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ORIGINAL RESEARCH

The Individual and Joint Associations of Sarcopenic Obesity and Malnutrition on Predicting the Severity of Acute Pancreatitis

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Purpose: This study aimed to evaluate the individual and joint associations of malnutrition and obesity on predicting the severity and prognosis of acute pancreatitis (AP).

Patients and Methods: A retrospective analysis of 240 AP patients was conducted. Malnutrition was diagnosed using Global Leadership Initiative on Malnutrition (GLIM) criteria. Obesity was defined using body mass index (BMI) and different ratios of psoas muscle area (PMA) to BMI, of which PMA/BMI was used to define sarcopenic obesity. Patients were categorized into four groups: well-nourished non-obese (WN), malnourished non-obese (MN), well-nourished obese (WO), and malnourished obese (MO). Logistic regression, and trend analyses were employed to assess associations with different AP outcomes.

Results: The MO group exhibited the highest C-reactive protein levels, lowest albumin/hemoglobin, and worst clinical outcomes. Sarcopenic obesity (MO and WO) significantly increased risks of moderately severe/severe AP (OR ≥2.74), composite outcomes (OR ≥2.69) and AP severity (OR ≥3.27), with the MO group having a risk 5–7 times higher than the WN group. As the PMA/BMI quartiles increase, the risk of moderately severe AP (MSAP)+ severe AP (SAP), composite outcomes and the AP severity significantly increased (all p for trend<0.003), and the group with a median PMA/BMI above (sarcopenic obesity) was significantly correlated with MSAP+SAP (OR ≥3.41), composite outcomes (OR ≥3.26), and the severity of AP (OR ≥3.66). Malnutrition alone did not independently elevate risks. However, no such association was observed in BMI based grouping.

Conclusion: Sarcopenic obesity, quantified by PMA/BMI, is a superior predictor of AP severity compared to BMI. The coexistence of malnutrition and sarcopenic obesity synergistically exacerbates inflammation and poor outcomes, emphasizing the need for body composition-guided nutritional interventions.

Keywords: acute pancreatitis, body composition, GLIM, malnutrition, sarcopenic obesity, clinical outcome

Introduction

Acute pancreatitis (AP) is one of the most common acute diseases of the digestive system.[1] According to the 2012 revision of the Atlanta Classification, the diagnosis of AP requires the fulfillment of at least two of the following three criteria: (1) characteristic abdominal pain, (2) serum lipase (or amylase) activity at least three times the upper limit of normal, and (3) characteristic imaging manifestations.[2] This classification further categorizes AP into mild (MAP), moderately severe (MSAP), and severe (SAP).[2] MAP usually resolves within 1–2 weeks; in contrast, the mortality rates for MSAP and SAP are approximately 2% and 36%-50%, respectively.[3] From 1990 to 2019, the age-standardized rates of incidence, prevalence, and mortality of pancreatitis in China decreased, but the incident cases and prevalent patients have increased constantly.[4]

In AP, especially in MSAP and SAP, inflammation and infectious complications increase metabolic demands, energy expenditure and proteolysis. Therefore, all AP patients are at risk for malnutrition and may require nutritional support.[5]

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Malnutrition is an important problem associated with poor clinical outcomes. The Global Leadership Initiative on Malnutrition (GLIM) Assessment Criteria proposed in 2019 are a global consensus for the diagnosis of malnutrition in adults and have gained increasing recognition over the years.[6] GLIM criteria consist of a two steps process: the first step is risk screening through a validated screening tool, and the second step is assessment of malnutrition. As a consensus, it requires validation across various disease conditions. However, evidence regarding malnutrition's impact on adult AP remains limited, with only our previous studies having validated the GLIM criteria in AP populations.[7–9] Notably, obesity as the opposite metabolic extreme of malnutrition also significantly influences AP outcomes. A meta-analysis demonstrated that body mass index (BMI) >25 kg/m² increases the risk of SAP, while BMI >30 kg/m² elevates mortality risk.[10] The potential coexistence of obesity and malnutrition may create a dual metabolic burden that could exacerbate AP complications, including organ failure and mortality. Currently, there are no studies on the prognostic impact of concomitant malnutrition and obesity in AP patients.

Since the phenotypic criteria in the GLIM only require one criterion of low BMI, weight loss, or reduced muscle mass, there are malnourished patients with sarcopenic obesity (high BMI with low muscle mass). This study aimed to evaluate the impact of malnutrition combined with overweight/obesity diagnosed using the GLIM criteria on the prognosis of AP patients.

Materials and Methods

Study Design and Setting

This was a retrospective study that included consecutive AP patients hospitalized from June 2019 to January 2022 at the Affiliated Hospital of Chengde Medical University. AP diagnosis adhered to the 2012 revised Atlanta Classification.[2] Inclusion criteria were as follows: (1) age ≥ 18 years, (2) diagnosis of AP according to the revised Atlanta Criteria, and (3) completeness of vital data. Patients were excluded if they were pregnant, diagnosed with chronic pancreatitis, were hospitalized for less than 48 hours, or lacked essential clinical data. A total of 240 participants were ultimately included in the final analysis. The study protocol was approved by the Hospital Ethics Committee (CYFYLL2022256), which waived the requirement for informed consent from patients due to the retrospective nature of the study. The study complied with the principles of the Declaration of Helsinki.

Clinical and Biochemical Data

We collected the initial clinical and laboratory parameters obtained after the onset of AP, along with data on disease severity and etiology. Clinical variables included gender, age, psoas muscle area (PMA), and BMI. Laboratory parameters encompassed white blood cell count, hemoglobin, albumin, C-reactive protein (CRP), blood glucose, triglycerides, total calcium, blood urea nitrogen, serum creatinine, amylase, and lipase. Corrected serum calcium (CsCa) was obtained through calculation. CsCa (mmol/L) = total serum calcium (mmol/L) + [40 - serum albumin (g/L)] × 0.02.[11]

AP etiology was categorized into four groups: hypertriglyceridemic, biliary, alcoholic, and others. Comorbidities were assessed by the updated Charlson comorbidity index.[12] We collected hospitalization costs, length of stay (LOS), infectious complications, local complications, organ failure, systemic complications, and mortality rate. All complications were defined according to the 2012 revised Atlanta classification.[2] Infectious complications were specifically defined in this study included infectious shock, septicemia, sepsis, infective endocarditis, severe pneumonia, abdominal infection, and a procalcitonin≥25 ng/mL in the absence of any of these diagnoses (except renal failure).Since there was only one patient death, we defined a composite outcomes, which included death, complications, and organ failure.

