Clinical Review of Acute, Recurrent, and Chronic Pancreatitis: Recent Updates of 2013–2019 Literature

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The increasing prevalence of pancreatic disorders worldwide has provided challenges in its clinical care and management. This review was aimed to evaluate recent literature on diagnosis, treatment, and management of acute pancreatitis (AP), recurrent acute pancreatitis (RAP), as well as chronic pancreatitis (CP) documented during the past 5–6 years. An extensive literature review was carried out based on studies within the last 6 years (2013–2019). Articles were selected based on updates and therapeutic management. Critical appraisal of literature was performed using the Mixed Methods Appraisal Tool (MMAT), and a PRISMA flowchart was used to avoid bias. The study identified recent updates on the prophylactic treatment in preventing RAP. The risk factors and the therapeutic management options were evaluated and discussed. The findings show that although many lifesaving new protocols are available for implementation in clinical practice, current literature lacks detailed and comprehensive guidelines that cover special populations and comorbidities. The literature evaluated showed that eight genes were involved in pancreatitis, CASR, CFTR, CLDN2, CPA1, CTRC, PRSS1, SBDS, and SPINK1, but the most common gene implicated was found to be CFTR, at 11%. Therefore, it is recommended that a comprehensive guideline should be formulated to facilitate the diagnosis, management, treatment, and prophylactic measures of pancreatic disease. This could in turn reduce disease complications and hospitalization time, and improve clinical practice for management of pancreatitis.

INTRODUCTION

The location of the pancreas reflects the typical pattern of pain that radiates to the back in patients with chronic and acute pancreatitis.[1] Pancreatitis is characterized as an inflammatory disease, and complications can extend to diabetes, infections, bleeding and renal insufficiency.[2] The incidence of pancreatitis is between 5 and 80 per 100,000 population, with the highest increase in incidence recorded in North America and New Zealand.[3] Pancreatitis remains to be a cause of hospitalization and requires multiple approaches in diagnosis and treatment. Understanding the severity, etiology, and underlying cause is critical for addressing the disease and determining the suitable approach for diagnosis, management, and treatment.[4] Although there are no laboratory markers that can record the course of the disease, new studies have emerged regarding early management of pancreatitis that shed some light on the...
Pancreatitis is generally categorized into three sections: acute pancreatitis (AP), recurrent acute pancreatitis (RAP), and chronic pancreatitis (CP). Studies have shown that AP can progress to RAP, then to CP in a continuous disease trajectory. Nevertheless, AP does not always progress to RAP, and RAP does not always progress to CP. This trajectory is largely influenced by multiple risk factors, including extrinsic factors such as smoking and alcohol use, and intrinsic factors such as autoimmune deficiencies and hereditary mutations.

The rapid and sudden nature of AP may lead to life-threatening complications with a mortality rate of 5%, and this number is further increased to alarming rates of 69%–80% in CP. Furthermore, a patient who presents to the emergency department with typical symptoms of AP, that is, abdominal pain, would be then administered standard pain medication, after which they may appear to be resting easily and contentedly. This leads to masking of further symptoms, and consequently, patients with AP tend to receive fragmented care as they may appear to be asymptomatic. These limitations found in current practice guidelines may have led to many shortcomings in clinical treatment of acute and chronic pancreatic disease, which could contribute to high morbidity and mortality rates among patients. Therefore, the objective of this review was to determine and evaluate pharmacotherapeutic updates of AP, RAP, and CP.

**Materials and Methods**

**Record selection and data extraction**

The reviewers conducted electronic searches in PubMed, UpToDate, and Google Scholar for studies published within the last 5–6 years. The search term used on PubMed was “Pancreatitis” [MeSH] OR “Pancreatitis, Acute” [MeSH] OR “Pancreatitis, Chronic” [MeSH], limiting English as the publication language. Results were reviewed for their actual relevance, and all potentially relevant studies were independently assessed for inclusion. Disagreements were resolved by consensus. Reviewers were not blinded to the journal, author, or institution of publication.

**Inclusion and exclusion criteria**

Inclusion criteria included journal articles published within the past 6 years (from 2013 onward) that discussed the etiology, treatment approaches to AP and CP, in addition to randomized controlled trials. From all the studies, eligible patients included those who were diagnosed with AP or CP. There were no limitations of age, race, and sex distribution. Exclusion criteria included all articles older than 6 years (older than 2013), and articles with studies and trials not conducted on humans. This is further illustrated in Figure 1.

