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The impact of obesity on clinical outcomes in acute pancreatitis: a systematic review

Zofia Kosztyła-Czech[1*], Marta Kowalska[1], Anna Mazur[1], Julia Brodziak[1], Katarzyna Fojcik[1], Wiktoria Ciszewska[1], Tomasz Wiśniewski[1], Michał Dworak[1], Wiktor Werenkowicz[1], Mateusz Matyja[2]

Zofia Kosztyła-Czech [ZKC]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: 0009-0001-0839-4476
E-mail: zkosztyla@gmail.com

Marta Kowalska [MK]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: https://orcid.org/0009-0001-1397-9102
E-mail: martaa.kowalska@yahoo.com

Anna Mazur [AM]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: https://orcid.org/0009-0007-3433-4550
E-mail: ankat.mazur@gmail.com

Julia Brodziak [JB]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: https://orcid.org/0009-0000-3332-9919
E-mail: juliak.brodziak@gmail.com

Katarzyna Fojcik [KF]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: https://orcid.org/0009-0000-5249-8398
E-mail: kat.fojcik@gmail.com

Wiktoria Ciszewska [WC]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: https://orcid.org/0009-0008-9090-9715
E-mail: wiktoria.ciszewska@gmail.com

Tomasz Wiśniewski [TW]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: https://orcid.org/0009-0001-7919-8309
E-mail: tomasz.wisniewski650@gmail.com

Michał Dworak [MD]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: https://orcid.org/0009-0006-9771-0421
E-mail: dworakmichal98@gmail.com

Wiktor Werenkowicz [WW]
Medical University of Silesia
Poniatowskiego 15, 40-055 Katowice
ORCID: https://orcid.org/0009-0008-8487-2426
E-mail: wiktorwerenkowicz@gmail.com

Mateusz Matyja [MM]
Wojewódzki Szpital Specjalistyczny nr 5 w Sosnowcu
Plac Medyków 1, 41-200, Sosnowiec
ORCID: https://orcid.org/0009-0001-8928-0170
E-mail: Matyja.Mateusz1@gmail.com

***Corresponding Author:**
Zofia Kosztyla-Czech, E-mail: zkosztyla@gmail.com

ABSTRACT

Background:

Obesity may influence the severity of acute pancreatitis (AP) by intensifying inflammatory and metabolic disturbances. However, evidence is inconsistent due to differences in patient characteristics and AP causes. Clarification is needed to determine if obesity reliably predicts severe AP and complications.

Aim:

To evaluate the association between obesity, disease severity, complications, and clinical outcomes in AP.

Methods:

We conducted a systematic review of observational studies following PRISMA guidelines. We searched databases for studies on adult AP patients assessing obesity or BMI in relation to disease severity, organ failure, local complications, ICU admission, or mortality. We excluded studies on visceral adiposity indices only, pediatric cohorts, non-original publications, and non-RCT/non-observational designs.

Results:

Eleven studies met the inclusion criteria. Most reported that obesity is linked with increased AP severity and higher rates of systemic or local complications, such as organ failure, necrosis, and greater healthcare utilization. Large database studies confirmed increased complication rates in obese patients. However, findings regarding mortality were inconsistent: some studies noted higher mortality in overweight individuals, others showed no difference. Studies using BMI as a continuous variable found a consistent association between higher BMI and increased AP severity.

Conclusions:

Obesity appears to be associated with more severe AP and selected complications, but its link to mortality remains unclear. BMI alone shows limited predictive value and should be interpreted alongside other clinical data. Further research is required to standardize the incorporation of obesity into early AP risk assessment.

Keywords: Obesity; Acute pancreatitis; Disease severity; Mortality; Clinical outcomes

1. INTRODUCTION

Acute pancreatitis (AP) is one of the most common causes of hospital admission in patients with acute gastrointestinal diseases, and its incidence has increased worldwide over the past decade. In most patients, the disease is mild and resolves spontaneously, but in approximately 20-30% of patients, the disease progresses to moderate or severe forms, accompanied by local necrosis or organ failure or both, resulting in a high risk of death and prolonged hospitalization (Frossard et al., 2009). Therefore, early identification of patients at risk of developing acute pancreatitis is a key element in clinical management, as it is crucial for decisions regarding the intensity of monitoring, level of care, and early treatment.

