCP]).In some patient, however, it is no possible to conclusively class them as one or the other of these two forms. Classification should be based on the International Consensus Diagnostic Criteria (ICDC).	Level of evidence: 2, strong consensus
Autoimmune pancreatitis should be diagnosed according to the International Consensus Diagnostic Criteria (ICDC) which reflect five major criteria (imaging, serology, histology, other organ involvement, response to systemic steroid medication).	Level of evidence: 2, strong consensus
Imaging morphology criteria for AiP include focal enlargement of a section of the pancreas or diffuse enlargement of pancreatic parenchyma with typical changes in contrast distribution and changes of the pancreatic duct (e.g., solitary or multiple strictures without upstream duct dilation). Other characteristic parenchymal and ductal changes in imaging morphology may be present in patients with AiP. MRI with contrast and/or endoscopic ultrasound ought to be primarily used to detect these changes.	Level of evidence: 2, consensus

Recommendation	Level of evidence, consensus
The definite diagnosis of type 1 AiP can be established based on the presence of clearly defined clinical features or a combination of these features together with response to steroid medication. If type 2 AiP is suspected, histological confirmation ought to be obtained.	Level of evidence: 2, strong consensus
All symptomatic AiP patients ought to be treated with corticosteroids. Given the high recurrence rate, patients with type 1 AiP ought to receive low-dose corticosteroid maintenance therapy.	Level of evidence: 2, strong consensus
In case of treatment failure or contraindications to corticosteroid treatment, patients with type 1 AiP may be treated with anti-CD20 antibodies.	Level of evidence: 3, strong consensus
The response to immunosuppressive treatment should be evaluated based on imaging and laboratory findings four to six weeks after start of treatment. In case of non-response to treatment, pancreatic cancer as a differential diagnosis should be ruled out again.	Level of evidence: 3, strong consensus
A surgical intervention should be performed in patients with AiP, if is not possible to rule out pancreatic cancer or biliary cancer.	Level of evidence: 1
A surgical intervention should also be performed in patients with AiP, if complications of chronic pancreatitis are present which did not respond to immunosuppressive therapy and require surgical treatment.	Level of evidence: 3, strong consensus
Long-term follow-up care for patients with AIP ought to be provided in keeping with the recommendation for chronic pancreatitis.	Consensus
In patients with type 1 AiP, screening for extrapancreatic manifestations of IgG4-associated disease ought to be performed.	Level of evidence: 3, consensus

eTABLE 2

Results of the literature search

Working groups	Articles in the screening	Assessed full-text articles
WG 1 AP	306	52
WG 2 AP	509	59
WG 3 AP	677	82
WG 4 AP	1051	98
WG 5 AP	464	144
WG 6 AP	1042	36
WG 7 AP	697	55
WG 8 AP	614	37
WG 1 CP	670	81
WG 2 CP	1778	24
WG 3 CP	211	31
WG 4 CP	899	84
WG 5 CP	380	66
WG 6 CP	196	15
WG 7 CP	528	40
WG 8 CP	646	190

WG, working group; AP, acute pancreatitis; CP, chronic pancreatitis

eTABLE 3

Rosemont classification for EUS-based diagnosis of chronic pancreatitis, adapted from (e1)

Reliability of the diagnosis	Required criteria
I. In agreement with chronic pancreatitis	A: 1 × A major criterion + ≥ 3 minor criteria B: 1 × A major criterion + B major criterion C: 2 × A major criterion
II. Suspicious of chronic pancreatitis	A: 1 × A major criterion + < 3 minor criteria B: B major criterion + ≥ 3 minor criteria C: ≥ 5 minor criteria
III. Chronic pancreatitis possible	A: 3 to 4 × minor criterion without major criterion B: B major criterion + alone or < 3 minor criteria
IV. Normal	≤ 2 minor criteria, no major criteria

Criterion	Definition	Major criterion	Minor criterion	Histological correlate
Hyperechoic foci with shadowing	Echogenic structures ≥ 2 mm (length x width) with shadowing	A	–	Parenchymal calcifications
Main pancreatic duct calcification	Echogenic structures within pancreatic duct with dorsal shadowing	A	–	Pancreatolithiasis
Lobularity	Well-circumscribed, ≥ 5 mm structures with hyperechoic rims and hypoechoic central structure			Unknown
A with honeycombing	Contiguous across ≥ 3 lobules	B		
B without honeycombing	Non-contiguous		Yes	
Hyperechoic foci without shadowing	Echogenic structures ≥ 2 mm (length x width) without shadowing		Yes	Unknown
Cysts	Anechoic, rounded or elliptic structures with or without stranding		Yes	Pseudocysts
Stranding	Hyperechoic lines ≥ 3 mm in length and in at least 2 different directions with respect to the imaged plane		Yes	Unknown
Irregular main pancreatic duct	Uneven or irregular contour of the duct with or without ectasia		Yes	Unknown
Dilated side branches	≥ 3 tubular anechoic structures with ≥ 1 mm in diameter, communicating with the main pancreatic duct		Yes	Side branch dilatation
Main pancreatic duct dilatation	≥ 3.5 mm in the pancreatic body or ≥ 1.5 mm in the tail		Yes	Main pancreatic duct dilatation
Hyperechoic main pancreatic duct	Echogenic, well-defined structure, occupying more than 50% of the diameter of the duct in the body and tail of the pancreas		Yes	Main pancreatic duct sclerosis

EUS, endoscopic ultrasound