**Quality assessment and data collection**

For quality appraisal of included studies, the Mixed Methods Appraisal Tool (MMAT) was used. Data extracted from each study included the first author’s name, year of publication, sample size, country, age, sex, study design, and outcome measures wherever applicable. Reference lists of original studies, narrative reviews, and previous systematic reviews were also examined. The content blueprint is presented in Figure 2.

**Results**

**General clinical data**

The 13 studies collected range from studies published in 2013–2019. All the studies shared similar information, such as etiology and management guidelines; however, there are apparent updates in the more recent studies. The earliest studies collected were dated 2013, and these studies provided recommendations on treatment, prophylaxis, and diagnosis. However, when compared to a study conducted in 2019, genetic risks became a prominent factor, where multiple genes, such as the presence of dominant PRSS1, was a strong indication for the presence of risk of the development of pancreatitis.

**Quality assessment of data**

The collected studies were screened for clear research questions and appropriate qualitative and quantitative approaches using the MMAT to respond to the screening questions. After careful evaluation, 14 studies were selected. The data collection methods in the studies chosen were found to adequately answer the research questions, and the data found addressed the objectives. The interpretation of the data was consistent with the treatment methods, and confounding factors were taken into consideration. The selected studies are shown in Table 1.

**Systematic review content blueprint**

To have a visual aid to serve as a guide for Table 1, a content blueprint was illustrated and is shown in Figure 2. The blueprint is divided into three main sections: AP, RAP, and CP. Each section is further divided into subsections. AP was divided into eight subsections: etiology, incidence, genetic variants, diagnosis, risk factors, management, complications, and guidelines. These eight subsections are linked to their corresponding articles organized in Table 1. However, it is important to note that the incidence subcategory was further broken down into two sections: adults...
and pediatrics. RAP was also divided into subsections genetic variants and risk factors, and then linked to their corresponding articles. Finally, CP was further categorized into etiology, genetic variants, risk factors, and management, and then further linked to their corresponding articles. Ultimately, the subcategories of each category were examined in close details in the Discussion section.

**DISCUSSION**

**Acute pancreatitis**

**Etiology**

A study published in 2019[11] discussed the different causes of AP and CP, where gallstones were seen to be the leading factor of AP, at 30%–50%.[11] Furthermore, gallstones are seen to occur more often in women than men, and therefore, women with gallstones are the predominant etiology for AP.[11] Genetic risk factors precipitate the onset of CP, along with alcohol abuse.[11] However, the studies found do not state whether age was a factor that influenced the onset of pancreatitis.[11] In addition to genetic risk factors, hypertriglyceridemia is shown to be the cause behind 10% of cases of AP, with an increased risk when combined with pregnancy.[11] Lastly, abdominal trauma is attributed to 20% of cases; however, the degree of force of trauma and the extent of damage to the pancreas have not been associated.[11] These data are further visually reflected in Figure 3. In terms of drug-induced causes, both AP and CP share common culprits, including azathioprine, statins, and oral contraceptives.[11]

**Risk factors**

Multiple risk factors are involved when it comes to AP. A study conducted in 2019[11] discussed genetic risks, where multiple genes, such as the presence of dominant PRSS1 mutations, have been discovered to
play a contributing factor to the development of AP. The presence of genetic factors can cause the patient to be at a higher risk of developing AP, especially when combined with other risk factors, such as alcohol abuse, smoking, and hypertriglyceridemia. In the study conducted by Magnusdottir et al., an analysis of patients with first time AP was done, and the findings concluded that patients with a history of alcoholism and smoking, especially in males, led to a higher risk of recurrence. Furthermore, mortality risk increases as patient age increases, especially among patients aged 60 or older, and the presence of comorbidities such as cancer, heart failure, and chronic kidney, liver disease, and obesity.