In parallel, the global epidemic of obesity has profoundly changed the risk profile of patients presenting with AP. Obesity is associated with gallstone formation, hypertriglyceridemia and metabolic syndrome, all recognized risk factors for AP, and it may also modify the clinical course once pancreatitis develops (Bonfrate et al., 2014; Hansen et al., 2020). Several pathophysiological mechanisms have been proposed to explain the adverse effects of excess adiposity: increased intra- and peri-pancreatic fat that can undergo lipolysis and necrosis; release of toxic free fatty acids; a chronic low-grade inflammatory state; and altered adipokine profiles, including leptin and adiponectin (Frossard et al., 2009). These mechanisms suggest that obese patients could be more prone to local complications, systemic inflammatory response and organ failure.

Early clinical studies appeared to support this hypothesis. Obesity has been identified as an independent risk factor for local complications, organ failure, and death in AP. Therefore, it has been suggested that BMI be included in prognostic outcomes (Abu Hilal and Armstrong, 2008). Cohort studies and subsequent meta-analyses suggest that obesity increases the risk of AP and the likelihood of severe disease (Katuchova et al., 2014; Kebkalo et al., 2019; Hansen et al., 2020). However, the latest evidence is not entirely conclusive. Extensive analyses of administrative databases have shown that obese patients may have higher rates of systemic complications and resource consumption. However, this does not necessarily mean higher mortality, and in some series, patients with normal body weight had the worst survival outcomes ("obesity paradox") (Dahiya et al., 2023; Khetpal et al., 2025; Biberci Keskin et al., 2020). In the case of alcoholic PA, obesity is associated with longer hospitalization and a higher risk of renal and respiratory failure, but its impact on mortality is unclear (Pellegrini et al., 2024). In cohorts of older patients, obesity did not translate into worse outcomes compared to patients of normal weight (Biberci Keskin et al., 2020).

These results are characterized by discrepancies that can be explained by differences in study design, populations, definitions of obesity, and the extent to which confounding factors such as age, comorbidities, and etiology are accounted for. Despite well-known limitations in distinguishing between visceral and subcutaneous fat, BMI has been used as a crude indicator of obesity in many studies. Therefore, more recent studies have focused on measures of visceral obesity, such as visceral fat area on computed tomography, visceral obesity index, or triglyceride-glucose-BMI indices. These may better reflect metabolic risk, but they have certain

limitations—they are not routinely available and are not directly comparable to the classic BMI-based definitions used in everyday practice (Xia et al., 2022; Angadi et al., 2024). These studies highlight the biological validity of obesity-related risk, but they do not answer the practical clinical question of how to interpret BMI-based obesity in patients with AP.

Furthermore, heterogeneous definitions of exposure (overweight and obesity, BMI as a continuous variable vs. a categorical variable) and outcomes (different severity scales, composite endpoints) were often combined by previous reviews and meta-analyses. More recent, large-scale database studies and cohorts with current AP management practices have not been included by many of them (Abu Hilal and Armstrong, 2008; Bonfrate et al., 2014). Several large retrospective and prospective cohorts have been published since then. Analyses of national inpatient databases and specialized single-center studies are important because they provide relevant data on different age groups, etiologies, and healthcare settings (Huang et al., 2023; Ak et al., 2023; Dahiya et al., 2023; Khetpal et al., 2025; Pellegrini et al., 2024).

Given the serious obesity problem and the clinical need to simplify tools for early risk stratification in AP, it is reasonable to update the evidence synthesis based on BMI. In particular, it is unclear to what extent obesity, defined primarily by BMI, is associated with (1) severe AP according to current severity classifications, (2) organ failure and local complications, (3) ICU admission and healthcare utilization, and (4) short-term mortality. It is important to know whether these associations differ across subgroups, e.g., elderly patients or patients with specific etiologies.

