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UNDERSTANDING THE DISEASE

Ten tips to manage severe acute pancreatitis in an intensive care unit

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The prevalence of acute pancreatitis has continuously increased over the past decades. Although the majority of patients with acute pancreatitis presents with mild, interstitial oedematous pancreatitis and fully recovers with supportive treatment within a few days, more than 10% develop a more severe course requiring hospitalisation in an intensive care unit (ICU) [1]. This article gives a quick guidance on how to approach these patients (Fig. 1).

Identify those patients who are at increased risk of developing a severe course

Most of the patient-related risk factors, laboratory parameters and scoring systems used to predict development of severe course show a high negative but a low positive predictive value and no prognostic score predicts severe disease with sufficient reliability [2]. The criteria to diagnose a systemic inflammatory response syndrome (SIRS) may be as accurate as other, more sophisticated scores, to predict severe course and the absence of SIRS on day 1 is associated with a high negative predictive value. An easily applicable score is the bedside index of severity of acute pancreatitis score. A score of ≥ 3 is associated with a substantially increased mortality [3]. Especially during the initial phase (first week), acute pancreatitis is a highly dynamic disease and predictive parameters should be re-evaluated on a daily basis.

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Admit patients who develop cardiovascular, respiratory or renal failure with or without infected pancreatic necrosis to an ICU

The severity of acute pancreatitis is determined by the development of organ failure(s) and local complications and classified according to the Revised Atlanta Classification or the Determinant-Based Classification [1]. Organ failure persisting > 48 h and/or infected (peri-)pancreatic necrosis define the severe or critical forms, which are associated with a mortality ranging from 39 to 54% [1]. Given the high rates of complications and mortality, patients with severe or critical acute pancreatitis should be treated in an ICU.

Perform additional examinations to identify the etiology

The most common causes of acute pancreatitis are alcohol abuse and biliary obstruction, each accounting for about 40%. Rarer etiologies comprise dyslipidemia, hypercalcemia or drugs. Thus, initial work-up includes aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyltransferase (GGT), alkaline phosphatase (ALP) and bilirubin and abdominal ultrasound to confirm or exclude biliary etiology. In addition, serum calcium and triglyceride concentrations should be determined. Hypertriglyceridemia can be considered to be the underlying etiology if serum triglyceride concentration exceeds 11.3 mmol/L or 1000 mg/dL [4]. In up to 30% of patients, no etiology of acute pancreatitis can be established necessitating complementary testing after stabilisation of the patient. This includes endoscopic ultrasound and/or magnetic resonance imaging (MRI)/magnetic resonance cholangiopancreatography (MRCP). Endoscopic ultrasound has a higher diagnostic accuracy than MRCP in the etiologic diagnosis of biliary disease, whereas secretin stimulated MRCP is superior in diagnosing pancreatic divisum [5].