CT-Demarcated Parameters

All patients underwent CT scanning of the abdomen after the onset of AP. Five patients who had undergone CT at other hospitals but had no CT records at our hospital were excluded. Bilateral PMA (cm^2) was measured manually in each patient by the same trained radiologist, who was unaware of the patient's diagnosis and complications. This was accomplished by measuring CT images at the level of the third lumbar vertebrae with a density threshold set between −29 and +150 Hounsfield units. The areas were then divided by the patient's BMI to derive the ratio of psoas muscle area to BMI (PMA/BMI), which reflect sarcopenic obesity. In order to avoid gender differences in the measurements, we added body surface area to correct for the formula: √body surface area (BSA)/(PMA/BMI)

(for simplicity, it is still referred to as PMA/BMI in the following text, which is its reciprocal).[13] The BSA was calculated using the formula proposed by Mosteller, BSA (m^2)=√height(cm)×weight(kg)÷3600.[14]

Malnutrition Screening and Assessment

The diagnosis of malnutrition was determined according to the two-step GLIM criteria. In the first step, as a validated screening tool, if the NRS2002≥ 3, the patient was considered to be at risk of malnutrition and proceeded to the next step of the evaluation. In the second step, malnutrition was identified when the patient met one of the three phenotypic criteria + one of the two etiologic criteria. Phenotypic criteria included (1) involuntary weight loss, > 5% within 6 months or > 10% over 6 months; (2) low BMI, < 18.5 kg/m^2 if < 70 years or < 20.0 kg/m^2 if ≥ 70 years; and (3) reduced muscle mass, based on the results of our previous study, PMA ≤ 11.50 cm^2 in males and PMA ≤ 8.22 cm^2 in females.[7] AP was considered to meet etiologic criterion.[15]

Assessment of Overweight/Obesity

Four methodologies were employed to evaluate overweight/obesity. 1.Conventional BMI classification: overweight (24 kg/m^2 ≤ BMI < 27.9 kg/m^2) and obesity (BMI ≥ 28 kg/m^2). 2.Sarcopenic obesity based on PMA/BMI median: PMA/BMI values ≥ the cohort median were classified as sarcopenic obesity. 3. Receiver operating characteristic (ROC)-derived PMA/BMI cutoff: Optimal cutoff for sarcopenic obesity were determined via ROC curve analysis (detailed in statistical analysis). 4.PMA cutoff from our prior research: A previously established PMA threshold combined with BMI was tested but excluded due to insufficient cases (n=2). Consequently, only the first three methods were utilized for subsequent analyses. The flowchart of this study is shown in Figure 1.

Statistical Analysis

SPSS 26 (IBM, USA) was used for statistical analysis of data. SAP was selected as the clinical endpoint. ROC curves were generated to evaluate the performance of the PMA/BMI, with optimal thresholds determined by maximizing Youden's index (sensitivity + specificity − 1). Patients were stratified into four groups using three overweight/obesity definitions combined with the GLIM criteria: well-nourished non-obese (WN), malnourished non-obese (MN), well-nourished obese (WO), and malnourished obese (MO). Continuous variables were presented as medians (interquartile range) and multiple comparisons were made using the Kruskal–Wallis test and Dunn's post hoc test. Categorical variables were presented as numbers (percentages) and compared using the chi-square test or Fisher's exact test. Bonferroni correction was conducted for multiple comparisons. Multivariate sequential logistic regression was performed to identify variables associated with the occurrence of different clinical outcomes. Patients were further categorized into four groups based on BMI (emaciated [<18.5 kg/m^2], normal [18.5–24 kg/m^2], overweight, and obese) and the quartiles of PMA/BMI levels. The associations between each group and different clinical outcomes were analyzed through binary and ordinal logistic regression analysis to explore the possible interaction effects of malnutrition, and trend tests were conducted. Categorical transformations were done for continuous variables in the regression models. In addition, following the advice of peer review, Firth's bias reduction method (sub-type of logistic regression) is used for rare event outcomes due to the low incidence of some events. When significant interactions emerged, stratified analyses by malnutrition status were performed. A two-tailed p-values < 0.05 considered statistically significant. Firth's logistic regression is performed in the R v(4.2.0).

Results

The median of PMA/BMI was 2.93. The optimal PMA/BMI cutoff for predicting SAP occurrence was 3.05, demonstrating an area under the curve of 0.697 with sensitivity of 0.792 and specificity of 0.584 (Figure 2).

Baseline Characteristic

According to the grouping of PMA/BMI (cutoff ≥ 3.05) and GLIM criteria, there were 118 cases (49.2%) in the WN group, 13 cases (5.4%) in the MN group, 89 cases (37.1%) in the WO group, and 20 cases (8.3%) in the MO group (Table 1).

The WN group exhibited the lowest female proportion, the youngest age, the highest PMA, and the highest white blood cell counts compared with other groups (all p<0.05). Although the WN group showed the highest triglyceride

