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Impact of sarcopenic obesity on predicting the severity of acute pancreatitis

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ABSTRACT

Background and aims: This work aimed to evaluate the impact of sarcopenia and sarcopenic obesity on the occurrence of severe pancreatitis and to study the performance of anthropometric indices to predict severe forms.

Methods: We conducted a single-center retrospective study at Caen University Hospital between 2014 and 2017. Sarcopenia was assessed by measuring the psoas area on an abdominal scan. The psoas area /body mass index ratio reflected sarcopenic obesity. By normalizing the value to the body surface, we obtained an index called sarcopancreatic index, avoiding sex differences in measurements.

Results: Among 467 included patients, 65 (13.9%) developed severe pancreatitis. The sarcopancreatic index was independently associated with the occurrence of severe pancreatitis (1.455 95% CI [1.028–2.061]; $p = 0.035$), as was the Visual Analog Scale, creatinine or albumin. The complication rate was not different depending on sarcopancreatic index value. Based on variables independently associated with the occurrence of severe pancreatitis, we constructed a score called Sarcopenia Severity Index. This score presented an area under the receiver operating characteristics curve of 0.84, comparable to the Ranson score (0.87) and superior to body mass index or the sarcopancreatic index to predict a severe form of acute pancreatitis.

Conclusions: Sarcopenic obesity seems to be associated with severe acute pancreatitis.

1. Introduction

Acute pancreatitis (AP) is one of the most common gastrointestinal causes of hospitalization in the world [1]. AP typically presents in its benign form as abdominal pain, which is quickly favorable with symptomatic treatment. However, 20% of patients can evolve severe AP, with infected necrosis or organ failure, requiring admission to an intensive care unit [2]. Mortality for severe AP is 15 to 20% and can reach up to 50%, while overall mortality is less than 5% [3–6].

The 1992 Atlanta classification revised in 2012 as well as the 2012 international classification define severe AP based on the presence of persistent organ failure and/or infected necrosis [7,8]. These classifications are statistically correlated with clinical prognosis, but they only classify AP once complications have occurred. In clinical situations, it is important to identify the factors associated with the development of a severe form as early as possible. Several clinico-biological scores (Ranson, APACHE-II, BISAP or the existence of systemic inflammatory response syndrome (SIRS)) or radiological scores (Balthazar score or CTSI) have demonstrated equivalent performance in predicting disease severity [9]. Given their lack of specificity or complexity of use, the American Guidelines of 2018 recommend a global approach including age, nutritional status, assessment of comorbidities, and presence of hemodynamic, biological or radiological disorders rather than the use of these scores [10]. A better understanding of the factors creating a predisposition for the development of a severe form remains a major issue.

Nutritional status influences the prognosis of many diseases. In AP, it has been shown that obesity, defined by a body mass index (BMI) greater than or equal to 30 kg/m^2, is associated with a more frequent occurrence of severe forms [11]. In contrast, there are limited data on the influence of malnutrition and sarcopenia in AP.

Sarcopenia is known to be an independent factor of unfavorable outcomes in many digestive diseases [12–16], especially in chronic pancreatitis. Studies have shown that patients with sarcopenia had a 2-fold increased risk of hospitalization (all causes) over one year of follow-up and a 6-fold increased risk of death at one year [17].

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To our knowledge, only a few studies have investigated the influence of sarcopenia or sarcopenic obesity on the prognosis of AP, with conflicting conclusions [18,19].

The aim of this study was to evaluate the influence of sarcopenia and /or sarcopenic obesity on the occurrence of a severe form of acute pancreatitis and to measure the performance of anthropometric indices to predict these severe forms.

2. Materials and methods

2.1. Study design and patients

We performed a retrospective study at the University Hospital Center of Caen. We used clinico-biological data from a cohort of patients hospitalized for AP at our center between January 2014 and December 2017 [20]. Patients were identified using coding K85.9 corresponding to the ICD-10 (International Classification of Diseases) diagnosis of AP. The diagnosis of AP was defined, according to the criteria used by the 1992 Atlanta classification revised in 2012, by the presence of at least two of the following three features: typical abdominal pain, serum lipase activity at least three times greater than the upper limit of normal, or characteristic findings of AP on contrast-enhanced CT [7]. Patients who were transferred from another hospital to the University Hospital of Caen more than 24 h after the beginning of their management were excluded, as were patients with iatrogenic pancreatitis (post-Endoscopic retrograde cholangiopancreatography or after radiological biliary drainage) and those for whom the data did not allow us to evaluate the severity.