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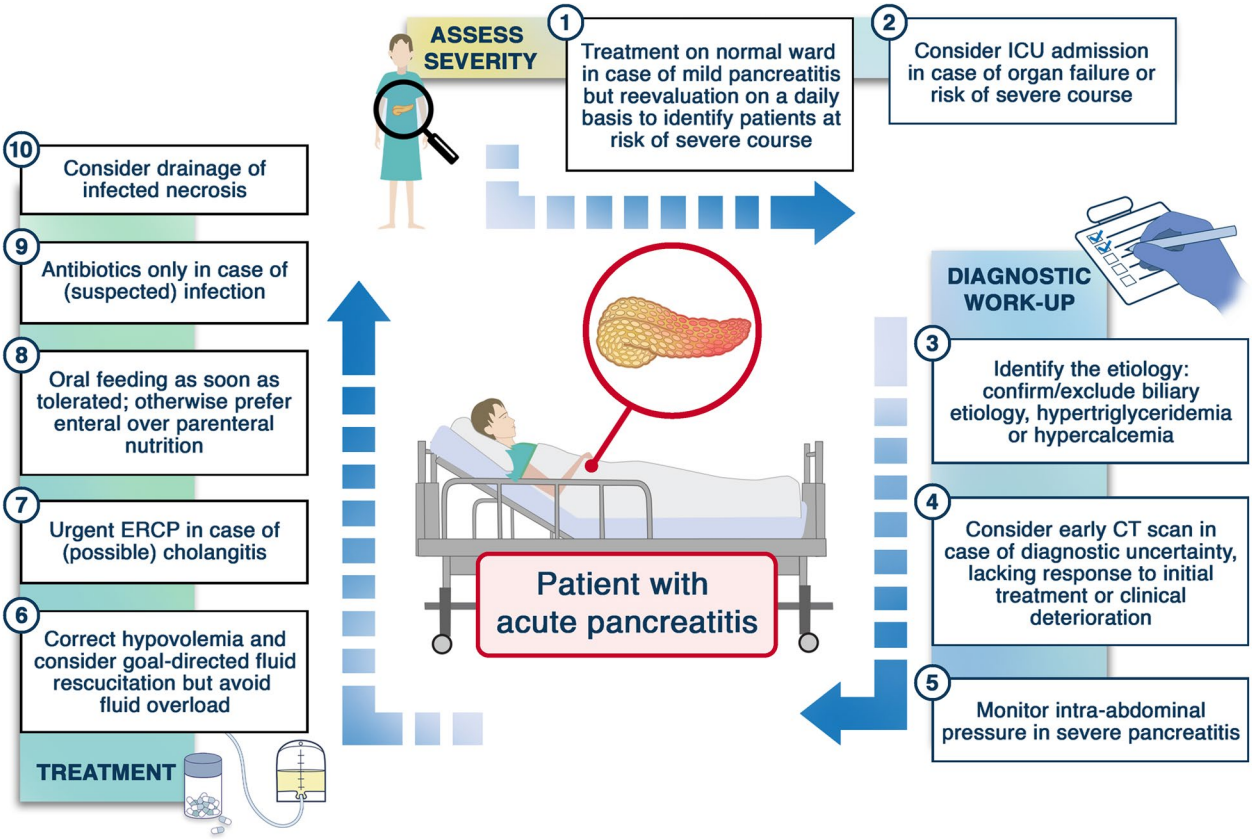

Fig. 1 Diagnostic work-up, allocation and treatment of a patient with acute pancreatitis

Consider abdominal computed tomography scan in the early course to exclude differential diagnoses

Early abdominal computed tomography (CT) scan within the first days after symptom onset is unreliable to detect the degree of necrosis or the presence of complications and, therefore, rarely modifies diagnosis and treatment [6]. Nevertheless, in patients admitted to the ICU, an abdominal CT scan should be carried out in case of diagnostic uncertainty, lacking response to initial treatment or clinical deterioration.

Monitor intra-abdominal pressure via measuring bladder pressure to prevent abdominal compartment syndrome in patients with severe acute pancreatitis

Intra-abdominal hypertension, defined by a sustained intra-abdominal pressure (IAP) $\geq$ 12 mmHg, occurs in the majority of patients with severe course and is associated with organ failure and increased mortality [7]. If IAP increases above 20 mmHg or abdominal compartment syndrome (i.e. IAP > 20 mmHg and new onset organ failure) develops, digestive tract suction, drainage of peritoneal effusion, limitation of fluid load, deepened sedation and, in some cases, neuromuscular blockade can be considered [4].

Correct initial hypovolemia but avoid routine infusion of large amounts of fluids

As shown by a recent trial, aggressive fluid resuscitation (bolus of 20 ml/kg of body weight, followed by 3 ml/kg/hour) is associated with an increased rate of fluid overload compared to moderate fluid resuscitation (bolus of 10 ml/kg in patients with hypovolemia or no bolus in patients with normovolemia, followed by 1.5 ml/kg/h) [8]. Considering the detrimental effects of fluid overload, contributing to respiratory failure and abdominal hypertension, fluid resuscitation should be goal-directed. To avoid fluid overload, early use of vasopressors may be necessary. Parameters that can guide volume therapy comprise haematocrit, blood urea nitrogen, creatinine, lactate, heart rate, blood pressure and persistence or resolution of organ failure or SIRS criteria. Regarding the type of fluids administered, balanced crystalloids should be preferred against normal saline and hydroxyethyl starch should be avoided [9].

