UDC 616.37-002:616-056.54
DOI http://doi.org/10.30978/GS-2025-4-73

ISSN 2786-5584 print
ISSN 2786-5592 online

# Prevention and treatment of acute secondary sarcopenia in patients with infected necrotizing pancreatitis. Literature review

**V. Teterina**

Bogomolets National Medical University, Kyiv

✉ Viktoriia Teterina: dr.teterina@nmu.ua

**V. Teterina**, http://orcid.org/0000-0001-8250-7646

This review of the current scientific literature focuses on the etiological factors, pathophysiological mechanisms, diagnostic approaches, and methods of prevention and treatment of acute secondary sarcopenia in patients with acute necrotizing pancreatitis. Acute secondary sarcopenia represents a severe complication of acute pancreatitis, resulting from a combination of systemic inflammation, physical inactivity, and nutritional deficiency. Scientific evidence indicates that sarcopenia and sarcopenic obesity are associated with higher mortality, an increased incidence of complications in acute pancreatitis, and longer hospital stays. According to the literature, the prevalence of secondary sarcopenia among patients with acute pancreatitis ranges from 18% to 70–80%, with variability in these indicators attributed to differences in diagnostic approaches, assessment criteria, and clinical characteristics of the patient cohorts studied. Contemporary studies have explored various approaches to diagnosing this condition, emphasizing the importance of early detection of secondary sarcopenia through functional tests, imaging, and instrumental diagnostic methods. It has been demonstrated that the prevention and treatment model for secondary sarcopenia requires a multidisciplinary team approach and includes effective anti-inflammatory therapy, optimization of nutritional support (early enteral nutrition with adequate protein and energy provision and correction of micronutrient deficiencies), the use of nutrients with anti-catabolic and anti-inflammatory properties (omega-3 polyunsaturated fatty acids, β-hydroxy-β-methylbutyrate, creatine), as well as early mobilization according to an individualized physiotherapy program. Clinical observations have confirmed that such interventions are associated with improved preservation and restoration of muscle mass and functional status, which directly influence survival rates, hospital stay duration, risk of complications, and disability. In summary, the review of international publications enabled the synthesis of current evidence on the diagnosis, prevention, and treatment of secondary sarcopenia in patients with acute necrotizing pancreatitis. The limited number of studies addressing this issue in the context of complicated acute pancreatitis underscores the relevance and necessity of further research aimed at refining and identifying optimal preventive and therapeutic strategies in this patient population.

**Keywords**
acute pancreatitis, necrotizing pancreatitis, secondary sarcopenia, sarcopenic obesity, diagnosis, nutritional support, multidisciplinary management.

**ARTICLE** • Received 2025-06-26 • Received in revised form 2025-07-03 • Published 2025-12-30


## Sarcopenia as a complication of acute pancreatitis: current state of research. Etiopathogenic mechanisms of acute secondary sarcopenia development

Secondary sarcopenia (SSP) is associated with increased mortality in the adult population and typically has a multifactorial etiology, including systemic inflammatory conditions, organ failure (OF), physical inactivity, and nutritional deficiencies [40]. According to the EWGSOP2 (2019) consensus, two forms are distinguished: an acute form (lasting < 6 months), which develops in the context of acute diseases or trauma, and a chronic form (> 6 months), which is mainly associated with chronic pathologies [8]. In clinical practice, cases of superacute, «fulminant» muscle loss developing within days or weeks have been reported in patients with sepsis, acute necrotizing pancreatitis (ANP), and following major surgical interventions [39]. According to systematic reviews, sarcopenia (SP) is an independent predictor of postoperative complications and mortality among emergency and surgical patients [20]. Thus,

in patients with acute pancreatitis (AP), sarcopenia (SP) is almost always secondary, acute in nature, and develops as a result of acute inflammation, OF, and nutritional deficiency (ND).

The scientific community's interest in studying the metabolic complications of AP is increasing every year; however, acute secondary sarcopenia (AS, an acute form of SSP) remains poorly understood. Currently, only a limited number of studies systematically and comprehensively analyze the relationship between AP and the reduction in skeletal muscle mass and function. Most of these studies have a retrospective design, use non-standardized criteria, include heterogeneous samples, and apply different methods and terminologies for assessing muscle mass. Few studies on SP classify pancreatitis according to morphology and etiology, and to date, no published research has analyzed AS specifically in infected necrotizing pancreatitis (INP).