Among 504 patients from the cohort, 20 patients did not have a CT during their hospitalization, and 17 had a CT in another center without the ability to collect the CT [20]. Finally, we were able to include 467 patients for whom measurement of the psoas muscle area was possible.

2.2. Assessment of sarcopenia

In our cohort of patients, we retrospectively determined the existence of sarcopenia by measuring the psoas muscle area in mm^2 on a CT of the L3 vertebra when both pedicles were visible, with a density threshold set between −29 and +150 Hounsfield Units (HU). Then, the areas were summed and divided by the patient's height squared (mm^2/m^2) to obtain the Total Psoas Area (TPA) or summed and divided by the patient's BMI to obtain the psoas area to BMI ratio (PA/BMI) which can reflect sarcopenic obesity. To avoid sex differences in measurements, this PA/BMI index was then related to the body surface area according to the following formula: $\sqrt{}$(10 000 x body surface area)/(PA/BMI). We named this index the "Sarcopancreatic Index". Measurement of the psoas muscles was performed manually by a nonradiologist clinician using Aquarius iNtuition software (TeraRecon Inc., San Mateo, CA, USA). The CT used for measurements was the earliest CT performed after the onset of pain during hospitalization.

2.3. Collected data

We collected clinical and biological data within 24 h of patient admission as well as data related to the severity and etiology of AP. Clinical data included sex, age, BMI, pain assessment with Visual Analog Scale (VAS), heart rate, Mean Arterial Pressure (MAP), temperature, oxygen saturation, and Glasgow score. Biological data included hematocrit, leukocyte and platelet counts, albumin, C-reactive protein (CRP), blood glucose, total calcemia, natremia, bicarbonate level, blood urea, blood creatinine, creatinine clearance, lipase and total bilirubin.

The etiology of AP was classified into three categories: alcoholic, biliary, and other. We collected the data needed to calculate the presence of SIRS. The presence of necrosis was assessed using the Balthazar score. We also recorded the occurrence of infected necrosis (defined as the identification of bacteria on bacteriological examination of necrotic collections), length of hospital stay, transfer to intensive care, presence of organ failure on admission and during the course of management, and mortality rate. The severity of AP was defined according to 2013 international guidelines criteria as the presence of infected necrosis or persistent organ failure beyond 48 h, defined by a modified Marshall score greater than or equal to 2 [21]. The cases of severe acute pancreatitis in our study corresponded to patients who developed one of these complications during hospitalization. The research protocol was approved by the local ethics committee of the Caen University Hospital.

2.4. Statistical analysis

Patient characteristics were expressed as numbers and percentages for categorical variables and as the means with their standard deviations for quantitative variables. We performed a univariate analysis to compare patients with a severe form to those with a nonsevere form. We compared qualitative variables by a Chi Squared test (or Fisher's exact test in the case of a theoretical number <5) and quantitative variables by Student's or Wilcoxon-Mann–Whitney test. Multivariate analysis by sequential logistic regression was performed to identify variables independently associated with the occurrence of a severe form of AP. Variables included were those that were associated with severity with a $p < 0.1$ in univariate analysis and clinically relevant.

Then, we created a score named the Sarcopenia Severity Index (SSI) from the regression coefficients of the variables associated with the occurrence of a severe form of AP in multivariate analysis. The threshold with the best performance of this score was identified using the highest value of the Youden index (sensitivity + specificity - 1). We then compared the performances of different prognostic scores by comparing the areas under the ROC curve (AUROC). The significance level was set at 0.05. Statistical analyses were performed using SAS 9.2 software (Cary, NY, USA).