Therefore, the aim of this systematic review is to critically appraise and synthesize available observational data on the impact of obesity, defined mainly using BMI categories, on the severity and clinical outcomes of acute pancreatitis in adults. By focusing on BMI-based definitions readily applicable in routine practice and explicitly describing study heterogeneity, we aim to clarify the strength and consistency of the association between obesity and adverse outcomes in AP and to identify gaps for future research.

2. REVIEW METHODS

Study Design. This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. The review

protocol was not registered in a publicly accessible database (e.g., PROSPERO), which should be considered a limitation of this study.

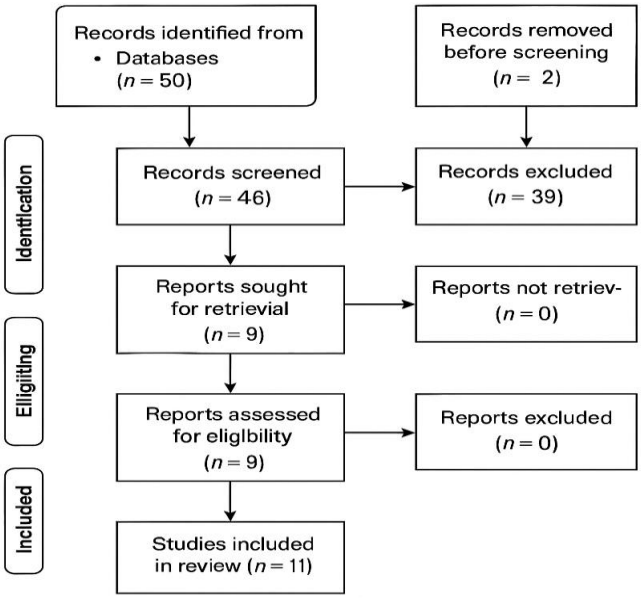
Databases and search strategy. A comprehensive literature search was performed in PubMed/MEDLINE, Embase and the Cochrane Library from their inception to January 2025. The search strategy combined Medical Subject Headings (MeSH) and free-text terms related to obesity and acute pancreatitis. The following key terms were used in various combinations: *"acute pancreatitis"*, *"obesity"*, *"body mass index"*, *"BMI"*, *"overweight"*, *"severity"*, *"organ failure"*, and *"mortality"*. Only full-text English-language articles were included.

Reference lists of relevant reviews and included studies were manually screened to identify additional eligible publications.

Qualification criteria. Included: an observational design (prospective or retrospective cohort, case–control, or cross-sectional), involved adult patients (≥18 years) with a confirmed diagnosis of acute pancreatitis, and examined obesity or body mass index as the exposure variable, reported categorically or continuously. Eligible studies were required to report at least one severity-related clinical endpoint, including standardized severity classifications, organ failure, local complications, ICU admission, or in-hospital or short-term mortality. Only full-text articles presenting original data were considered. Excluded: review articles, meta-analyses, editorials and commentaries; cohorts limited to pediatric populations; studies assessing only visceral adiposity metrics without BMI-based exposure groups; mechanistic, genetic, animal or other experimental investigations; case reports or case series comprising fewer than ten participants; and studies lacking extractable outcome data or not incorporating BMI in their analyses. Excluded examples include works focused exclusively on visceral adiposity measures or on machine-learning predictive models without BMI categorization.

Study selection and reasons for exclusion. All records identified through database searches were imported into Rayyan, where duplicates were automatically removed. Two reviewers independently screened titles and abstracts against predefined criteria. Full-text screening was subsequently performed for all articles marked as "include" or "maybe." Disagreements were resolved through discussion and, if necessary, by a third reviewer. A PRISMA flow diagram summarises the study selection process.

Figure 1. PRISMA 2020 diagram



A standardized data extraction template was developed prior to the full-text assessment. From each eligible study, the following information was collected: study characteristics (first author, publication year, country, study design, sample size); participant demographics and clinical features (age, sex distribution, etiology of acute pancreatitis); the definition or categorization of obesity or BMI; the severity classification system employed; and all relevant outcomes, including severity of acute pancreatitis, organ failure, pancreatic necrosis, pseudocyst formation, ICU admission, and mortality. Extracted data also included reported effect estimates (odds ratios, relative risks, hazard ratios) and variables incorporated into multivariable adjustment. Data extraction was conducted independently by two reviewers, with disagreements resolved through consensus.