According to individual studies, the prevalence of SP among patients with AP ranges from 18 % (assessed in a mixed cohort) [16] to 70–80 % (assessed in cohorts with severe AP treated in intensive care units (ICUs)) [32].

Secondary sarcopenia often has a multifactorial origin; however, the following types are conventionally distinguished based on pathophysiological mechanisms: inflammation-associated (mainly occurring in the context of systemic inflammatory response syndrome), immobilization-associated (resulting from loss of mobility due to trauma, prolonged stay in intensive care, etc.), endocrine (associated with hypothyroidism, diabetes mellitus, and other hormonal disorders), nutritional (due to malabsorption and/or reduced protein intake), and neurological (resulting from denervation caused by stroke or neurodegenerative diseases) [35].

Scientific studies rarely distinguish between primary and SSP in AP, which limits the ability to analyze specific mechanisms. According to most researchers, malnutrition and physical inactivity are considered the main factors contributing to SP, although the results of some studies show a weak or no association between muscle loss and baseline nutritional status, emphasizing instead the roles of physical inactivity and inflammation [18, 21].

The inflammatory mechanism of AS involves a cascade of reactive changes–systemic inflammation, immune-endocrine dysregulation, and anabolic resistance–that begins with the activation of serum cytokines (IL-6, IL-1β, TNF-α). These cytokines stimulate gene expression within the ubiquitin–proteasome system (UPS), leading to a rapid depletion of the muscle protein pool. In the context of a cytokine storm and energy depletion, autophagy becomes excessive, further exacerbating muscle loss. Among patients in severe and/or septic conditions, the key pathophysiological mechanisms underlying SP development include activation of the UPS, impaired insulin metabolism, and inhibition of mTOR-dependent anabolic processes [10, 41]. The above changes are accompanied by endocrine disturbances resulting from activation of the hypothalamic–pituitary–adrenal axis. Elevated cortisol levels, together with decreased concentrations of IGF-1, insulin, and testosterone, as well as increased myostatin levels, contribute to a pronounced predominance of catabolism over anabolism, leading to muscle dysfunction and structural remodeling (inflammation, fibrosis, fatty infiltration). In such a pathological environment, the anabolic capacity of muscle fibers declines, contributing to anabolic resistance, which substantially reduces the effectiveness of therapeutic interventions, including nutritional support (NS), dietary modifications, and physical activity, even when these are optimally implemented. Unlike systemic immune–endocrine dysregulation, which involves multiple physiological systems, anabolic resistance is a pathophysiological phenomenon specific to skeletal muscle tissue [9]. It has also been demonstrated that both SP and AP are associated with persistently elevated cytokine levels (TNF-α and IL-6) and, therefore, share a common catabolic profile [44].

Nutritional deficiency is considered a key pathogenetic mechanism of AS, as inflammation increases total energy expenditure and leads to the redistribution of amino acids for the synthesis of acute-phase proteins, activation of the immune response, and restoration of tissue integrity. Even with adequate nutritional intake, the protein–energy balance remains negative, resulting in hepatic gluconeogenesis and an increased demand for amino acids derived from skeletal muscle. Consequently, even with sufficient NS, this process–similar to cachexia–is only partially reversible, particularly in patients with severe disease [3]. Nutritional deficiency in AP is also driven by malabsorption and maldigestion. Exocrine pancreatic insufficiency, which is especially pronounced in severe AP, leads to impaired digestion of proteins and fats, steatorrhea, and micronutrient deficiencies, ultimately resulting in weight loss, especially of muscle mass [1], and predisposing to the development of SP.

Micronutrient deficiencies are independent risk factors for SP. For example, serum vitamin D levels are directly correlated with indicators of muscle strength [9]. It should be noted that this factor has also been investigated in the context of AP, where vitamin D deficiency has been associated with

a more severe disease course and adverse clinical outcomes [19]. This finding suggests a synergistic effect of vitamin D deficiency in the setting of the pathophysiological interplay between SP and AP during the disease course.