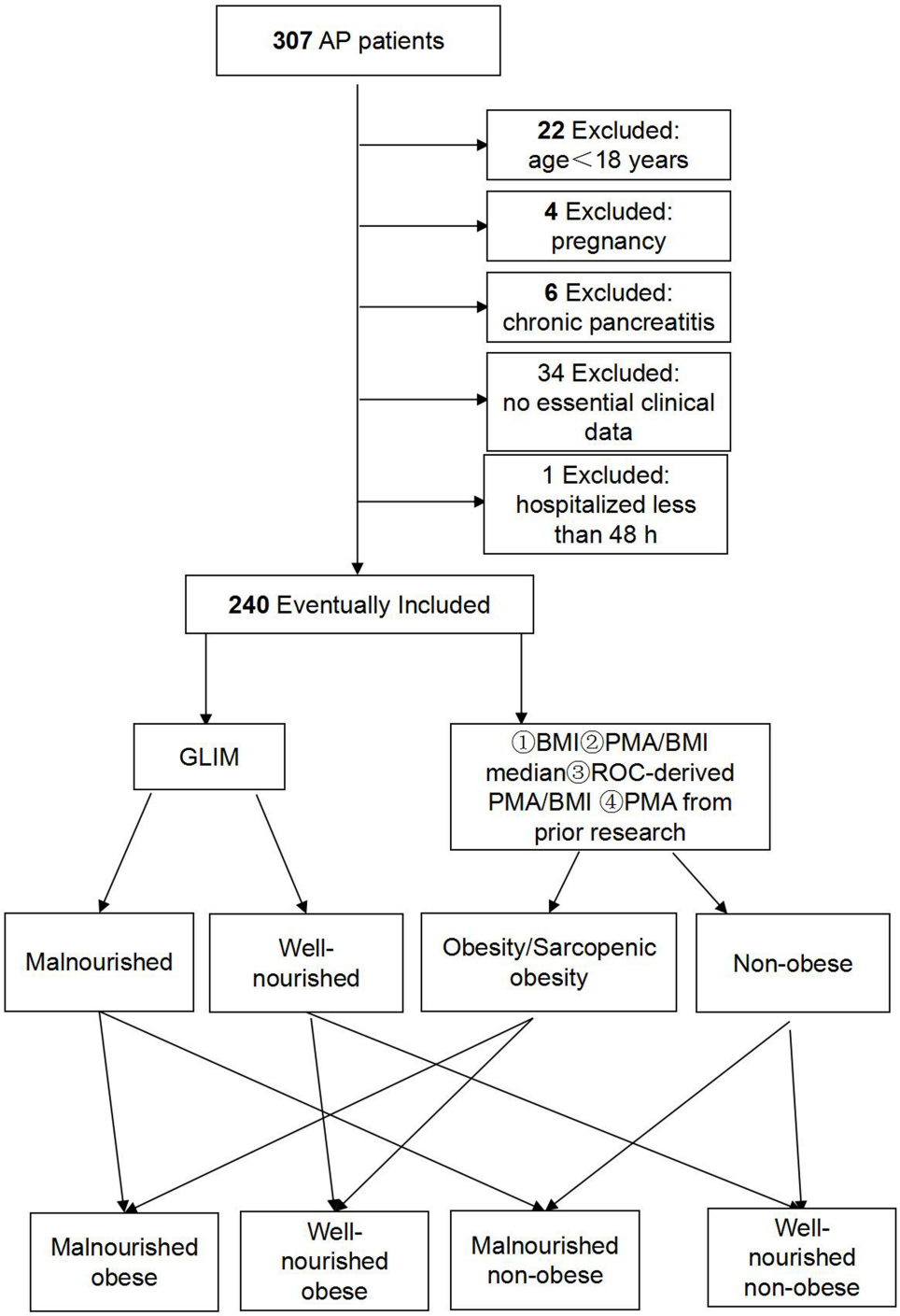

Figure 1 Flowchart of the study.

levels, this difference reached statistical significance only when compared with the MN group. Both non-obese groups (WN and MN) demonstrated significantly higher albumin levels, hemoglobin concentrations, and PMA/BMI ratios along with lower female proportions compared to obese groups (WO and MO). Well-nourished groups (WN and WO) had significantly higher BMIs than their malnourished counterparts.

The MO group displayed the oldest age and highest CRP levels among all groups, while simultaneously showing the lowest PMA values, albumin levels, and hemoglobin concentrations, with statistical significance when compared to the WN group ($p<0.05$). The WO and MN groups generally showed intermediate characteristics between the two extreme groups

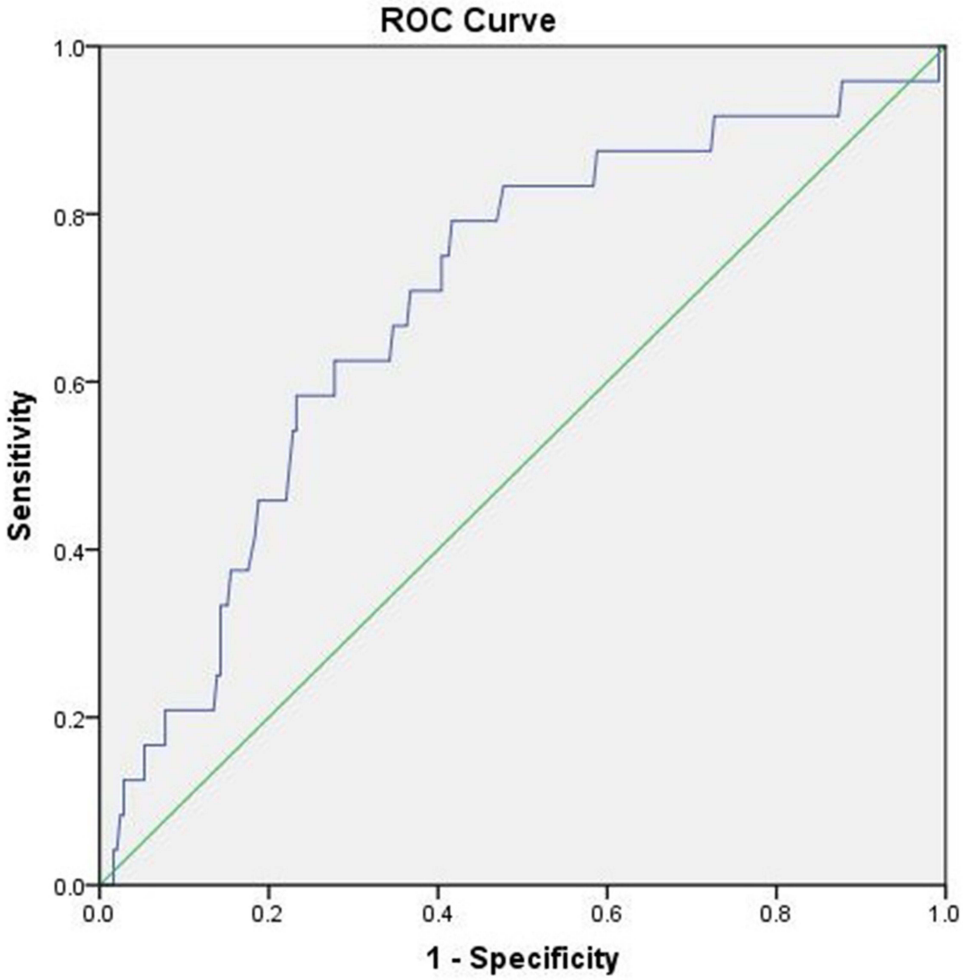

Figure 2 Receiver operating characteristic curve for predicting sever acute pancreatitis based on PMA/BMI.