Quality Assessment. Although ROBINS-I was originally designed for non-randomized intervention studies, in this review it was applied descriptively to assess key domains of bias relevant to observational studies with BMI as the exposure. Studies were evaluated across domains including confounding, participant selection, exposure classification, outcome measurement, missing data, and selective reporting. Risk-of-bias assessments informed the narrative synthesis but did not lead to exclusion of studies. Overall, most studies were judged to have a moderate risk of bias, primarily due to residual confounding and exposure

misclassification, while large administrative database studies showed a higher risk in the domains of confounding and outcome measurement. The tool was used to structure domain-specific judgments rather than to derive a single composite risk-of-bias score.

Data synthesis. Because of substantial methodological heterogeneity across studies - including differences in BMI categorization, severity definitions, outcome reporting and adjustment for confounders - a quantitative meta-analysis was not performed. Instead, a structured narrative synthesis was conducted, comparing results across similar outcomes and highlighting consistent and divergent findings.

3. RESULTS

A database search identified a large number of records. After removing duplicates and reviewing titles and abstracts, a full assessment of the relevant articles was conducted. Eleven studies met all inclusion criteria and were included in the final synthesis. The main reasons for exclusion were the lack of BMI grouping, reliance solely on visceral obesity measurements, pediatric populations, or insufficient outcome data. The summary of the selection process is presented in the PRISMA diagram (Figure 1).

The included studies comprised retrospective and prospective cohort analyses from Europe, Asia, and the United States (Katuchova et al., 2014; Kebkalo et al., 2019; Biberci Keskin et al., 2020; Huang et al., 2023; Ak et al., 2023; Dahiya et al., 2023; Pellegrini et al., 2024; Muscat et al., 2024; Khetpal et al., 2025). The sample size ranged from less than 100 to several hundred thousand in large studies based on databases of hospitalized patients. The definitions of obesity varied, but most studies used a BMI $\geq$30 kg/m^2. Some used continuous BMI values without categorical thresholds (Huang et al., 2023; Ak et al., 2023; Muscat et al., 2024).

The revised Atlanta criteria, the Ranson scale, the modified CT severity index, APACHE II, and BISAP were used for severity classification, depending on the study. The distribution of etiology varied across cohorts, with gallstones, hypertriglyceridemia, and alcohol being the most common causes (Katuchova et al., 2014; Huang et al., 2023).

Table 1. Characteristics of included studies

Study	Country	Study design	Sample Size	Definition of Obesity	Definition of Severity
Katuchova et al., 2014	Slovakia	Prospective cohort	384	BMI + waist circumference	Ranson, APACHE II, CT severity index
Kebkalo et al., 2019	Ukraine/Poland	Retrospective cohort	482	BMI ≥30 kg/m²	Revised Atlanta
Biberci Keskin et al., 2020	Turkey	Retrospective cohort	190	BMI ≥30 kg/m²	Revised Atlanta
Hansen et al., 2020	Denmark	Prospective cohort	118,085	BMI categories	Registry-based definitions
İnce et al., 2022	Turkey	Retrospective cohort	1,134	BMI categories (NW/OW/OB)	Atlanta + Balthazar-CTSI
Huang et al., 2023	China	Prospective cohort	194	BMI (continuous)	Revised Atlanta
Ak et al., 2023	Turkey	Retrospective cohort	514	BMI (continuous)	Revised Atlanta
Dahiya et al., 2023	USA	Retrospective NIS database	420,600	BMI-coded	Not fully specified
Pellegrini et al., 2024	USA	Retrospective NIS database	229,510	BMI-coded	Revised Atlanta (inferred)
Muscat et al., 2024	UK	Retrospective cohort	59	BMI (continuous)	Revised Atlanta
Khetpal et al., 2025	USA	Retrospective NIS database	267,000	BMI-coded categories	Administrative severity proxies

Footnote: “Higher” indicates a statistically significant increase in risk compared with the reference group, as reported in the original study. “Mixed” denotes inconsistent findings across

outcomes or subgroups. “Not reported” indicates that the outcome was not explicitly analyzed. Risk of bias was assessed using the ROBINS-I tool. In registry-based and administrative database studies, severity was inferred using proxy measures such as ICU admission, mechanical ventilation, organ failure codes, length of hospital stay, or ICD-coded complications, as defined by the original authors.