Other risk factors for the development of SP in patients with AP–associated with the inhibition of muscle regeneration and the intensification of catabolic processes–have also been reported, including a prolonged stay in the intensive care unit, characteristic of severe AP [18], and the occurrence of OF [32].

Against the backdrop of acute inflammation, prolonged periods of physical inactivity–such as bed rest or immobilization–serve as potent triggers of muscle atrophy through the activation of several pathophysiological pathways. Suppression of the IGF-1/Akt/mTOR signaling pathway, which normally stimulates protein synthesis, results in a markedly reduced capacity for renewal of the structural muscle protein pool. This process is further enhanced by the activation of FOXO (Forkhead box O) transcription factors, which induce the expression of E3 ubiquitin ligases–MuRF1 and MAFbx–thereby stimulating the UPS and promoting proteolysis. The accumulation of reactive oxygen species (ROS) and disturbances in calcium homeostasis further damage protein structures and activate proteolytic enzymes such as calpains and caspases, ultimately triggering apoptosis [15].

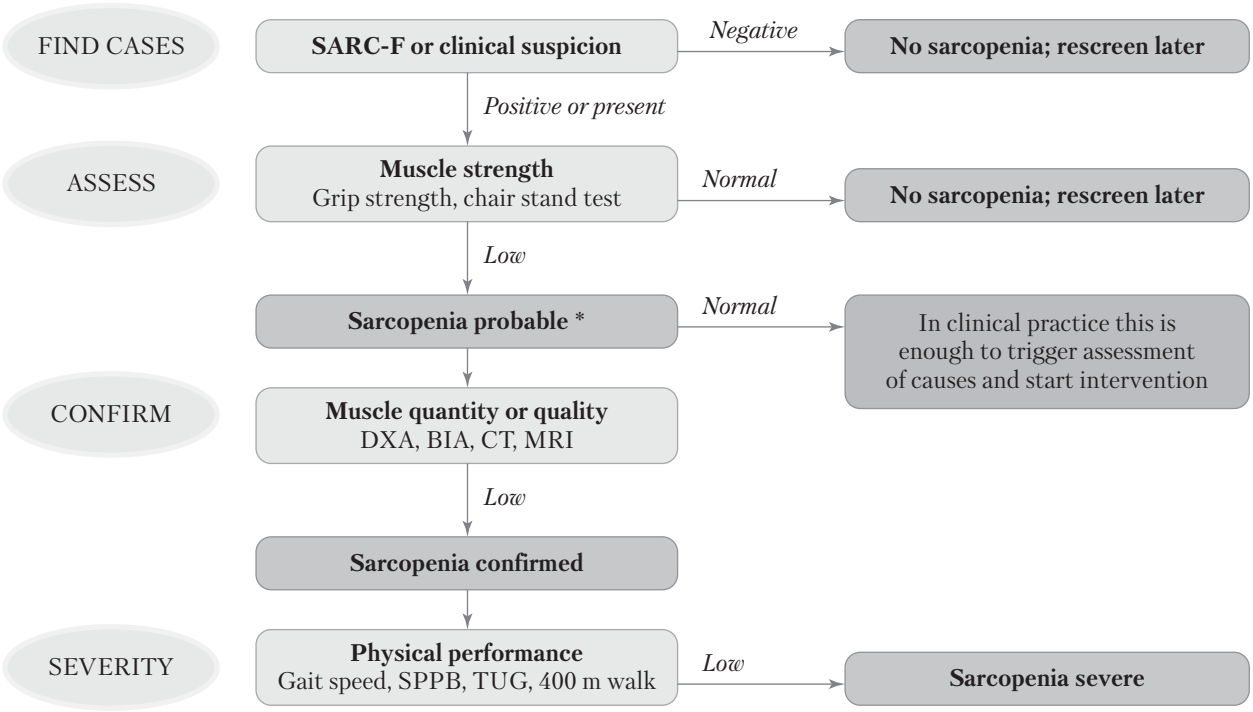
## Diagnosis of sarcopenia in acute pancreatitis

Diagnosing and assessing the severity of SP in AP is a challenging task, as each available approach has both advantages and limitations. The presence of acute pain and systemic inflammation, combined with restricted access to diagnostic techniques, reduces the validity and objectivity of assessment methods, particularly in the ICU setting. For the diagnosis of SP in the European population, the EWGSOP2 algorithm is considered the most appropriate tool (Figure).