3. Results

3.1. Population

Among the 467 patients included, the majority were men (66.2%) with a mean age of 57 years. One hundred sixteen patients (23.1%) were obese. The most frequent etiology was biliary (40.9%), followed by ethylic origin (32.1%). A total of 186 patients had a necrotic form (39.8%) and 141 patients (30.2%) had SIRS at admission. In our population, 65 patients (13.9%) developed a severe form of AP, of which 53 (11.3%) were related to persistent organ failure and/or 17 (3.6%) to necrosis infection. The number of deaths was 19, representing a mortality rate of 4.1%. All patients who died had a severe form of acute pancreatitis, representing 29.2% of this population (Table 1).

Table 1 compares the characteristics of the populations who developed or did not develop a severe form of AP. Patients in the severe group were older (64 ± 15 vs. 56 ± 19 years; $p = 0.001$). The severe group had a longer mean length of stay (38.1 ± 58.0 vs. 8.4 ± 26.2 days; $p = 0.0003$) and a higher rate of persistent SIRS at 48 h (63.1% vs. 19.4%; $p<0.001$).

3.2. Population anthropometric parameters

Table 2 shows the anthropometric characteristics of the studied population according to their sex and the severity of AP. In the

Table 1
Patient characteristics and factors associated with the severity of acute pancreatitis; univariate analysis.

	Population $n = 467$	Severe pancreatitis $n = 65$	Nonsevere pancreatitis $n = 402$	p
Sex (Male)	309 (66.2)	50 (76.9)	259 (64.4)	*0.065*
Age (years)	57 ± 19	64 ± 15	56 ± 19	*0.001*
Causes of pancreatitis				*0.269*
Alcoholic	150 (32.1)	18 (27.7)	132 (32.8)	
Biliary	191 (40.9)	24 (36.9)	167 (41.5)	
Other	126 (27.0)	23 (35.4)	103 (25.6)	
Clinical data at admission				
Heart rate (bpm)	86.5 ± 22.1	95.1 ± 25.5	85.1 ± 21.2	*0.001*
MAP (mmHg)	101 ± 17	98 ± 20	102 ± 16	*0.174*
Hypothermia (<36 °C)	45 (9,6)	11 (16,9)	34 (8,5)	*0.03*
Oxygen saturation (%)	97 ± 3	96 ± 4	97 ± 2	*0.020*
Visual Analog Scale	5.0 ± 3.2	5.8 ± 3.4	4.9 ± 3.1	*0.044*
SIRS on admission	141 (30.2)	38 (58.5)	103 (25.6)	*<0.0001*
Laboratory data at admission				
Hematocrit (%)	41.8 ± 5.5	41.6 ± 8.1	41.8 ± 5.0	*0.898*
Leukocytes (G/L)	12.8 ± 5.1	14.1 ± 5.6	12.6 ± 5.0	*0.024*
Platelets (G/L)	249.8 ± 103.6	271.6 ± 150.6	246.3 ± 93.5	*0.532*
Albumin (g/L)	39.1 ± 6.4	34.7 ± 8.3	39.8 ± 5.8	*<0.001*
CRP (mg/L)	64 ± 92	120 ± 130	55 ± 81	*<0.001*
Glycemia (mmol/L)	8.0 ± 5.7	9.6 ± 6.4	7.8 ± 5.6	*0.043*
Calcemia (mmol/L)	2.32 ± 0.20	2.19 ± 0.33	2.34 ± 0.17	*<0.001*
Natremia (mmol/L)	137 ± 4	135 ± 6	137 ± 4	*0.052*
Creatinin (µmol/L)	89 ± 71	167 ± 152	76 ± 31	*<0.001*
Total bilirubin (µmol/L)	34 ± 50	53 ± 94	31 ± 38	*0.001*
Complication				
Persistent SIRS at 48 h	118 (25.5)	41 (63.1)	77 (19.4)	*<0.001*
Necrotic form	186 (39.8)	42 (64.6)	144 (35.8)	*<0.001*
Permanent organ failure	53 (11.3)	53 (81.5)	0 (0.0)	*<0.001*
Necrosis Infection	17 (3.6)	17 (26.2)	0 (0.0)	*<0.001*
Length of hospital stay (days)	12.6 ± 34.0	38.1 ± 58.0	8.4 ± 26.2	*0.0003*
ICU admission	35 (7.5)	32 (49.2)	3 (0.7)	*<0.001*
Death	19 (4.1)	19 (29.2)	0 (0.0)	*<0.001*

MAP: Mean Arterial Pressure; SIRS: Systemic Inflammatory Response Syndrome; CRP: C-Reactive Protein; ICU: Intensive Care Unit.
Values are expressed as number and percent or mean ± standard deviation.

overall population, in both women and men, the mean BMI and mean psoas muscle area were not different between severe and nonsevere patients. However, there were more obese patients in the severe group than in the nonsevere group in the overall population and in men (32.9% vs. 21.4%; $p = 0.015$ and 30.9% vs. 16.8%; $p = 0.009$, respectively).