(WN and MO) or demonstrated no statistically significant differences in most parameters. The WO group exhibited significant differences in comorbidity scores compared to the WN group despite sharing the same median of 0. The MN group showed higher CsCa than both the MO and WN groups.

Table 1 Baseline Demographics of Acute Pancreatitis Patients Grouped According to Obesity (PMA/BMI ≥ 3.05) and GLIM Criteria

Characteristic	Malnourished Obese	Well-Nourished Obese	Malnourished non-Obese	Well-Nourished non-Obese	p
Total n (%, N = 240)	20(8.3)	89(37.1)	13(5.4)	118(49.2)	
Female, n (%)	15(65.2)[c]	72(73.5)[b,c]	4(26.7)	20(15.0)	<0.001
Age (years)	63.5(51.0–76.5)[c]	53.0(42.0–66.0)[c]	59.0(38.0–78.0)[c]	44.0(37.0–54.0)	<0.001
Comorbidity score	0(0–0)	0(0–1.0)[c]	0(0–2.0)	0(0–0)	0.005
Etiology, n (%)					
Hypertriglyceridemic	4(17.4)	18(18.4)	0(0)	25(18.8)	0.001
Biliary	10(43.5)	30(30.6)	4(26.7)	25(18.8)	
Alcoholic	3(13.0)	15(15.3)[c]	6(40.0)	54(40.6)	
Other	6(26.1)	35(35.7)	5(33.3)	29(21.8)	
PMA(cm^2)	7.80(6.84–9.10)[a,b,c]	12.04(10.01–14.24)[c]	14.00(10.04–16.77)[c]	20.59(16.88–24.14)	<0.001

(*Continued*)

Table 1 (Continued).

Characteristic	Malnourished Obese	Well-Nourished Obese	Malnourished non-Obese	Well-Nourished non-Obese	p
BMI(kg/m²)	22.26(19.86–26.01)[a,c]	26.57(24.03–28.52)[b]	17.96(16.96–18.38)[c]	25.74(23.66–28.34)	<0.001
PMA/BMI	0.34(0.30–0.44)[b,c]	0.45(0.40–0.54)[b,c]	0.75(0.55–0.83)	0.76(0.66–0.91)	<0.001
CRP(mg/L)	130.08(3.48–224.24)[b]	74.56(14.76–134.84)	12.21(1.73–15.88)	63.48(13.27–138.76)	0.029
Albumin (g/L)	36.37(33.06–42.10)[c]	41.95(36.80–43.87)[c]	41.96(37.77–44.87)	43.14(39.02–45.46)	<0.001
Leukocyte(10^9/L)	10.07(7.32–11.83)[c]	11.69(9.20–15.41)[b]	8.45(7.31–9.15)[c]	12.37(9.62–15.25)	<0.001
Hemoglobin (g/L)	131.0(118.5–144.0)[c]	146.0(134.0–158.0)[c]	154.0(135.0–164.0)	159.0(141.0–171.0)	<0.001
Blood glucose (mmol/L)	7.63(5.64–11.44)	8.55(7.31–11.61)	7.08(5.88–8.76)	7.76(6.56–10.55)	0.021
Triglycerides (mmol/L)	1.33(1.01–3.42)	2.30(1.39–6.44)[b]	1.01(0.92–1.46)[c]	3.66(1.23–8.59)	0.002
CsCa (mmol/L)	2.20(2.02–2.22)[b]	2.20(2.08–2.26)	2.24(2.23–2.32)[c]	2.18(2.12–2.24)	0.013
Creatinine (μmol/L)	59.40(51.45–103.05)	56.20(44.00–76.40)	63.70(58.90–72.50)	60.85(51.70–74.90)	0.232
Urea (mmol/L)	6.04(3.32–9.20)	5.11(4.20–6.77)	5.28(3.85–6.86)	4.66(3.99–5.88)	0.050
Amylase(U/L)	379.65(151.75–704.40)	475.40(150.00–1171.40)	417.00(337.60–678.80)	268.20(119.40–799.20)	0.070
Lipase(U/L)	1452.40(354.48–2939.85)	2000.00(520.73–5984.65)	2404.00(1836.00–3699.00)	1383.01(474.16–4415.00)	0.080

Notes: Compare each group with one or more groups on the right. p-value < 0.05 for comparison against a Well-nourished obese, b Malnourished non-obese, c Well-nourished non-obese. In order to keep the table tidy, the group on the right has not been repeated with superscripts.
Abbreviations: BMI, body mass index; CRP, C-reactive protein; CsCa, corrected serum calcium; GLIM, Global Leadership Initiative on Malnutrition; PMA, psoas muscle area.

Consistent results were observed when applying the PMA/BMI cutoff of ≥2.93 (median value, Table S1). However, significant variations emerged when using BMI-based obesity classification (Table S2): 1) The WO group became predominant (n=152, 63.3%) while MO group decreased substantially (n=8, 3.3%); 2) The MO group reached the sarcopenia threshold from our previous studies with the lowest PMA, contrasting with WO group showing the highest PMA; 3) The MO group had the lowest levels of albumin and hemoglobin, but only showed statistical differences compared to the WO group; The WO group had the highest levels of these two items among the four groups, with statistically significant differences in hemoglobin levels compared to the other three groups.

Intergroup Comparison of Prognostic Outcomes Grouped According to Obesity and GLIM Criteria

According to the grouping of PMA/BMI (cutoff ≥ 3.05) and GLIM criteria, the MO group demonstrated the highest incidence rates of SAP, infectious complications, local complications, organ failure, and composite outcomes among the four groups, with all parameters showing statistically significant differences compared to the WN group. Although LOS was longest in the MO group, there were no significant differences compared to other groups. Notably, the WO group exhibited significantly higher SAP incidence, systemic complications, and prolonged LOS compared to the WN group. However, there were no significant differences between MN and WN groups across any outcomes (Table 2).