It is recommended to begin the assessment with the SARC-F rapid screening questionnaire [26], which is based on the patient's subjective evaluation of five functional characteristics (Table).

Only one published study has reported the assessment of muscle strength in patients with AP using handgrip dynamometry [16]. Functional tests for evaluating physical performance are not feasible in patients during the early stages of AP due to weakness, impaired consciousness, OF, and severe pain. These limitations significantly reduce the comprehensiveness and objectivity of SP severity assessment in this population [18].

Bioelectrical impedance analysis (BIA) was used to assess SP in patients with AP in two studies. The authors of one of them emphasized that the method is not optimal due to significant errors associated with water–electrolyte imbalance, edema, and body




* Consider other possible causes of reduced muscle strength (e.g., depression, stroke, balance disorders, peripheral vascular disease)

Figure. **EWGSOP2 algorithm for diagnosing sarcopenia [8]**

Table. **SARC-F questionnaire for SP screening [26]**

| Question | Scoring |
|---|---|
| **Strength** | |
| How much difficulty do you have in lifting and carrying 10 pounds (4.5 kg)? | |
| None | 0 |
| Some | 1 |
| A lot or unable | 2 |
| **Assistance in walking** | |
| How much difficulty do you have walking across a room? | |
| None | 0 |
| Some | 1 |
| A lot, use aids, or unable | 2 |
| **Rise from a chair** | |
| How much difficulty do you have transferring from a chair or bed? | |
| None | 0 |
| Some | 1 |
| A lot or unable without help | 2 |
| **Climb stairs** | |
| How much difficulty do you have climbing a flight of 10 stairs? | |
| None | 0 |
| Some | 1 |
| A lot or unable | 2 |
| **Falls** | |
| How many times have you fallen in the past year? | |
| None | 0 |
| 1–3 falls | 1 |
| 4 or more falls | 2 |

Note. SARC-F score ≥ 4 best predicts the need for further, more comprehensive evaluation to confirm evidence of sarcopenia.

position [35]. There is also very limited availability of equipment for measurements in the ICU, as examinations are mainly performed in a standing position.

Computed tomography (CT) is considered the most optimal method for confirming SP, as most patients with AP undergo abdominal CT, which enables quantitative assessment of skeletal muscle mass and density at the L3 vertebral level. However, this approach may delay the identification of SP, since imaging is typically performed not at the time of hospital admission but after a certain period of disease progression [18]. A number of potential predictors of SP in AP have been identified, including low muscle density on CT and a reduced skeletal muscle index (SMI) measured at the level of the third lumbar vertebra (L3), both recognized as independent risk factors for mortality and the development of AP-related complications. However, this method also has certain limitations, such as the need for manual or semi-automatic muscle segmentation, insufficient standardization of measurement parameters, and limited feasibility for dynamic monitoring [17, 24].

Ultrasound examination (US) is one of the primary diagnostic methods for patients with AP and is also recognized as an effective tool for evaluating muscle parameters in the context of SP diagnosis. The technique for measuring muscle thickness, cross-sectional area, pennation angle, and echogenicity of the lower-limb muscles is being actively investigated and is considered a promising approach for dynamic monitoring of SP; however, it has not yet been applied in patients with AP.

Histological studies of muscle tissue in patients with AP in the context of SP diagnosis and treatment have not been conducted to date. In experimental mouse models of severe AP, pronounced muscle fiber atrophy has been observed, accompanied by a reduction in cross-sectional area and increased expression of catabolic protein markers (MuRF-1 and MAFbx) [11]. In a rat model of ANP, morphological alterations and functional impairment of the diaphragm and peripheral muscles were also reported [27]. Given the absence of clinical histological data, this area remains poorly explored and offers new perspectives for further investigation.

### Specific features of sarcopenia in acute pancreatitis complicated by infected necrosis

INP is defined as an infection of pancreatic and/or peripancreatic necrotic tissue, confirmed by the presence of gas collections on CT or by microbiological evidence of infection in aspirated fluid or necrotic material [2]. Infected necrosis (INP) develops in 20–40 % of severe AP cases, significantly complicating the disease course, increasing mortality, and raising the need for surgical interventions within the step-up approach [22].