The TPA was significantly lower in women in the severe group (408.6 ± 128.0 vs. 486.5 ± 122.1 mm^2/m^2; $p = 0.025$). The PA/BMI ratio was significantly lower in women and men in the severe group (37.9 ± 13.9 vs. 47.7 ± 13.5 $mm^2/kg/m^2$; $p = 0.011$ and 68.8 ± 19.1 vs. 76.5 ± 21.3 $mm^2/kg/m^2$; $p = 0.023$, respectively) but was not significantly different in the overall population ($p = 0.164$). The Sarcopancreatic Index freed from any gender difference was significantly higher in the severe group (2.76 ± 1.67 vs. 2.36 ± 0.89; $p = 0.006$).

3.3. Factors independently associated with severe pancreatitis

Multivariate analysis, summarized in Table 3, showed a significant association between the occurrence of a severe form and admission values of Visual Analog Scale (odds ratio (OR) = 1.236 CI [1.082–1.411]; $p = 0.0018$), creatinine (OR = 1.022 CI [1.014–1.030]; $p < 0.0001$), albumin (OR = 0.899 CI [0.851–0.951]; $p = 0.0002$), and Sarcopancreatic Index (OR = 1.455 CI [1.028–2.061]; $p = 0.0346$). The constant term of the multivariate logistic regression was - 2.1808.

3.4. Pancreatitis complication rate depending on sarcopancreatic index

We compared the AUROC values of two anthropometric indices, BMI and Sarcopancreatic Index, with different prognostic scores from the literature (Ranson, BISAP, and persistent SIRS) to predict the occurrence of severe AP (Fig. 1). The AUROC for both of these indices was 0.57, which was significantly lower than the Ranson score of 0.87. The optimal threshold of Sarcopancreatic index to predict the occurrence of a severe form was 2.1827. The disease complication rates were not different in patients with a Sarcopancreatic Index lower or higher than this optimal threshold either for the rate of transfer to Intensive Care Unit (ICU) (10% vs. 5.5%; $p = 0.89$), the rate of necrosis infection (3.3% vs. 4.6%; $p = 0.51$), or the mortality rate (2.4% vs. 4.1%; $p = 0.31$) (Fig. 2).

3.5. Development of a predictive score for the severity of acute pancreatitis

Using the regression coefficients of the variables associated with the occurrence of a severe form of AP in multivariate analysis, we constructed a severity predictive score named the Sarcopenia Severity Index (SSI). This score was calculated according to the following formula: SSI = - 2.1808 + 0.0218*creatinine + 0.2115*VAS - 0.1062*albumin + 0.3751*SSI.

We compared the AUROC of the SSI with those of the different prognostic scores (Ranson, BISAP, and persistent SIRS) and anthropometric indices (BMI and sarcopancreatic index) previously used (Fig. 1). The AUROC of SSI was 0.84. In comparison, the AUROC of Ranson score in our cohort was of 0.87 (Fig. 1). The AUROCs of all other scores were significantly lower than that of the Ranson index. The AUROC of SSI was significantly higher than the AUROC of BMI, sarcopancreatic index and persistent SIRS ($p<0.0001$, $p<0.0001$ and $p = 0.088$, respectively). The Hosmer-Lemeshow test indicated that SSI, Ranson and BISAP demonstrated good calibration (respectively $p = 0.93$; 0.97 and 0.38).

Table 2
Comparison of anthropometric characteristics depending on sex and severity of pancreatitis.