Table 2 Prognostic Outcomes of AP Patients Grouped According to Obesity (PMA/BMI ≥3.05) and GLIM Criteria

Outcomes	Malnourished Obese	Well-Nourished obese	Malnourished non-Obese	Well-Nourished non-Obese	p
Total n (%, N = 240)	20(8.3)	89(37.1)	13(5.4)	118(49.2)	
AP severity					
Mild AP	10(43.5)[b,c]	61(62.2)[c]	14(93.3)	105(78.9)	0.001
Moderately severe AP	8(34.8)	23(23.5)	1(6.7)	23(17.3)	
Severe AP	5(21.7)[c]	14(14.3)[c]	0(0)	5(3.8)	
Infectious complications	5(21.7)[c]	10(10.2)	0(0)	3(2.3)	0.002
Local complications	12(52.2)[a,b,c]	21(21.4)	1(6.7)	13(9.8)	<0.001
Organ failure	8(34.8)[c]	21(21.4)	0(0)	16(12.0)	0.007

(*Continued*)

Table 2 (Continued).

Outcomes	Malnourished Obese	Well-Nourished obese	Malnourished non-Obese	Well-Nourished non-Obese	p
Systemic complications	5(21.7)[c]	15(15.3)[c]	0(0)	5(3.8)	0.002
Composite outcomes	13(56.5)[b,c]	36(36.7)	1(6.7)	28(21.1)	<0.001
Length of stay, days	12.5(8.0–15.5)	10.0(7.0–14.0)[c]	7.0(4.0–9.0)	9.0(6.0–11.0)	0.023
Hospitalization costs, yuan	13623.96 (9649.38–21,981.49)[c]	10678.38 (7168.04–18,316.44)[c]	10910.59 (5607.58–13,655.16)	8495.90 (6207.89–12,541.02)	0.001

Notes: Compare each group with one or more groups on the right. p-value < 0.05 for comparison against a Well-nourished obese, b Malnourished non-obese, c Well-nourished non-obese. In order to keep the table tidy, the group on the right has not been repeated with superscripts.
Abbreviations: AP, acute pancreatitis; BMI, body mass index; GLIM, Global Leadership Initiative on Malnutrition; PMA, psoas muscle area.

When applying an alternative PMA/BMI cutoff≥2.93 (Table S3), the overall findings remained consistent except for the disappearance of statistical significance between WO and WN groups. Substitution with BMI classification (Table S4) revealed only significant intergroup differences in infectious and local complications, though without demonstrating statistical significance between MO and WN groups for infectious complications.

Relationship Between Obesity Combined with Malnutrition Subgroups and Clinical Outcomes

Because the rates of complications were low, we mainly analyzed the MSAP+SAP and composite outcomes. Sarcopenic obesity (MO and WO), regardless of nutritional status, demonstrated significant associations with elevated risks of MSAP+SAP (OR >2.74, p ≤0.008) and composite outcomes (OR >2.69, p ≤0.009). Notably, the MO group exhibited twice the risk of these clinical outcomes compared to the WO group. In contrast, no such associations were observed in BMI-defined obesity groups. Malnutrition alone (MN) did not increase risks of MSAP+SAP or composite outcomes, suggesting that malnutrition acquires clinical relevance only when coexisting with sarcopenic obesity. (Tables 3 and 4)

Table 3 Relationship Between Obesity Combined with Malnutrition Grouping and MSAP+SAP

Subgroup	Multivariate OR (95% CI)	p-value
Combined subgroups of BMI and GLIM		
Well-nourished non-obese	Reference	
Malnourished non-obese	1.09(0.37–3.24)	0.875
Well-nourished obese	1.05(0.52–2.12)	0.885
Malnourished obese	4.90(0.97–24.75)	0.054
Combined subgroups of PMA/BMI (≥ 2.93) and GLIM		
Well-nourished non-obese	Reference	
Malnourished non-obese	0.40(0.04–3.60)	0.411
Well-nourished obese	2.74(1.30–5.79)	0.008
Malnourished obese	5.26(1.82–15.21)	0.002
Combined subgroups of PMA/BMI (≥ 3.05) and GLIM		
Well-nourished non-obese	Reference	
Malnourished non-obese	0.38(0.04–3.34)	0.381
Well-nourished obese	3.48(1.62–7.50)	0.001
Malnourished obese	6.70(2.24–20.04)	0.001

Notes: Adjusted for sex, age, etiology, co-morbidity score, and corrected serum calcium.
Abbreviations: BMI, body mass index; GLIM, Global Leadership Initiative on Malnutrition; MSAP, moderately severe acute pancreatitis; PMA, psoas muscle area; SAP, severe acute pancreatitis.

Table 4 Relationship Between Obesity Combined with Malnutrition Grouping and Composite Outcomes

Subgroup	Multivariate OR (95% CI)	p-value
Combined subgroups of BMI and GLIM		
Well-nourished non-obese	Reference	
Malnourished non-obese	1.08(0.36–3.21)	0.888
Well-nourished obese	1.03(0.51–2.06)	0.944
Malnourished obese	4.99(0.99–25.11)	0.051
Combined subgroups of PMA/BMI (≥ 2.93) and GLIM		
Well-nourished non-obese	Reference	
Malnourished non-obese	0.39(0.04–3.56)	0.406
Well-nourished obese	2.69(1.27–5.68)	0.009
Malnourished obese	5.34(1.85–15.45)	0.002
Combined subgroups of PMA/BMI (≥ 3.05) and GLIM		
Well-nourished non-obese	Reference	
Malnourished non-obese	0.37(0.04–3.29)	0.375
Well-nourished obese	3.37(1.56–7.27)	0.002
Malnourished obese	6.76(2.26–20.23)	0.001

Notes: Adjusted for sex, age, etiology, co-morbidity score, and corrected serum calcium.
Abbreviations: BMI, body mass index; GLIM, Global Leadership Initiative on Malnutrition; PMA, psoas muscle area.

Similar results were obtained using Firth's logistic regression, except that the WO group lost correlation with adverse clinical outcomes when using PMA/BMI ≥ 2.93 as the criterion for sarcopenic obesity (Tables S5 and S6).