Obesity and Severity of Acute Pancreatitis

Most of the included studies showed a positive association between obesity and the severity of acute pancreatitis, although a minority of cohorts did not confirm this relationship, likely reflecting differences in sample size, study design, and population characteristics. Studies using BMI category thresholds consistently reported a higher incidence of moderate and severe acute pancreatitis among obese individuals (Katuchova et al., 2014; Kebkalo et al., 2019; Dahiya et al., 2023), and analyses of large national databases confirmed this pattern (Dahiya et al., 2023; Khetpal et al., 2025). Most studies analysing BMI as a continuous variable also demonstrated a positive association with AP severity (Huang et al., 2023; Ak et al., 2023), although one small cohort did not confirm this finding, probably due to sample size limitations (Muscat et al., 2024).

Obesity and Organ Failure

Many studies have shown an increase in the incidence of organ failure, including renal and respiratory failure, in obese individuals (Kebkalo et al., 2019; Pellegrini et al., 2024; Dahiya et al., 2023). In cases of alcoholic pancreatitis, obesity is associated with an increased risk of renal failure and severe respiratory failure (Pellegrini et al., 2024). It is worth noting that a study conducted on patients aged 65 and older did not show a significant increase in the number of organ failure cases among obese individuals. This may indicate a reduced risk of obesity in older age groups (Biberci Keskin et al., 2020).

Obesity and Local Complications

Multiple studies demonstrated an association between obesity and local complications, including pancreatic necrosis, peripancreatic collections, and higher CT severity scores

(Katuchova et al., 2014; Kebkalo et al., 2019). However, one cohort reported a lower incidence of necrosis in obese patients despite an overall more severe disease course, highlighting heterogeneity in imaging-based outcomes (Biberci Keskin et al., 2020), which is likely related to differences in imaging methods, terminology, and definitions.

Obesity and admission to intensive care units

Many studies have shown that admission to intensive care units was more common among obese patients, especially in large administrative databases, indicating greater use of health services (Dahiya et al., 2023; Khetpal et al., 2025). Some smaller studies found no significant differences (Muscat et al., 2024), while others reported higher hospitalization rates among obese or overweight patients when additional risk factors such as hypertriglyceridemia or smoking were present (İnce et al., 2022).

Obesity and mortality

The results regarding mortality were heterogeneous. Several studies reported increased mortality among obese patients - especially those with severe disease or alcoholic etiology (Pellegrini et al., 2024; Kebkalo et al., 2019). However, other studies did not find such an association and even reported higher mortality among individuals without elevated BMI, particularly in the older patient group (Biberci Keskin et al., 2020). Differences in age distributions, comorbidities, and coding methods across administrative databases may contribute to variability in study results.

Impact of etiology

The association between obesity and clinical outcomes varied by etiology. A higher percentage of adverse outcomes and longer hospital stays were associated with alcoholic AP (Pellegrini et al., 2024). In the case of AP caused by hypertriglyceridemia, obesity may have exacerbated inflammatory responses (Huang et al., 2023). An epidemiological association between obesity and AP of gallstone etiology has been demonstrated, but results regarding disease severity have been inconsistent across studies (Katuchova et al., 2014).

Summary of evidence

Overall, the association between obesity and increased severity and complications of acute pancreatitis is confirmed by most of the studies included. However, the results regarding mortality are inconsistent, suggesting that obesity alone may not be a reliable prognostic marker in all patient subgroups. The variety of BMI definitions, severity classifications, and population characteristics contributes to the heterogeneity of results. More precise measures of obesity and metabolic health may improve future risk stratification.