Global population	Total (n = 467)	Severe pancreatitis (n = 65)	Nonsevere pancreatitis (n = 402)	p
Weight (kg)	77.6 ± 18.9	80.5 ± 16.9	77.2 ± 19.2	*0.199*
BMI (kg/m^2)	27.0 ± 6.5	28.1 ± 5.7	26.8 ± 6.6	*0.156*
Obese patients	116 (23.1%)	24 (32.9%)	92 (21.4%)	*0.015*
Psoas area (mm^2)	1714 ± 599	1698 ± 667	1717 ± 588	*0.816*
TPA (mm^2/m^2)	585.2 ± 188.5	582.1 ± 237.0	585.7 ± 179.9	*0.890*
PA/BMI (mm^2/kg/m^2)	65.5 ± 23.2	61.6 ± 22.2	66.1 ± 23.4	*0.164*
Sarcopancreatic Index	2.41 ± 1.05	2.76 ± 1.67	2.36 ± 0.89	*0.006*
Women	**Total (n = 159)**	**Severe pancreatitis (n = 15)**	**Nonsevere pancreatitis (n = 144)**	**p**
Weight (kg)	73.2 ± 18.7	81.2 ± 19.0	72.4 ± 18.6	*0.095*
BMI (kg/m^2)	28.0 ± 7.4	30.3 ± 7.2	27.7 ± 7.4	*0.217*
Obese patients	53 (30.5%)	7 (38.9%)	46 (29.5%)	*0.25*
Psoas area (mm^2)	1253 ± 329	1117 ± 363	1268 ± 324	*0.093*
TPA (mm^2/m^2)	479.3 ± 124.3	408.6 ± 128.0	486.5 ± 122.1	*0.025*
PA/BMI (mm^2/kg/m^2)	46.8 ± 13.7	37.9 ± 13.9	47.7 ± 13.5	*0.011*
Men	**Population (n = 308)**	**Severe pancreatitis (n = 50)**	**Nonsevere pancreatitis (n = 258)**	**p**
Weight (kg)	79.8 ± 18.6	80.3 ± 16.5	79.8 ± 19.1	*0.858*
BMI (kg/m^2)	26.5 ± 5.9	27.4 ± 5.0	26.3 ± 6.1	*0.237*
Obese patients	63 (19.1%)	17 (30.9%)	46 (16.8%)	*0.009*
Psoas area (mm^2)	1951 ± 567	1872 ± 640	1966 ± 552	*0.287*
TPA (mm^2/m^2)	641.4 ± 192.7	634.9 ± 238.1	642.6 ± 183.2	*0.803*
PA/BMI (mm^2/kg/m^2)	75.2 ± 21.1	68.8 ± 19.1	76.5 ± 21.3	*0.023*

BMI: Body Mass Index; TPA: Total Psoas Area; PA/BMI: Psoas Area to BMI ratio.
Values are expressed as number and percent or mean ± standard deviation.

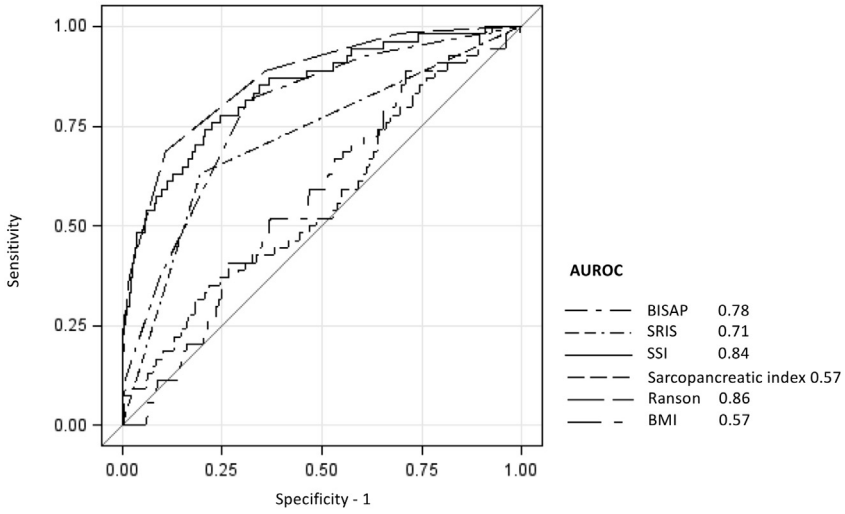

Fig. 1. Areas under the Receiver Operating Characteristics curves of the different scores and indices used to predict the severity of acute pancreatitis.