Relationship Between Different PMA/BMI or BMI Levels Alone and Clinical Outcomes

As the PMA/BMI quartiles (<2.34,2.34–2.92, 2.93–3.84, >3.84) increase, the risk of MSAP+SAP and composite outcomes significantly increases (p for trend=0.003 and 0.002, respectively). Groups above the median PMA/BMI (sarcopenic obesity) were significantly associated with MSAP+SAP (OR ≥3.41, p ≤0.011) and composite outcomes (OR ≥3.26, p ≤0.015). The highest quartile group exhibited particularly pronounced risks, with over 7-fold increases for both MSAP+SAP and composite outcomes (all OR >7.0, p ≤0.001). However, neither these associations nor the trend significance were observed in BMI-based classifications. No significant interactions were detected between PMA/BMI or BMI and GLIM-defined malnutrition. (Table 5) Similar results were obtained using Firth's logistic regression, except that the Q2 ≤ PMA/BMI<Q3 subgroup lost correlation with the composite outcome (Table S7).

Table 5 Relationship Between PMA/BMI and Various Outcomes

Outcomes	Subgroup	Multivariate OR (95% CI)	p-value	p for Trend	p Interaction with GLIM
MSAP+SAP	PMA/BMI<Q1	Reference		0.003	0.608
	Q1≤PMA/BMI<Q2	1.52(0.60–3.80)	0.376		
	Q2≤PMA/BMI<Q3	3.41(1.32–8.79)	0.011		
	PMA/BMI≥Q3	7.05(2.34–21.27)	0.001		
Composite outcomes	PMA/BMI<Q1	Reference		0.002	0.614
	Q1≤PMA/BMI<Q2	1.51(0.60–3.78)	0.381		
	Q2≤PMA/BMI<Q3	3.26(1.26–8.40)	0.015		
	PMA/BMI≥Q3	7.43(2.45–22.50)	<0.001		

(*Continued*)

Table 5 (Continued).

Outcomes	Subgroup	Multivariate OR (95% CI)	p-value	p for Trend	p Interaction with GLIM
MSAP+SAP	BMI<18.5 kg/m² 18.5kg/m²≤BMI<24 kg/m² 24 kg/m² ≤ BMI < 27.9 kg/m² BMI ≥ 28 kg/m²	Reference 3.14(0.59–16.80) 3.52(0.62–19.96) 4.24(0.72–24.96)	 0.182 0.156 0.111	0.460	0.309
Composite outcomes	BMI<18.5 kg/m² 18.5kg/m²≤BMI<24 kg/m² 24 kg/m² ≤ BMI < 27.9 kg/m² BMI ≥ 28 kg/m²	Reference 3.23(0.60–17.25) 3.49(0.62–19.78) 4.38(0.75–25.78)	 0.171 0.157 0.102	0.435	0.301

Notes: Adjusted for sex, age, etiology, co-morbidity score, and corrected serum calcium.
Abbreviations: BMI, body mass index; GLIM, Global Leadership Initiative on Malnutrition; MSAP, moderately severe acute pancreatitis; PMA, psoas muscle area; SAP, severe acute pancreatitis.

Relationship Between Obesity and Malnutrition and the Severity of AP

When analyzing obesity separately, there was an overall trend of increased risk of AP severity with increasing BMI (p trend=0.012), but the individual analysis of each group did not reach statistical significance. Conversely, rising PMA/BMI ratios demonstrated a steep escalation in AP severity risk (p for trend<0.001), particularly in the highest quartile group (OR=8.7, p<0.001). Sarcopenic obesity subgroups (regardless of nutritional status) consistently showed significant associations with AP severity, with the MO group exhibiting the highest risk. In BMI-GLIM combined analyses, potential risk elevation for AP severity was observed only when malnutrition coexisted with overweight/obesity (p approaching 0.05), though statistical evidence remained inconclusive. (Table 6) Similar results were obtained using Firth's logistic regression. However, in the two subgroups using PMA/BMI combined with GLIM, due to the fact that the number of severe AP cases is 0, the information matrix cannot be uniquely identified, and therefore the OR value cannot be obtained (Table S8).

Table 6 Relationship Between Subgroups and AP Severity

Subgroup	Multivariate OR (95% CI)	p-value	p for trend	p Interaction with GLIM
Subgroups of BMI BMI<18.5 kg/m² 18.5kg/m²≤BMI<24 kg/m² 24 kg/m² ≤ BMI < 27.9 kg/m² BMI ≥ 28 kg/m²	 Reference 2.52(0.52–12.35) 3.18(0.62–16.43) 3.78(0.70–20.35)	 0.253 0.167 0.121	0.012	0.585
Subgroups of PMA/BMI PMA/BMI<Q1 Q1≤PMA/BMI<Q2 Q2≤PMA/BMI<Q3 PMA/BMI≥Q3	 Reference 1.41(0.57–3.48) 3.66(1.47–9.12) 8.70(2.99–25.33)	 0.456 0.005 <0.001	<0.001	0.595
Combined subgroups of BMI and GLIM Well-nourished non-obese Malnourished non-obese Well-nourished obese Malnourished obese	 Reference 1.02(0.36–2.90) 1.18(0.59–2.36) 4.10(0.98–17.17)	 0.978 0.647 0.054	-	-

(*Continued*)

Table 6 (Continued).

Subgroup	Multivariate OR (95% CI)	p-value	p for trend	p Interaction with GLIM
Combined subgroups of PMA/BMI (≥ 2.93) and GLIM			-	-
Well-nourished non-obese	Reference			
Malnourished non-obese	0.39(0.05–3.36)	0.391		
Well-nourished obese	3.27(1.59–6.73)	0.001		
Malnourished obese	5.24(1.93–14.27)	0.001		
Combined subgroups of PMA/BMI (≥ 3.05) and GLIM			-	-
Well-nourished non-obese	Reference			
Malnourished non-obese	0.38(0.04–3.24)	0.378		
Well-nourished obese	4.54(2.16–9.56)	<0.001		
Malnourished obese	6.97(2.49–19.51)	<0.001		

Notes: Adjusted for sex, age, etiology, co-morbidity score, and corrected serum calcium.
Abbreviations: AP, acute pancreatitis; BMI, body mass index; GLIM, Global Leadership Initiative on Malnutrition; PMA, psoas muscle area.

Discussion

This study aimed to elucidate the relationships between different obesity definitions, nutritional status, and clinical outcomes in AP patients. Our findings highlight the pivotal role of malnutrition combined with sarcopenic obesity in determining AP prognosis. The PMA/BMI-based classification demonstrated superior performance over BMI alone in identifying high-risk patients, with a PMA/BMI-dependent increase in adverse outcome risks. The MO group exhibited the highest inflammatory markers (CRP), lowest nutritional indices (hemoglobin, albumin), and worst clinical outcomes, showing significantly higher rates of adverse events compared to MN and WO groups. In contrast, WN individuals displayed the most favorable metabolic profiles and clinical outcomes, suggesting a synergistic effect between malnutrition and sarcopenic obesity. Notably, conventional BMI classification failed to effectively stratify high-risk populations.