4. DISCUSSION

This systematic review synthesizes evidence from eleven observational studies assessing the relationship between obesity and clinical outcomes in acute pancreatitis. Across diverse populations and clinical settings, excess adiposity was most consistently associated with a higher likelihood of severe disease, increased risk of organ failure, greater frequency of local complications, and higher healthcare utilization. However, the magnitude and consistency of these associations varied across studies, reflecting differences in study design, exposure definitions, severity classifications, and patient characteristics. By integrating these findings with prior meta-analyses and mechanistic research, our results support the interpretation that obesity represents an important modifier of disease course in acute pancreatitis rather than a uniform or independent prognostic determinant.

Table 2. Summary of clinical outcomes.

Study	**Severity in Obese**	**Organ Failure**	**Local Complications**	**Mortality**
Katuchova et al., 2014	Higher	Higher	Higher	Higher
Kebkalo et al., 2019	Higher	Higher	Higher	Higher
Biberci Keskin et al., 2020	No difference	No difference	Lower or similar	Lower
Hansen et al., 2020	Not applicable	Not applicable	Not applicable	Not reported

İnce et al., 2022	Higher	Higher	Higher	Higher
Huang et al., 2023	Higher	Higher	Higher	Not reported
Ak et al., 2023	Higher	Not reported	Not reported	Not reported
Dahiya et al., 2023	Mixed	Mixed	Higher in normal weight	Lower in obese
Pellegrini et al., 2024	Higher (acute alcoholic pancreatitis)	Higher	Not reported	Higher
Muscat et al., 2024	No difference	No difference	No difference	No difference
Khetpal et al., 2025	Higher	Higher systemic complications	Not reported	Similar

Obesity and the severity of acute pancreatitis

Most studies included in this review showed a clear association between obesity and increased severity of AP. Several cohort studies have shown that obese patients were at greater risk of developing moderate or severe pancreatitis (Huang et al., 2023; Ak et al., 2023; Kebkalo et al., 2019). This association is consistent with previous meta-analyses, which demonstrated a strong dose-response effect, with the risk of severe AP gradually increasing with increasing BMI.

For example, Dobszai et al. (2019), after analyzing 10,000 patients, concluded that the likelihood of developing severe AP is almost three times higher in people with a BMI above 25 kg/m² compared to patients of normal weight. This is further supported by a large meta-analysis of the Asian population, which found that obesity (BMI ≥30 kg/m²) was associated with a 2.75-fold increase in the likelihood of developing severe AP (Vadukoot Lazar et al., 2025). These consistent observations confirm the clinical significance of obesity as a prognostic factor for AP severity.

However, this correlation has not been confirmed by all studies. A study conducted by Muscat et al. (2024) did not show a significant association between BMI and disease severity, length

of hospital stay, or complications. Possible reasons for these conflicting results include differences in sample size, ethnicity, prevalence of comorbid metabolic disorders, and distribution of mild and severe cases.

Organ failure and systemic complications

Organ failure is one of the main factors determining mortality in AP. A higher rate of organ failure among obese patients has been demonstrated by most of the studies included in this review.

For example, Khetpal et al. (2025) found that obesity is associated with an increased likelihood of systemic complications such as respiratory failure and renal dysfunction. Similar conclusions were drawn by Pellegrini et al. (2024), who reported a higher incidence of acute renal failure and respiratory failure in obese individuals with alcohol-induced pancreatitis.

A meta-analysis of individual patient data conducted by Smeets et al. (2019) showed that obesity was independently associated with organ failure (RR 1.38) and multiple organ failure (RR 1.81).

Systemic complications in obese patients are likely to be caused by interactions between inflammatory responses and metabolic disorders. Visceral adipose tissue secretes pro-inflammatory cytokines (TNF-α, IL-6), chemokines, and adipokines (leptin, resistin), which induce activation of the immune system. Excess fat contributes to increased lipolysis, releasing free fatty acids that induce mitochondrial dysfunction, endothelial damage, and lung damage (Frossard et al., 2009). These pathophysiological pathways are associated with the occurrence of respiratory and renal failure.

Local complications: necrosis

Several studies have found that obese patients are more likely to experience local pancreatic complications, including necrosis, pseudocyst formation, and infected accumulations. Kebkalo et al. (2019) and Katuchova et al. (2014) reported a significantly higher rate of necrosis among obese patients. Huang et al. (2023) showed that high BMI correlates with gastrointestinal barrier dysfunction and necrosis (34 cases in the cohort).