Although all articles share similar risk factors such as smoking, age, body weight, and alcohol abuse,
| Reference no. | Year  | Authors                        | Study design                  | Variables used                        | Conclusion                                                                 |
|--------------|-------|--------------------------------|-------------------------------|---------------------------------------|---------------------------------------------------------------------------|
| [2]          | 2013  | Wu B, Banks P                  | Review                        | Management of AP                      | Diagnosis, risk and prognostic factors, treatment, complications, recommendations from current practice guidelines |
| [4]          | 2018  | Garber A, Frakes C, Arora Z, et al. | Review                        | Mechanisms, treatment, and management of AP | Treatment relies heavily on fluid resuscitation and nutrition with advanced endoscopic techniques and cholecystectomy  |
| [5]          | 2017  | Stigliano S, Sternby H, de Madaria E | Review                        | Early management of AP                 | Analgesia, fluid resuscitation, antibiotics, nutrition, and endoscopic retrograde cholangiopancreatography  |
| [6]          | 2018  | Pham A, Forsmark C             | Review                        | CP etiology, management, and risk factors | The toxic-metabolic, idiopathic, genetic, autoimmune, recurrent and severe acute pancreatitis, obstructive [TIGAR-O] classification system categorizes known causes and factors that contribute to chronic pancreatitis  |
| [7]          | 2013  | Tenner S, Baillie J, DeWitt J, et al. | Review of guidelines       | AP management and guidelines          | Recommendations for the management of AP; routine use of prophylactic antibiotics not recommended  |
| [9]          | 2016  | Greenberg J, Hsu J, Bawazeer M, et al. | Guideline                    | Management of AP                      | Evidence-based recommendations for the management of mild and severe AP, complications of AP, and gallstone-induced pancreatitis  |
| [11]         | 2019  | Weiss FU, Laemmerhirt F, Lerch MM | Population-based cohort study | AP and CP etiology and risk factors   | Involves the pancreatic digestive protease/antiprotease system  |
| [12]         | 2018  | Shah AP, Mourad MM, Bramhall SR | Review                        | AP diagnosis and management           | Gallstones are the most common cause with epidemiological trends indicating a rising incidence, clinical guide outline provided  |
| [13]         | 2017  | Krishna S, Kamboj A, Hart P, et al. | Retrospective analytical study | Epidemiological trends of AP           | In the preceding decade, AP hospitalizations are increasing but associated mortality is declining; associated CP has emerged as a leading contributor for AP-related hospitalizations  |
| [14]         | 2018  | Abu-El-Haija M, El-Dika S, Hinton A, et al. | Case studies                 | AP admission trends among pediatric population | Admission of children for AP constitutes a significant healthcare burden, with a rising number of admissions with age  |
| [15]         | 2019  | Abu-El-Haija M, Valencia CA, Hornung L, et al. | Gene sequencing study    | Genetic variants in AP, RAP, and CP among pediatric population | Genetics have a significant role in progression to RAP and CP from the first attack of pancreatitis  |
| [16]         | 2019  | Magnusdottir BA, Baldursdottir MB, Kalaitzakis E, et al. | Population-based cohort study | Risk factors for CP and RAP           | RAP occurred in one-fifth of patients; development of CP was infrequent; both RAP and CP were related to alcoholic AP, whereas RAP was associated with smoking and male gender, and CP to RAP, organ failure, and local complications  |
| [17]         | 2019  | Kotagal M, Slusher J, Ahmed S, et al. | Retrospective study          | In-hospital and 90-day outcomes after total pancreatectomy with islet autotransplantation | TPIAT is an effective option when debilitating disease persists despite maximal medical and endoscopic therapy  |

AP = acute pancreatitis, CP = chronic pancreatitis, RAP = recurrent acute pancreatitis, TPIAT = total pancreatectomy with islet autotransplantation
every article places different importance on each risk factor. Wu and Banks\cite{2} took into consideration weight and comorbidities as well as age, whereas both Weiss et al.\cite{11} and Magnusdottir et al.\cite{16} focused on lifestyles with high risk, such as smoking and alcohol abuse. However, none focused on special populations, such as geriatrics and pregnant women.

**Incidence**

As illustrated in Figure 4, the trend of adults and children affected by AP and subsequently hospitalized has declined since 2009 in the United States. However, the decline of incidence in adults is a much steeper decline than that reported in children. This can be attributed to the recent surge in health education, and improvements in guidelines that resulted in a decline in hospitalization rates.\cite{13,15}

**Genetic variants**

A genetic risk of AP is not to be confused with hereditary pancreatitis, which is a rare autosomal dominant genetic disorder of the pancreas with incomplete penetrance. In a study conducted by Abu-El-Haija et al.,\cite{15} eight genes were detected by TruSeq enrichment and Illumina sequencing to be involved in pancreatitis: \textit{CASR}, \textit{CFTR}, \textit{CLDN2}, \textit{CPA1}, \textit{CTR}, \textit{PRSS1}, \textit{SBDS}, and \textit{SPINK1}. The most common gene implicated was found to be \textit{CFTR}, at 11%. The study findings were similar to others previously published, with similar patient populations.\cite{15}