Published Studies on the Association Between Overweight/Obesity or Malnutrition and the Prognosis of AP

Previous investigations have primarily focused on isolated associations of overweight/obesity or malnutrition with AP prognosis. A meta-analysis revealed that BMI >25 kg/m² increases the risk of SAP, while BMI >30 kg/m² or <18.5 kg/m² elevates mortality risk.[10] Another meta-analysis identified obesity as an independent risk factor for organ failure development, though not associated with mortality or pancreatic necrosis.[16] Contrary to these findings, a US study reported higher mortality rates and pancreatic complications in normal-weight AP patients compared to overweight/obese counterparts.[17] These conflicting results underscore the limitations of BMI as a sole obesity metric. Emerging evidence suggests visceral adiposity may outperform BMI in predicting pancreatitis severity.[18–21]

Alternative anthropometric measures have shown similar predictive utility. Jean et al identified waist circumference as an independent risk factor for systemic inflammatory response syndrome and organ failure,[22] while Xia et al developed a visceral obesity index incorporating waist circumference, BMI, triglycerides, and HDL-cholesterol for severity assessment in hyperlipidemic AP.[23] Particularly relevant to our findings, Thomas et al and Robert et al reported associations between sarcopenic obesity and severe AP outcomes, including mortality.[13,24]

Body composition analysis through CT imaging has yielded inconsistent results. Our previous work established PMA as a reliable predictor of AP severity,[7] whereas Wang et al and Pedro et al found skeletal muscle density and intermuscular adipose tissue but not muscle area or visceral fat correlated with prognosis respectively.[25,26] Roberto et al identified peritoneal cavity circumference as the sole independent predictor among various abdominal CT parameters.[27] Contrary to the above, Chen et al found that neither area and radiodensity of muscle nor area and radiodensity of visceral adipose tissue obtained by CT at the level of the third lumbar vertebrae were associated with the severity of hypertriglyceridemia-induced pancreatitis.[28]

Notably, current literature lacks investigations into adult malnutrition-AP outcome relationships, particularly the combined effects of malnutrition with obesity or sarcopenic obesity. Our study addresses this critical knowledge gap by

demonstrating the clinical significance of malnutrition-sarcopenic obesity comorbidity in AP prognosis, proposing PMA/BMI as a superior risk stratification tool compared to traditional BMI classification.

The Impact of Obesity Definition on Risk Stratification: Clinical Advantages of PMA/BMI

Our study demonstrates that sarcopenic obesity, as defined by the PMA/BMI ratio (regardless of cutoff thresholds), exhibited superior predictive capability for disease risk and prognosis compared to traditional BMI-based classifications. Evidence suggested that sarcopenic obese patients face 1.5–2 times higher all-cause mortality than those with isolated obesity or sarcopenia alone.[29] In cardiovascular populations, this condition increased heart failure hospitalization risk by 40%, while serving as an independent predictor of postoperative mortality (HR=2.1–3.4) in chronic kidney disease and hepatocellular carcinoma patients - outcomes unattainable through BMI-defined obesity.[30] Similarly, colorectal and pancreatic cancer patients with sarcopenic obesity experienced heightened chemotherapy toxicity (OR=1.8) and reduced survival (HR=1.6), risks undetectable through BMI or body fat percentage assessments.[29] In terms of metabolic diseases, the insulin resistance and the risk of type 2 diabetes in sarcopenic obese patients were higher than those in obese patients with BMI matching.[31,32] In addition, the sarcopenic obese population also showed elevated risks of cognitive decline (HR=1.60) and severe COVID-19 outcomes (OR=2.88).[32,33] Conversely, BMI-based studies showed nonsignificant associations[34] or paradoxical relationships with clinical complications.[17]

The PMA/BMI stratification system not only enhances the identification of malnutrition-obesity (MO) patients but also reveals strong correlations with AP severity, complications, and poor prognosis. This diagnostic superiority stems from BMI's inherent limitations in distinguishing muscle from adipose mass,[35,36] which leads to misclassification of muscular individuals as overweight/obese (supported by the WO group's significantly higher PMA) while underestimating true sarcopenic obesity. PMA/BMI provides a better explanation for the association between sarcopenic obesity and disease prognosis by quantifying the muscle fat ratio. Our modified PMA/BMI formula, adapted from Thomas et al who demonstrated its independent association with severe pancreatitis (OR=1.455, 95% CI [1.028–2.061]; p=0.035),[13] showed consistent clinical relevance across different cutoffs. Though limited by insufficient SAP cases for definitive analysis, we identified significant associations between PMA/BMI and both MSAP+SAP development (OR≥3.26, p≤0.015) and composite outcomes, with consistent results across multiple thresholds effectively serving as sensitivity analyses.

The Interaction Effect Between Malnutrition and Obesity

The MO group demonstrated distinct clinical characteristics, including the oldest age (nearly 20 years older than the WN group), the highest inflammatory markers (CRP), and the lowest nutritional indices (albumin, hemoglobin). While MN and WN groups showed comparable clinical outcomes, suggesting that malnutrition significantly impacts prognosis only when combined with sarcopenic obesity. This implies potential synergistic effects between malnutrition and obesity, though our data revealed no statistically significant interaction (p-interaction >0.3). This may reflect sample size limitations, where low adverse outcome incidence in subgroup analyses reduced statistical power for interaction testing, necessitating validation in larger cohorts.