Despite the relevance of the problem, no studies specifically addressing SP or sarcopenic obesity (SO) in INP had been published at the time of preparing this literature review. Therefore, the only data currently available include patient cohorts with severe AP, particularly those with ANP.,However, the number of studies focusing on sarcopenic complications in these clinical entities also remains very limited.

The data demonstrate a correlation between reduced skeletal muscle mass and the severity of acute pancreatitis, including mortality. A lower SMI at the L3 level correlates with higher MCTSI/Balthazar scores and increased in-hospital mortality in patients with severe AP [24]. Moreover, decreased

morphometric and qualitative parameters of the psoas major muscle–psoas muscle index (PMI) and psoas muscle density (PMD)–have been associated with a higher incidence of systemic complications, infectious events, and prolonged hospital stays [17]. In studies focusing on severe AP–where the inclusion criterion was the presence of OF–a relationship was identified between SP or SO and increased 30-day mortality, as well as higher mortality during subsequent follow-up periods [14]. The overall prognostic value of confirmed SP in the context of AP requires further investigation, given the heterogeneity and limited quantity of available data. A systematic review of four studies found insufficient evidence of an association between SP, assessed by CT, and the development of necrotic and systemic complications, disease recurrence, or increased mortality related to AP and its complications [21].

A pronounced long-term decline in muscle mass was observed among ICU patients with severe AP, particularly those with ANP, where the reduction in the iliopsoas muscle area reached approximately 48 %. A higher rate of muscle mass loss was associated with worse outcomes–about 1.34 % per day in non-survivors and 0.74 % per day in survivors [18]. In a cohort of patients with ANP, a decrease in both the cross-sectional area and density of the iliopsoas muscle (Hounsfield Unit Average Calculation, HUAC) was significantly associated with infected necrosis, OF, and mortality [41].

SO may pose a particular risk to patients with severe AP and serve as an additional predictor of mortality, alongside factors such as age and the number of OFs. The mortality rate among patients with severe AP and SO was 45 %, whereas in the cohort of patients with AP without obesity, the mortality rate was 20 %, and among those without any manifestations of OF, it was 10 % [14]. In another study, in addition to the association with a more severe course of AP, a correlation was also observed between visceral obesity and reduced muscle mass and quality on CT [6]. These particularly important findings suggest a bidirectional relationship–a «vicious cycle» mechanism–within cohorts of patients with AP and obesity. At the same time, some authors have denied the existence of a consistent link between body composition (particularly the amount of visceral fat) and mortality in AP, instead emphasizing muscle quality deterioration as the key determinant of an unfavorable outcome. Although baseline muscle characteristics on CT are not considered independent predictors of mortality, a i 10 % decrease in skeletal muscle HUAC within one month was significantly associated with increased in-hospital mortality [37].

### Prevention and treatment of secondary sarcopenia in infected necrotizing pancreatitis

To date, no published studies have specifically addressed the prevention or treatment of SP in patients with either INP or AP in general. Therefore, this section summarizes the principles of NS recommended by leading clinical nutrition societies–ESPEN (European Society for Clinical Nutrition and Metabolism) and ASPEN (American Society for Parenteral and Enteral Nutrition)–for patients with AP and for critically ill individuals, as well as the current consensus statements regarding SP.

The primary clinical objective is to prevent or minimize the development of SP in patients during the acute phase of AP. In patients with mild disease, early reintroduction of oral feeding is recommended, with gradual progression from liquid to soft and solid foods as soon as pain and nausea subside–even within the first day of the AP episode. In cases of moderate to severe AP, particularly INP, oral feeding is often not feasible due to the severity of symptoms. Therefore, enteral nutrition (EN) is recommended within 24–72 hours of hospitalization. This approach significantly reduces the risk of infectious complications and mortality compared with delayed or parenteral nutrition (PN) [1, 31]. It should be noted that complications related to EN may occur only under specific clinical conditions that represent absolute contraindications to its early initiation–such as suspected intestinal ischemia or perforation, complete obstruction, or prolonged paralytic ileus–until the underlying cause is resolved [1, 26]. Relative contraindications include hemodynamic instability [38], recurrent vomiting with a risk of aspiration [29], uncontrolled intra-abdominal hypertension, and abdominal compartment syndrome [1].