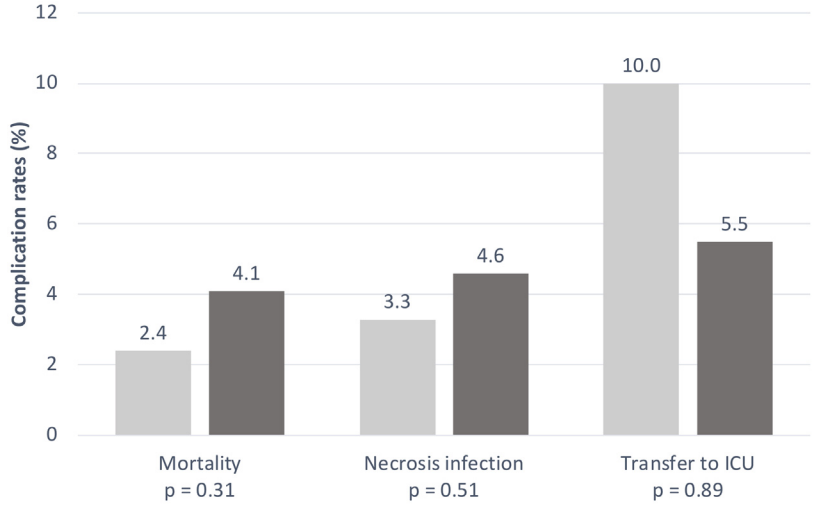

Fig. 2. Comparison of pancreatitis complication rates depending on the Sarcopancreatic Index (dark grey: high sarcopenic index; white: low sarcopenic index).

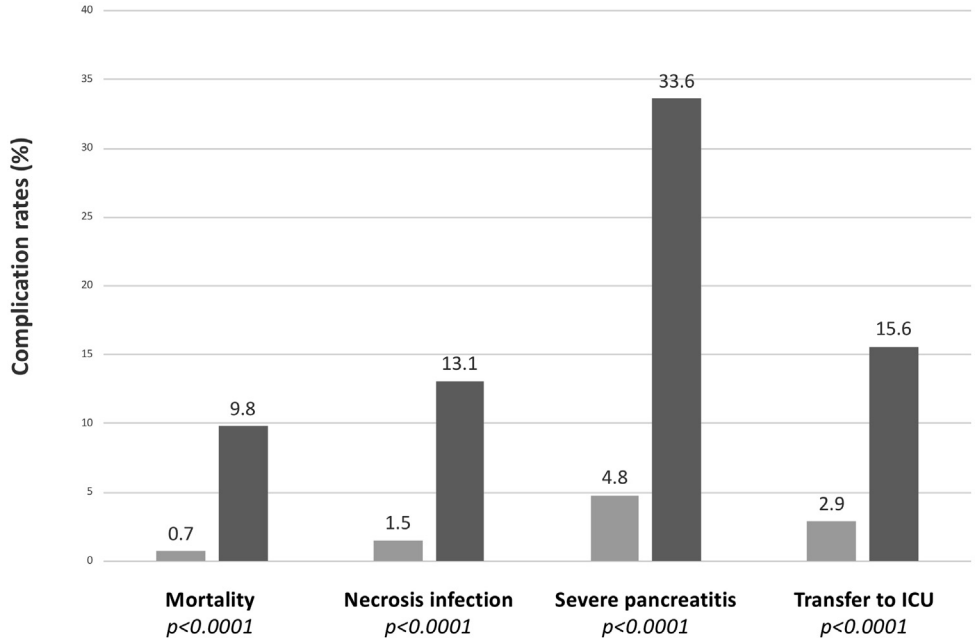

Fig. 3. Comparison of pancreatitis complication rates depending on the Sarcopenia Severity Index (SSI) (black: high SSI; grey: low SSI).

Table 3
Factors independently associated with the severity of acute pancreatitis; multivariate analysis.