The coexistence of malnutrition and sarcopenic obesity likely exacerbates AP progression through multiple interrelated mechanisms. The hypoalbuminemia and elevated CRP indicate a vicious cycle of protein-energy wasting and systemic inflammation that may promote pancreatic necrosis and infection. The established pathophysiology of obesity in AP involves pancreatic triglyceride lipase release into visceral adipose tissue, where it generates excessive non-esterified fatty acids (NEFA). These NEFA compounds induce cellular damage through mitochondrial dysfunction, apoptosis, and necrosis in pancreatic acinar cells, renal tubules, and pulmonary tissue. Simultaneously, NEFA stimulates proinflammatory cytokine production (monocyte chemotactic protein-1, tumor necrosis factor-α, interleukin-6), driving systemic inflammation and multiorgan failure (such as acute respiratory distress syndrome, acute kidney injury, etc).[37] And malnutrition itself often has acute and chronic inflammation, which exacerbates the systemic inflammatory response in obese AP patients. At the same time, the above process can also cause insulin resistance, leading to the accumulation of branched-chain amino acids and lactate, inhibiting muscle protein synthesis, which exacerbates muscle loss and

malnutrition.[31] On the other hand, malnutrition and obesity lead to a decrease in gut microbiota diversity, a reduction in short chain fatty acid (SCFA) production, and an increase in protein fermentation products (such as indole sulfates), exacerbating systemic inflammation and inhibiting muscle cell regeneration by upregulating muscle atrophy related genes (atrogin-1, myostatin).[31,32] These cascading effects worsen pancreatic microcirculatory dysfunction, while respiratory muscle wasting and immune impairment heighten organ failure risks.

Malnutrition alone (MN group) did not show an increased risk, which may be due to the following reasons. First, the small sample size in the MN group resulted in a low incidence of adverse outcomes. Second, regardless of the grouping method used, the MO group had the lowest PMA, while the MN group had the lowest BMI and inflammatory markers (CRP). Additionally, the nutritional indicators (albumin, hemoglobin) in the MN group were not significantly different from those in the WN group. This suggests that low BMI may reflect chronic energy deficiency with relatively stable metabolic status, and its pathophysiological mechanisms may be independent of obesity-related inflammatory pathways.[38]

Our finding that clinical significance emerges only when malnutrition coexists with sarcopenic obesity supports the revised "obesity paradox" concept - the prognostic impact of obesity critically depends on body composition profiles.[39] Previous studies have also indicated that sarcopenic obesity may be associated with higher levels of metabolic dysfunction and increased mortality risk compared to obesity or sarcopenia alone.[40] Sarcopenic obesity represents more than simple co-occurrence of adiposity and muscle loss; it establishes a self-perpetuating cycle through energy metabolism dysregulation and inflammatory crosstalk, substantially amplifying disease severity. This pathophysiological synergy underscores the necessity of comprehensive body composition assessment in clinical management and prognostic evaluation of AP patients.

Clinical Insights: Precise Intervention for Malnutrition Combined with Sarcopenia Obesity

For AP patients with comorbid malnutrition and sarcopenic obesity, intensified early enteral nutritional support is critical to improve prognosis and reduce healthcare costs. In patients tolerating oral intake or enteral nutrition, a regimen combining moderate caloric restriction with protein supplementation is recommended, alongside adequate fiber, SCFA, vitamins, and trace elements. Daily caloric intake should be moderately restricted (by 500 kcal/day), while protein intake should be increased to 1.0–1.2 g/kg body weight. Further elevation to 1.4 g/kg may significantly preserve lean mass, with whey protein prioritized for its superior stimulation of myofibrillar protein synthesis.[29,41] Fermentable fibers (eg, galactooligosaccharides, inulin, and resistant starch blends) should be incorporated to enhance distal colonic SCFA production (eg, acetate), thereby improving metabolic health.[31] Adjunctive supplementation with vitamin D, selenium, and magnesium may optimize muscle function, while combined vitamin D and leucine administration may synergistically augment muscle protein synthesis.[29,41]

Limitations

This study has several constraints inherent to its single-center retrospective design, including potential selection bias. Key inflammatory biomarkers and body composition assessments via bioelectrical impedance analysis or waist circumference measurements were unavailable due to either excessive missing data or institutional testing limitations. As a secondary analysis of prior research, abdominal adiposity quantification via CT was also unavailable. Our study did not obtain a clear cutoff value for PMA/BMI diagnosis of sarcopenic obesity, but our sensitivity analysis demonstrated that a cutoff value within the range of 2.93–3.05 may be appropriate. Furthermore, extremely small malnutrition subgroups undermined statistical reliability: eg,0 organ failure in MN group may due to type II error rather than true biological effect, and the effect sizes in MO group were unstable.

Conclusion

This study systematically elucidates the critical impact of obesity definition methodologies on risk stratification and prognostic evaluation in AP patients, identifying PMA/BMI as a superior metric for sarcopenic obesity that outperforms BMI in detecting high-risk subgroups. We observed that patients with combined malnutrition and sarcopenic obesity exhibited the highest inflammatory burden, poorest nutritional markers, and worst clinical outcomes, suggesting potential synergistic pathophysiological interactions. However, due to the limited number of malnourished patients, the above

conclusion still needs to be validated in larger scale studies. Furthermore, large-scale prospective multicenter studies are warranted to establish optimal CT-based diagnostic thresholds for sarcopenic obesity and validate the mechanistic interplay between malnutrition and sarcopenic obesity in AP progression. These findings advocate for body composition-guided nutritional interventions to refine clinical management and improve outcomes in this vulnerable population.

Abbreviations

AP, acute pancreatitis; BMI, body mass index; BSA, body surface area; CRP, C-reactive protein; CsCa, corrected serum calcium; CT, Computer Tomography; GLIM, Global Leadership Initiative on Malnutrition; LOS, length of stay; MAP, mild acute pancreatitis; MN, malnourished non-obese; MO, malnourished obese; MSAP, moderately severe acute pancreatitis; NEFA, non-esterified fatty acids; NRS2002, Nutritional Risk Screening 2002; PMA, psoas muscle area; ROC, receiver operating characteristic; SAP, severe acute pancreatitis; SCFA, short chain fatty acid; WN, well-nourished non-obese; WO, well-nourished obese.

Ethics Statement

The study was in line with the principles of the Declaration of Helsinki and approved by the Ethics Committee of the Affiliated Hospital of Chengde Medical University (No. CYFYLL2022256). Due to the retrospective nature of the study, the Ethics Committee waived the requirement for written informed consent. Prior to analysis, identifying information was removed to protect patient confidentiality.

Author Contributions

All authors made substantial contributions to the conception and design, acquisition of data, or analysis and interpretation of data; took part in drafting, revising or critically reviewing the article; have agreed on the journal to which the article has been submitted; gave final approval of the version to be published; and agree to be accountable for all aspects of the work.

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Disclosure

The authors report no conflicts of interest in this work.

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