Enteral nutrition via a nasogastric tube is considered to be as effective and safe for patients with acute pancreatitis as feeding via a nasojejunal tube [12]. The latter is preferred in cases of severe gastroesophageal reflux, gastric paresis, mechanical pyloric obstruction, aspiration, or other persistent intolerance to EN that remains despite standard safety measures (such as the use of prokinetic agents and elevating the head of the bed by 30–45°) [1, 29].

To prevent refeeding syndrome (RFS), it is advisable to initiate EN slowly (10–20 mL/hour) at 70–80 % of the target requirements (approximately 20–25 kcal/kg/day), with gradual titration to the full target dose over 4–7 days, depending on tolerance [1, 4, 26, 31]. Monitoring of serum phosphorus and magnesium levels is mandatory before initiation and during the first 72 hours of EN. Thiamine, a critical cofactor of glucose oxidation enzymes,

should be administered at a dose of 100–200 mg 30–60 minutes before initiation and during the first days of EN to prevent typical complications of RFS–lactic acidosis, heart failure, and Wernicke's encephalopathy [8, 31]. If signs of intolerance to EN occur, feeding should be temporarily stopped, with the duration of the pause depending on the severity of symptoms, and the position of the feeding tube should be verified [4, 12]. In cases of aspiration or abdominal compartment syndrome, EN should be postponed until the patient's condition stabilizes, or temporary PN should be initiated, with continued prophylactic administration of thiamine [1, 13].

Adequate protein and energy support play a key role in the treatment and postoperative recovery of patients with acute pancreatitis. First-line therapy consists of polymeric isocaloric and isonitrogenous formulas for enteral nutrition [1, 4]. Second-line therapy includes oligomeric or peptide-based formulas, which are indicated in cases of intolerance to first-line products or in the presence of malabsorption (e.g., steatorrhea or diarrhea) [1, 12]. Specialized immunomodulatory formulas–enriched with arginine, omega-3 fatty acids, nucleotides, and other bioactive compounds–do not demonstrate significant advantages for routine use and are recommended only in selected cases [1, 31].

The acute phase of AP is characterized by a high risk of both hypervolemia and hypovolemia. Therefore, adequate fluid resuscitation aims to minimize the risks of edema, ascites, pleural effusion, and progression of abdominal hypertension, intestinal edema, and pulmonary congestion. In this context, it is also important to consider the total fluid volume when selecting an EN formula and to prescribe high-caloric formulas (1.5–2 kcal/mL) for patients with severe edema [1, 13].

The recommended target energy intake is 25–30 kcal/kg/day, including 1.2–2.0 g/kg/day of protein [1, 31]. For a long time, it was believed that fat intake worsened the course of AP, but this hypothesis has not been confirmed. According to current guidelines, fat restriction is not recommended in patients with AP unless severe malabsorption is present. In cases of diarrhea and/or steatorrhea, the use of enteral formulas containing medium-chain triglycerides (MCTs) is recommended [1, 9]. Strict glycemic control should also be maintained, keeping blood glucose levels between 7.8 and 10 mmol/L [4, 31].

Correction of nutritional deficiencies is an integral component of dietary management in AP. Patients rapidly deplete their stores of water-soluble vitamins ($B_6$, $B_{12}$, folate, and vitamin C) due to their catabolic state, significant fluid losses, and limited food intake [1, 31]. Malabsorption and steatorrhea can also rapidly lead to deficiencies of fat-soluble vitamins (A, D, E, and K) as a result of impaired lipid absorption [1, 25].

Daily monitoring of phosphorus, potassium, and magnesium levels is an essential component of RFS prevention [1, 9]. In cases of ANP and hypoalbuminemia, ionized or corrected calcium should also be monitored, as necrosis leads to the release of large amounts of calcium-binding fatty acids (saponification). Control of systemic inflammation in patients with INP is another key strategy for slowing catabolic processes, since a persistent inflammatory and infectious focus, even under conditions of adequate NS, promotes insulin resistance, hyperglycemia, proteolysis, and lipolysis, ultimately leading to muscle mass loss and a poorer prognosis [1, 31].