	Odds Ratio	95% confidence interval	*p*
Visual Analog Scale	1.236	1.082; 1.411	*0.0018*
Creatinine (µmol/L)	1.022	1.014; 1.030	*<0.0001*
Albumin (g/L)	0.899	0.851; 0.951	*0.0002*
Sarcopancreatic Index	1.455	1.028; 2.061	*0.0346*

Variables included in the analysis: Age; VAS; Hypothermia; Creatinine; Albumin; Calcemia; CRP; Bilirubin; Sarcopancreatic Index.

The optimal threshold of SSI to predict the occurrence of a severe form was −2.0786. Using this threshold to predict the severity of AP, SSI had the following diagnostic performance: a sensitivity of 75.9%, a specificity of 76.3%, a positive predictive value of 33.6%, and a negative predictive value of 95.2%.

3.6. Pancreatitis complications rate based on SSI

In our cohort, patients with an SSI higher than the optimal threshold had a significantly higher risk of transfer to the ICU (15.6% vs. 2.9%; $p < 0.0001$), having a necrotic infection (13.1% vs. 1.5%; $p < 0.0001$), having a severe form (33.6% vs. 4.8%; $p < 0.0001$), or dying (9.8% vs. 0.7%; $p < 0.0001$) (Fig. 3).

4. Discussion

In our study, the assessment of sarcopenia was performed using psoas muscle area measures to calculate the PA/BMI ratio which can reflect sarcopenic obesity. By sex, the PA/BMI ratio was associated with severity of acute pancreatitis. When indexed to body surface area to adjust for gender differences in measures, the Sarcopancreatic Index was independently associated with the occurrence of severe acute pancreatitis. When this index was integrated with other classical clinico-biological parameters of severity to construct a prognostic score named the Sarcopenia Severity Index, it showed interesting performance, comparable to the best prognostic scores in acute pancreatitis in our cohort.

4.1. Obesity and acute pancreatitis prognosis

Obesity is known to be a risk factor for developing severe forms of acute pancreatitis, although data are discordant on its association with mortality [11,22,23]. Several studies have investigated how the presence of obesity can be used to improve the prediction of progression to a severe form. Papachristou et al. [11] showed that the presence of obesity added to the APACHE-II score was not more predictive of progression to a severe form of the disease. In our study, 23.1% of patients had a BMI greater than or equal to 30, with significantly more obese patients in the severe group than in the nonsevere group. However, BMI alone was not significantly associated with disease severity and did not perform well in predicting the occurrence of a severe form, with an AUROC of 0.57. Obesity alone or in combination with other severity parameters does not seem to be an effective predictor of the development of a severe form of AP. Some authors suggest that android obesity or the association of obesity with sarcopenia would be more efficient in predicting severe AP than obesity alone [24].

4.2. Sarcopenia measurement method

Sarcopenia is associated with several digestive diseases (cirrhosis, IBD or chronic pancreatitis) with an unfavorable outcome, such as more postoperative complications, higher mortality or an altered quality of life [12,13,15,17]. Sarcopenia can be assessed by various methods, such as functional tests or cross-sectional imaging [25]. In acute pancreatitis, studies that have studied the influence of sarcopenia or obesity used the Skeletal Muscle Index (SMI), muscle density, and visceral or subcutaneous fat measurements [18,19,24]. In our study, we retrospectively assessed the presence of sarcopenia by measuring the areas of the psoas muscles on a cross-section at L3 of a CT that had been performed the earliest after the onset of pain during hospitalization. This measurement has the advantage of being simple, feasible by nonradiologists, reliable and reproducible [26]. We chose to relate psoas muscle area to BMI rather than height squared to consider the morphology of patients in a more adapted way, especially in the case of obesity. The PA/BMI ratio obtained therefore considered the quantity of muscle and the presence of obesity. Interestingly, some authors

suggest that the presence of obesity and sarcopenia could lead to a condition called sarcopenic obesity. This condition would be responsible for more chronic inflammation than obesity or sarcopenia alone, which could be the cause of more complications in the case of acute diseases [27].