**Diagnosis**

AP is a highly time-sensitive diagnosis and must be discovered immediately.\cite{12} According to a study published in 2018,\cite{12} the UK Working Party guidelines state that to accurately diagnose AP, the patient should be subjected to extensive study and examination.\cite{12} This is done to determine the exact underlying etiology in at least 80% of AP cases. The guidelines also state that “idiopathic” should only be cited as the etiology of 20% or less of these cases.\cite{12} Furthermore, the guidelines go on to discuss the importance of obtaining a complete patient’s history and clinician’s examination.
findings, in combination with laboratory findings, including amylase levels (the gold standard for the diagnosis) greater than the upper normal limit by three or four times, trypsinogen-activation peptide (TAP), and trypsinogen-2. On the other hand, an older study conducted in 2013 stated that diagnosis of AP is based simply on physical symptoms such as upper abdominal pain, serum levels of amylase or lipase ≥3 times the upper normal limit, and a confirmation of diagnosis by the use of cross-sectional imaging analysis, as in ultrasound. Other studies stated that endoscopic retrograde cholangiopancreatography (ERCP) can be used as a diagnostic tool to identify and treat pancreatitis.
Although both Wu and Banks\(^2\) and Shah et al.\(^{12}\) discussed the approaches to diagnosing AP, their tactics and methodology differ in detail. Whereas Wu and Banks\(^2\) took a more detailed approach by including a cautionary note for the analysis of amylase in cases of hyperlipidemia due to possible fluctuations, Shah et al.\(^{12}\) did not. On the other hand, the study conducted by Shah et al.\(^{12}\) attempted to place importance on inclusion of biomarkers that are more accurate, including TAP and trypsinogen-2. Seeing as there was a gap of 5 years between the two studies\(^{2,12}\) it can be said that updates were made and applied in the later study\(^{12}\) However, both studies\(^{2,12}\) focused on diagnosis of AP in adult patients, and do not venture into diagnosing special populations, such as children, pregnant women, or geriatric patients. Additionally, the diagnosis guidelines are not specific to gender.

**Management**

*Managing and treating AP within the critical first 24 h:*

The pathophysiology and etiology must be determined to establish treatment approaches for pancreatitis, as well as to prevent further complications. AP requires intensive supportive care, as complications could
occur within hours to days, and infection prevention measures should be applied whenever appropriate to avert further complications. The approach for treatment of AP differs from CP where the key is speed of treatment. AP has a rapid onset and treatment within the first 24 h is critical in the health of the patient.\textsuperscript{[2,4]}

Early management of AP is seen to improve patient outcomes and has shifted from invasive surgical measures to symptom-based treatment and prophylaxis. Strong evidence in the study conducted by Stigliano \textit{et al}.\textsuperscript{[5]} suggested that the most effective method of management includes nutrition support and prophylactic antibiotic use, as well as ERCP in severe cases.\textsuperscript{[5]} There have been studies\textsuperscript{[2,4,5]} where pain management and fluid resuscitation were also recommended; however, more studies are needed to investigate their effectiveness in early treatment of AP.

Once AP is diagnosed in the emergency room, management efforts are primarily supportive to mitigate and avoid further complications, via the use of severity scoring systems.\textsuperscript{[4]} Although there is no unanimity on the most effective method of treatment, goal-directed fluid resuscitation seems to be common among all, as illustrated in the studies conducted by Garber \textit{et al}.\textsuperscript{[8]} and Shah \textit{et al}.\textsuperscript{[8]} AP management strategies are similar to those of early AP management, including nutritional support and the use of prophylactic antibiotic therapy. The goals of medical management include providing intensive supportive care, limiting infection, and identification and treatment of complications. Treatment is largely supportive based on the pathophysiology and includes pain relief, fluid resuscitation, nutritional support, and inflammatory response management. Effective prophylactic antibiotic therapy is at times implemented to prevent infective complications. However, laparoscopic cholecystectomy

\textbf{Figure 8: Flowchart of management of chronic pancreatitis}
is recommended to those with mild acute gallstone pancreatitis, and the addition of bile duct exploration is advised in those with concurrent choledocholithiasis. Those with severe gallstone pancreatitis, cholangitis, and choledocholithiasis are advised with ERCP. Patients with concurrent acute gallstone pancreatitis and choledocholithiasis are advised to undergo single-stage laparoscopic cholecystectomy and bile duct exploration.[6]