Perform urgent endoscopic retrograde cholangiopancreatography as soon as possible in case of cholangitis

Early endoscopic retrograde cholangiopancreatography (ERCP) within 24 hours from the time of hospitalization dramatically reduces mortality in patients with cholangitis [10]. In contrast, it does not reduce mortality and local or systemic complications in patients with (predicted) severe acute biliary pancreatitis without biliary obstruction or cholangitis. Urgent ERCP with sphincterectomy did also not reduce major complications or mortality in the recent APEC trial in patients with (predicted) severe acute biliary pancreatitis with cholestasis but without cholangitis [11]. Nevertheless, in doubt patients should undergo ERCP as soon as possible given the challenges in the diagnosis of cholangitis. Early transfer to an expert ERCP centre should be considered.

Feed patients with severe acute pancreatitis orally as soon as tolerated. If oral feeding is not possible, prefer enteral nutrition over parenteral nutrition

A significant proportion of patients with severe acute pancreatitis tolerates oral feeding within 72 hours of symptom onset. Oral diet does not increase the risk of infection, organ failure or mortality compared to early enteral feeding (< 24 hours) [12] and hunger-based feeding is associated with shorter length of hospitalisation and fasting duration compared to conventional feeding [13].

Numerous studies have highlighted that enteral nutrition as compared to parenteral nutrition reduces mortality, infectious complications, organ failure and hospital length of stay in patients with acute pancreatitis. Nasogastral feeding is as effective as nasojejunal feeding. Nutrition should be started at low doses and progressively increased to a caloric target of 20–25 kcal/kg/day [14]. If enteral nutrition substantially increases intra-abdominal pressure, it must be reduced or even discontinued.

Do not use prophylactic antibiotics but treat (suspected) infections

There is no robust data that prophylactic antibiotics prevent superinfection of necrotic tissue, occurrence of extra-pancreatic infections including pneumonia and cholangitis or decreases mortality. Anti-infective treatment of infected necrosis should target resistant Enterobacteriaceae, *Enterococcus faecium* and *Pseudomonas aeruginosa*. Routine antimycotic prophylaxis is not recommended but anti-infective therapy must be adapted if fungal infection is detected [4].

Establish the diagnosis of infected necrosis by contrast-enhanced CT

As neither clinical signs nor markers of inflammation (e.g., C-reactive protein, procalcitonin) are sufficiently specific to differentiate inflammation caused by the pancreatitis per se from other infectious complications, diagnosis of infected necrosis is challenging. If infection of necrosis is suspected, contrast-enhanced abdominal CT scan should be performed. The presence of gas configuration within the necrosis is regarded pathognomonic but is only found in approximately half of infected necroses. Acute kidney injury represents no contraindication, if therapeutic consequences are expected from the CT scan. The optimal interventional management should be discussed interdisciplinary and drainage of infected necrosis can be considered, especially in septic patients. When necrotic collections are largely encapsulated, endoscopical drainage via transgastral or transduodenal approach, followed, if necessary, by endoscopic necrosectomy is an alternative to a radiology-guided percutaneous approach and surgical debridement [15].

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Declarations

Conflicts of interest
AF reports no potential conflict of interest relevant to this article. SJ reports receiving consulting fees from Drager, Medtronic, Mindray, Fresenius, Baxter, and Fisher & Paykel. MJ has received honoraria or research support from Baxter Healthcare Corp, AM-Pharma, CLS Behring, Fresenius, Takeda and Novartis.

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