4.3. Impact of sarcopenia and sarcopenic obesity on pancreatitis prognosis

Studies focusing on the influence of sarcopenia on the outcome of AP are limited and contradictory. In a retrospective study, Sternby et al. assessed muscle mass using SMI and measurement of muscle attenuation at a single cross-section in L3 [18]. A decrease in muscle attenuation was associated with more severe forms of AP with an OR of 4.09, but severe forms had a significantly higher SMI than nonsevere forms. None of these parameters could strongly predict the severe form. The use of muscle attenuation was probably not appropriate because its measurement is influenced by age and sex and varies according to the injection times of CT [28]. In another retrospective study, Van Grinsven et al. assessed the association between mortality and visceral fat or muscle mass assessed using SMI and muscle density measurement in 496 patients with necrotizing pancreatitis [19]. These parameters were not associated with higher mortality. In this study, patients were divided into tertiles according to their SMI. Patients in the lowest tertile were considered to have sarcopenia. Because their study population was young and had limited comorbidities, patients included in this group may have been falsely interpreted as having sarcopenia without it being present, thus resulting in the lack of association with mortality. A Korean study investigated visceral fat, subcutaneous fat, SMI, and a visceral fat/SMI ratio (named VMR) among 203 patients with AP [24]. A low value of this ratio was associated with the occurrence of a severe form and had an AUROC of 0.757 to predict the evolution to severe acute pancreatitis. However, the complexity of its measurement makes it difficult to use this tool in clinical practice. Moreover, since the measurements were made in patients of Asian origin, these results require validation in a Caucasian population.

In our study, only the PA/BMI ratio which can reflect sarcopenic obesity was independently associated with the occurrence of severe acute pancreatitis. As explained previously, we believe that the risk conferred by sarcopenic obesity is different from that of obesity or sarcopenia alone. In the absence of a consensual definition of sarcopenia, the discrepancy of results in the literature could depend on whether or not sarcopenia was considered alone or associated with visceral fat or obesity.

4.4. Sarcopenic obesity as a predictive tool

In our study, the Sarcopancreatic Index, used to assess the presence of sarcopenia in patients with pancreatitis, was independently associated with the occurrence of a severe form of acute pancreatitis with an OR of 1.455. Using the Sarcopancreatic Index alone did not predict the severity of acute pancreatitis effectively, as its AUROC was 0.57. The same was observed for BMI. When the Sarcopancreatic Index was integrated with other variables independently associated with the severity of acute pancreatitis, such as VAS, creatinine and albumin at admission, a score called SSI was obtained that had better predictive performance for pancreatitis severity. Its AUROC was 0.84, comparable to that of the Ranson score in our cohort. Although this score has not been validated in an independent cohort, it presents interesting properties compared with the data in the literature. Depending on the studies, the AUROCs for the Ranson score vary from 0.85 to 0.94, those for BISAP approximately 0.80 and those for SIRS approximately 0.73, as observed in our population [9,29]. We believe that the interesting performance of SSI in this study demonstrates that the integration of new parameters such as nutritional parameters could open new perspectives to develop more efficient scores to predict the severity of acute pancreatitis.

4.5. Limitations

Our study has several limitations. This was a single-center, retrospective study. In our cohort, patients who were transferred more than 24 h after the onset of the episode were excluded due to a lack of data at the initial phase. These patients, initially managed in other hospitals, could have been transferred to our center because of a severe clinical course. They represent a group of potentially severe patients that we did not include, leading to a possible selection bias. Due to the retrospective design of our study, the evaluation of sarcopenia was not possible at admission, because the CT used was the one performed to evaluate the severity of acute pancreatitis, usually more than 72 h after the onset of pain. SSI would therefore only be applicable from the time of the CT and would not be calculable at admission. Furthermore, its lack of simplicity compared with scores such as SIRS is a limitation to its use. In the future, it would be interesting to evaluate other methods for measuring sarcopenia that are more easily calculable at admission, such as the handgrip test. The use of such anthropometric parameters at admission in association with classic clinical-biological parameters could make it possible to develop new prognostic tools.

5. Conclusion

Sarcopenic obesity seems to be associated with the occurrence of a severe form of acute pancreatitis. Its evaluation, combined with other classical clinical and biological prognostic parameters, could be considered as a tool to more efficiently predict the development of a severe form of the disease. These results suggest that anthropometric and sarcopenia parameters should be prospectively recorded in future studies that attempt to develop new prognostic tools in acute pancreatitis.

Declaration of Competing Interest

None declared

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