Complications

There are generally two classifications of AP complications.[2,9,16] Two studies[2,16] have classified complications due to pancreatitis based on local and systemic presentations.[2,16] Local complications were classified according to the Atlanta criteria,[16] which involve identification with CT scan,[16] accumulation of pancreatic fluid, development of pancreatic pseudocysts, as well as formation of patches of necrotic tissue in the pancreatic parenchyma and peripancreatic tissue.[2,9] These patches may be infected or sterile, and with the progression of time, necrosis may be walled off, which can be identified by an elevated white blood count.[2,9] Systemic complications, according to the Atlanta criteria, generally include aggravation of preexisting conditions such as liver disease, lung disease, or cardiovascular diseases.[2]

Complications that may occur as a result of exacerbation of AP include shock, pulmonary and renal failure, gastrointestinal bleeding, multiorgan system failure,[2,9,16] as well as pancreatic exocrine insufficiency (PEI), which is a condition where insufficient pancreatic enzymes are secreted.[20] Prevention is always key, and thus, prophylactic antimicrobial therapy is recommended for prevention of necrosis.[2] However, in cases where infected necrosis is confirmed, antimicrobial therapy is indicated,[9] and it should be tailored for each patient via prior sensitivity testing before initiation of antibiotics.[9]

However, it should be noted that there have not been guidelines set in place on empirical antimicrobial therapy in the case of necrotic pancreatitis.[9]

Guidelines

Comparison of therapeutic guidelines: Several studies were found to discuss the recommendations for managing AP and gallstone-induced pancreatitis,[2,7,9] but no consensus was reached on prophylactic antibiotic therapy. Greenberg et al.[9] suggested that antibiotics should be prescribed only in patients with infected necrosis confirmed by fine-needle aspiration or if there is gas within a collection visualized on CT scan. Wu and Banks[2] suggested that antibiotics show prophylactic treatments with all patients with AP, whereas Tenner et al.[7] stated that the routine use of prophylactic antibiotics is not recommended.

All the aforementioned studies[2,7,9] discussed recommendations for AP and included considerations for various laboratory values that include hypercalcemia, hypertriglyceridemia, and serum lipase.[2,7,9] In the study conducted by Greenberg et al.,[9] recommendations were classified according to strength of evidence. In the study conducted by Tenner et al.,[7] the recommendations were classified based on initial management, management of local complications, and management of extrapancreatic complications. In the study conducted by Wu and Banks,[2] the guideline was systemized according to mild, moderate, and severe complications. However, none of the guidelines in the articles stated specific recommendations according to gender, age, or total body weight.

Recurrent acute pancreatitis

Risk factors

According to a retrospective analysis conducted in 2019,[16] the cases of recurrent attacks were highest in cases of males, smokers, excessive alcohol consumption, and biliary disease. The highest rates of recurrence were found in alcoholics; therefore, alcoholic patients with recurrent pancreatitis are encouraged to enroll in rehabilitation programs. Additionally, in the case of biliary disease, cholecystectomy was recommended and found to reduce the recurrence of AP.[16] As a visual aid, this is demonstrated in Figure 5.

Genetic variants

In a study conducted in 2019,[15] genetic variants were detected by TruSeq enrichment and Illumina sequencing in eight genes involved in pancreatitis. In terms of the type of genes involved in the progression to RAP, having CFTR and PRSS1 gene mutations was not statistically significant with regard to time to progression to RAP, but having a SPINK1 mutations was significantly associated with faster progression to RAP.[15]

Chronic pancreatitis

Etiology

According to the studies conducted by Pham and Forsmark[8] and Weiss et al.,[11] the most common cause of CP is chronic alcoholism, followed by idiopathic causes, hyperlipidemia, hereditary and genetic causes, and obstructive CP.[8] In both the studies,[6,11] there is a consensus that alcoholism is the primary etiological factor involved in CP. Furthermore, Weiss et al.[11] took gender into consideration and further stated that women were at higher risk of CP, even in cases
of consuming relatively lower levels of alcohol. The hierarchy of etiology is illustrated in Figure 6.

Risk factors
According to the studies conducted by Garber et al., Pham and Forsmark, and Magnusdottir et al., the TIGAR-O risk factor classification system is the most commonly used risk factor classification system for CP. This classification system categorizes the risks based on modifiers, not on the basis of etiology. These modifiers include toxic-metabolic, idiopathic, genetic, autoimmune, recurrent and severe AP-associated CP, and obstructive factors. This is illustrated in Figure 7.