During a prolonged stay in the ICU–which is common in complicated necrotizing pancreatitis–a specific pathological condition often develops, characterized by hypodynamia and contributing to the progression of SP: ICU-acquired weakness (ICU-AW) [30, 33]. To preserve muscle function, early mobilization strategies are recommended, including frequent repositioning, passive range-of-motion exercises, gradual verticalization, and the early introduction of active movements under the supervision of a rehabilitation specialist [30, 31]. In patients in the early postoperative period after laparotomy, excessive physical activity may increase the risk of complications; therefore, management is limited to elevating the head of the bed, passive limb mobilization, breathing exercises, and preventive measures against deep vein thrombosis [30, 33].

The above SP prevention model for patients with AP emphasizes the importance and relevance of involving a multidisciplinary medical team to ensure effective inflammation control, comprehensive glycemic management, and minimization of the risk of infection or sepsis, along with meeting AP patients' energy requirements.

Modern approaches to the inpatient management of established SSP, particularly in patients with AP, emphasize the need for early intervention aimed at restoring muscle mass and function during the patient's stay in the ICU or hospital. The main clinical challenge is to initiate muscle recovery during the acute phase of the disease and to minimize catabolic processes.

In cases of severe catabolism and negative nitrogen balance, which are characteristic of postoperative states, sepsis, INP, and continuous renal replacement therapy, intensified NS is indicated. For patients in these categories, the daily protein requirement may reach 2.2–2.5 g/kg/day, with a standard energy intake of 25–30 kcal/kg/day [1, 31]. If target

values cannot be achieved using standard polymeric formulas, it is recommended to supplement with high-protein formulas or to combine them with PN [1, 25]. Determining the nitrogen balance (NB = total protein intake in grams/6.25 – 4) is an important component in assessing changes in nutritional status; however, this method has significant limitations due to the inability to accurately account for extraurinary nitrogen losses (e.g., via drainage, recurrent vomiting, or diarrhea) [31].

Replacement enzyme therapy is an essential component of NS for patients with severe maldigestion and steatorrhea, aimed at improving the absorption of macro- and micronutrients and thereby significantly enhancing the effectiveness of nutritional therapy. The recommended dosage ranges from 10,000 to 40,000 units of lipase per meal, depending on the amount of food consumed, with subsequent titration according to clinical response [1].

The use of specific nutraceuticals for SP correction has not been studied in cohorts of patients with AP, particularly those with INP. However, data from several systematic reviews and meta-analyses indicate beneficial effects of β-hydroxy-β-methylbutyrate (HMB), omega-3 polyunsaturated fatty acids, and creatine (as an adjunct supplement) in preserving and restoring muscle mass and strength [5, 7, 23, 34, 36]. Therefore, the use of these nutraceuticals may be considered an experimental component of NS in patients with AP, especially those with INP, to justify further targeted research in this population.

Early initiation of physical therapy is one of the main components of treatment for AS in patients with AP. The primary goal is to restore muscle strength and physical performance during hospitalization. According to current guidelines, patients are advised to begin early and gradual verticalization (sitting, standing, and walking) under the supervision of a multidisciplinary medical team, along with simple strength exercises such as isometric contractions and movements using resistance bands or body weight. These interventions have been shown to slow muscle loss and shorten the length of stay in the ICU. For patients who have undergone laparotomy in the early postoperative period, the range and intensity of movement should be determined individually in consultation with both the surgeon and the rehabilitation specialist [28, 31, 43].

Thus, modern inpatient approaches to the treatment of acute secondary sarcopenia in infected necrotizing pancreatitis are multifaceted and require the involvement of a multidisciplinary team of specialists. The preservation and restoration of muscle mass and function directly influence patient survival, length of hospitalization, risk of complications, and long-term disability. The limited number of studies addressing this issue in the context of complicated acute pancreatitis underscores the relevance and necessity of further research to refine and optimize treatment strategies for this patient population.