However, data on the exact impact of visceral obesity compared to BMI alone remain limited. Visceral fat area (VFA), which is a more detailed anthropometric measurement, appears to be a better predictor of severe AP and the development of local complications. A prospective Indian study by Angadi et al. (2024) showed significantly higher VFA in patients with severe AP. VFA predicted severe disease progression—AUC 0.722. Other studies have confirmed that VAI outperforms BMI in predicting AP severity (Gu et al., 2025; Xia et al., 2022).

This suggests that not all obesity phenotypes are associated with the same risk of developing AP and its severity. Individuals with a predominance of visceral fat may be at significantly higher risk of necrotizing pancreatitis.

Mortality and the "obesity paradox."

Findings regarding mortality remain inconsistent across studies, partly due to differences in age structure, comorbidity burden, and coding methodology. Some cohorts, such as that of Kebkalo et al. (2019), reported significantly higher mortality among obese individuals, whereas others observed the opposite pattern. Dahiya et al. (2023) found that patients with normal body weight were more likely to die during hospitalization despite having fewer comorbidities, raising the possibility of an "obesity paradox".

There are several explanations for this paradox:

1. BMI does not distinguish between muscle mass and fat mass. A high BMI does not always indicate visceral obesity, as is the case with athletes, for example.
2. One speculative explanation for this so-called 'obesity paradox' is that obese patients may be admitted earlier due to a higher burden of comorbidities, potentially allowing earlier intervention and closer monitoring; however, this hypothesis has not been consistently demonstrated.
3. Unclear regression error — BMI measured on admission may be underestimated in critically ill patients due to blood concentration and dehydration.

Evidence from meta-analyses is also inconclusive. Dobszai et al. (2019) found that obesity approximately tripled the risk of death. However, a meta-analysis of individual patient data by Smeets et al. (2019) found no association between obesity and mortality.

Role of metabolic syndrome and hypertriglyceridemia

Several studies included in our review highlight the role of metabolic syndrome components—especially hypertriglyceridemia—in modulating AP outcomes. HTG-AP is known to be more severe, and obesity often coexists with dyslipidemia, insulin resistance, and hypertension.

Fu et al. (2024) demonstrated that the accumulation of metabolic syndrome components synergistically increases the risk of systemic complications, renal failure, and severe AP. In particular, low HDL-C concentration has been found to be a strong predictor of poor treatment outcomes. These findings support the concept that metabolic disorders, rather than BMI alone, are the main risk-increasing factor.

According to molecular studies, lipid metabolism plays a key role in the pathogenesis of AP in obese individuals. Ji et al. (2024) identified Hydroxyacyl-CoA Dehydrogenase (HADH) as a potential lipid metabolism–related molecular marker potentially linking high BMI and AP severity, suggesting that lipid-related pathways may exacerbate pancreatic injury.

Impact of sarcopenia and body composition

Sarcopenic obesity is a distinct phenotype characterized by high fat mass and low muscle mass. A study by Cortés et al. (2024) showed that sarcopenic obesity is associated with a more severe course of AP, even though body composition measurements were not consistently associated with recurrent AP. This confirms the approach that muscle loss and changes in metabolic reserves have a real impact on inflammatory responses and disease progression. Studies focusing exclusively on elderly patients, such as the study by Biberci Keskin et al. (2020), found that obesity did not worsen AP treatment outcomes in older adults, suggesting that changes in body composition and age-related diseases may attenuate the effect of BMI.

Hyperlipidemic pancreatitis and intestinal barrier dysfunction

Huang et al. (2023) presented valuable information confirming the link between obesity, intestinal damage, and the severity of AP. In this study, a higher BMI correlated with elevated markers of intestinal permeability (d-lactate, DAO, I-FABP) and gastrointestinal damage. This

correlates with experimental evidence suggesting that free fatty acids exacerbate intestinal barrier dysfunction, facilitating bacterial translocation and, consequently, infectious necrosis. Given the high prevalence of hyperlipidemia among individuals with high BMI, these findings help explain the frequent co-occurrence of systemic inflammation, necrosis, and infection in this population.