Genetic variants
According to the study conducted by Abu-El-Haija et al., genetic variants were detected by TruSeq enrichment and Illumina sequencing in eight genes involved in pancreatitis. Having two genes involved was associated with faster progression to CP compared to having one or no genes involved. The presence of SPINK1 gene involvement was associated with CP. Progression to CP over time was faster in cases where genes were found positive for variants compared to cases where gene variants were not present, such as CFTR-, SPINK1-, and PRSS1-positive gene cases. The involvement of genetic variants was not previously linked this extensively to the progression of CP, and thus, this is a notable update.

Management
Clinical management of CP is typically symptomatic, and treatment and pharmacotherapy options are largely dependent on presenting symptoms, such as pain and malabsorption, as well as concurrent conditions, such as PEI. According to the United European Gastroenterology evidence-based guidelines for the diagnosis and therapy of CP, endoscopic therapy was recommended for uncomplicated painful CP, but surgery provided more desirable results with regard to pain relief, quality of life, and maintaining exocrine pancreatic function. Pancreatic enzyme replacement therapy (PERT) was indicated for patients with concurrent CP, PEI, and symptoms of malabsorption. A study conducted by Pham and Forsmark discussed in detail the pharmacotherapy options in management of CP. The data collected were compiled into Figure 8 and organized based on symptoms. Although this study briefly discusses the presence of secondary complications due to CP, it does not go into detail on the management of these secondary complications. Furthermore, it does not discuss the role of management in situations of comorbidities, age, gender, or malnutrition.

CLINICAL IMPLICATIONS OF DATA
Based on the data collected on pancreatitis, it can be said that clinical implications can range from mild to severe implications, and each implication requires a guideline to be implemented to provide the best care.

AP is unique in that it can be a mild, self-limiting disease that requires no more than supportive measures, but it can also progress to a more severe state with complications that drastically increase the risk of mortality. It requires quick action and rapid treatment to both prevent acute recurrence and progressive worsening to CP.

In the case of RAP, the steps to be taken require extensive patient counseling because recurrent pancreatitis can result from lifestyle habits that may aggravate AP. Additionally, there has been recommendations on the use of surgical prophylaxis, such as undergoing a cholecystectomy, to prevent recurrence. Surgical prophylactic options have been shown to decrease the rate of readmission and the progression of pancreatic events into CP.

Finally, CP has been found to develop in around 3.7% of patients after the initial occurrence of AP. The steps to be taken to manage CP are provided in a flowchart shown in Figure 8. These steps include the most recent updated steps and recommendations as of the year 2018. Based on the data collected from the years 2013 to 2019, updates in clinical guidelines in addition to the updates in the diagnostic parameters used to evaluate for pancreatitis can be clearly noted, with the most novel and significant findings to be in diagnostic parameters. TAP and trypsinogen-2 serve as the most accurate biomarkers to date. Therefore, this update can be implemented in practice to aid in early detection of AP, which can help greatly decrease morbidity and mortality rates.

CONCLUSION
Although most studies reviewed are similar in their approach of treatment and management of pancreatic disorders, discrepancies have been found, more notably, in the recent articles. Recent updates have limited data on treatment and prophylaxis of AP, CP, and RAP. It is highly recommended to individualize each patient’s treatment according to the updated guidelines; however,
a combination of the updates is needed to integrate all aspects of treatment approaches and act as one updated standardized guideline. This review also recommends the implementation of a specific and distinct guideline for special populations and comorbidities, as one is not yet available in literature. These comprehensive guidelines will reduce mortality and morbidity risks.

Limitations

Although the data collected were extensive, these are not without limitations. The data collected were mostly based on reviews\(^2,4-6,12\) and guidelines,\(^7,9\) but were not specific to special populations. Most reviews tackled the issue of pancreatitis, whether acute, recurrent, or chronic, in a broad-spectrum manner and did not discuss the complications that can occur as a result of age, pregnancy, or comorbidities. Additionally, the guidelines are standardized for adult patients only and do not expand to include special populations such as pediatrics, geriatrics, or pregnant women, or other comorbidities and conditions. Furthermore, not enough data are based on clinical trials. In addition to the lack of clinical trials data, there is also inconclusive local data in the UAE. The statistics\(^18,19\) on the incidence of AP in adults and children collected were national data only limited to the United States. Similar statistics could not be found for incidence of pancreatitis in the UAE. Therefore, these limitations acted as shortcomings for this systematic review.

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Conflicts of interest

There are no conflicts of interest.

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