Methodological considerations and sources of heterogeneity

The main factors contributing to the heterogeneity of studies include:

1. Differences in BMI thresholds (≥25 vs. ≥30 vs. Asia-specific thresholds).
2. Different definitions of severity (Revised Atlanta vs. Ranson vs. APACHE II).
3. Population differences in etiology (gallstones vs. hypertriglyceridemia vs. alcohol).
4. Differences in the consideration of confounding factors (some studies took age, gender, and comorbidities into account, while others did not).
5. Advances in medicine - newer studies may show better results due to more advanced care during hospitalization.

These factors limit the comparability of results and highlight the need for standardization of reporting.

Clinical implications

The results of this review have several practical implications:

1. Early risk stratification — Obesity should be considered a high-risk factor when a patient presents to the emergency department, prompting closer monitoring and possibly earlier assessment in the intensive care unit.
2. Body composition assessment — Anthropometric indices (VAI, LAP) and visceral fat measurements should be included in future risk models, as they are more reliable than BMI.
3. Treatment of metabolic syndrome — Optimising lipid and glucose metabolism may help reduce the risk of AP and treatment complications.

Future research

Future research should apply standardized definitions and analytical adjustment strategies, examine acute pancreatitis outcomes across distinct obesity phenotypes (including visceral, sarcopenic and normal-weight obesity), and integrate imaging-based assessments of body composition. Further work is also needed to elucidate the so-called obesity paradox and to generate large, prospective cohort data across heterogeneous populations.

5. CONCLUSIONS

This systematic review demonstrates that obesity is an important modifier of clinical outcomes in acute pancreatitis. Across contemporary studies, excess adiposity was consistently associated with a higher likelihood of severe disease, increased risk of organ failure and greater healthcare utilization. Although the association between obesity and mortality has remained variable across cohorts, the most reliable and consistent finding—supported by both observational studies and meta-analysis of individual patient data—is an increased risk of organ dysfunction in obese patients.

Furthermore, evidence suggests that the impact of obesity on acute pancreatitis is multifactorial and goes well beyond BMI alone. Increased susceptibility to systemic complications is associated with visceral obesity, metabolic disorders, and lipid-mediated inflammatory pathways. The dose-response relationship between BMI increase and severe disease progression has been highlighted by recent meta-analyses. At the same time, they reveal the potential vulnerability of underweight patients, suggesting that deviations from normal nutritional status in either direction may adversely affect treatment outcomes.

Given the global problem of increased prevalence of obesity and metabolic syndrome, these findings underscore the need for targeted prevention strategies and early risk stratification in patients with acute pancreatitis.

Future research should incorporate more precise measures of adiposity, account for etiological differences, and use prospective, standardized approaches to elucidate the mechanisms linking metabolic health to disease severity. Understanding these pathways may guide the development of personalized interventions to reduce morbidity and improve outcomes in this increasingly common clinical condition.

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Author's contribution:

Conceptualization: **[ZKC], [AM]**

Methodology: **[ZKC], [JB]**

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Check: **[JB], [MM]**

Formal analysis: **[WC], [MD]**

Investigation:**[TW], [JB] , [WW]**

Resources: **[MK], [TW], [MD]**

Data curation: **[AM], [KF]**

Writing - rough preparation: **[MM], [WC]**

Writing - review and editing: **[MK], [MD], [WW]**

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AI.

AI (Grammarly and Deepl) was utilized for two specific purposes in this research. Text analysis of clinical reasoning narratives to identify linguistic patterns associated with specific logical fallacies. Assistance in refining the academic English language of the manuscript, ensuring clarity, consistency, and adherence to scientific writing standards. **AI** were used for additional linguistic refinement of the research manuscript, ensuring proper English grammar, style, and clarity in the presentation of results. It is important to emphasize that all AI tools were used strictly as assistive instruments under human supervision. The final interpretation of results, classification of errors, and conclusions were determined by human experts in clinical medicine and formal logic. The AI tools served primarily to enhance efficiency in data processing, pattern recognition, and linguistic refinement, rather than replacing human judgment in the analytical process.

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