## Declaration of interests

The author declares that she has no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

## References


1. Arvanitakis M, Ockenga J, Bezmarevic M, Gianotti L, Krznarić Ž, Lobo DN, Löser C, Madl C, Meier R, Phillips M, Rasmussen HH, Van Hooft JE, Bischoff SC. ESPEN guideline on clinical nutrition in acute and chronic pancreatitis. Clin Nutr. 2020 Mar;39(3):612-31. doi: 10.1016/j.clnu.2020.01.004. Epub 2020 Jan 22. PMID: 32008871.
2. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, Tsiotos GG, Vege SS; Acute Pancreatitis Classification Working Group. Classification of acute pancreatitis-2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013 Jan;62(1):102-11. doi: 10.1136/gutjnl-2012-302779. Epub 2012 Oct 25. PMID: 23100216.
3. Barone M, Baccaro P, Molfino A. An Overview of sarcopenia: focusing on nutritional treatment approaches. Nutrients. 2025 Apr 1;17(7):1237. doi: 10.3390/nu17071237. PMID: 40218995; PMCID: PMC11990658.
4. Boullata JI, Carrera AL, Harvey L, Escuro AA, Hudson L, Mays A, McGinnis C, Wessel JJ, Bajpai S, Beebe ML, Kinn TJ, Klang MG, Lord L, Martin K, Pompeii-Wolfe C, Sullivan J, Wood A, Malone A, Guenter P; ASPEN Safe Practices for Enteral Nutrition Therapy Task Force, American Society for Parenteral and Enteral Nutrition. ASPEN Safe Practices for Enteral Nutrition Therapy [Formula: see text]. JPEN J Parenter Enteral Nutr. 2017 Jan;41(1):15-103. doi: 10.1177/0148607116673053. Epub 2016 Nov 5. PMID: 27815525.
5. Chen W, Song J, Gong S. Advances in nutritional metabolic therapy to impede the progression of critical illness. Front Nutr. 2024 Jul 5;11:1416910. doi: 10.3389/fnut.2024.1416910. PMID: 39036495; PMCID: PMC11259093.
6. Chhoda A, Bohara M, Liyen Cartelle A, Manoj MA, Noriega MA, Olivares M, Kelly J, Brook O, Freedman SD, Bezuidenhout AF, Sheth SG. Association of Visceral Adiposity and Sarcopenia with Geospatial Analysis and Outcomes in Acute Pancreatitis. J Clin Med. 2025 Apr 26;14(9):3005. doi: 10.3390/jcm14093005. PMID: 40364037; PMCID: PMC12072196.
7. Cornish SM, Cordingley DM, Shaw KA, Forbes SC, Leonhardt T, Bristol A, Candow DG, Chilibeck PD. Effects of Omega-3 Supplementation Alone and Combined with Resistance Exercise on Skeletal Muscle in Older Adults: A Systematic Review and Meta-Analysis. Nutrients. 2022 May 26;14(11):2221. doi: 10.3390/nu14112221. PMID: 35684018; PMCID: PMC9182791.
8. Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, et al; Writing Group for the European Working Group on Sarcopenia in Older People 2 (EWGSOP2), and the Extended Group for EWGSOP2. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing. 2019 Jan 1;48(1):16-31. doi: 10.1093/ageing/afy169. Erratum in: Age Ageing. 2019 Jul 1;48(4):601. doi: 10.1093/ageing/afz046. PMID: 30312372; PMCID: PMC6322506.
9. Da Silva JSV, Seres DS, Sabino K, Adams SC, Berdahl GJ, Citty SW, Cober MP, Evans DC, Greaves JR, Gura KM, Michalski A, Plogsted S, Sacks GS, Tucker AM, Worthington P, Walker RN, Ayers P; Parenteral Nutrition Safety and Clinical Practice Committees, American Society for Parenteral and Enteral Nutrition. ASPEN Consensus Recommendations for Refeeding Syndrome. Nutr Clin Pract. 2020 Apr;35(2):178-95. doi: 10.1002/ncp.10474. Epub 2020 Mar 2. Erratum in: Nutr Clin Pract. 2020 Jun;35(3):584-85. doi: 10.1002/ncp.10491. PMID: 32115791.

10. Damanti S, Senini E, De Lorenzo R, et al. Acute sarcopenia: mechanisms and management. Nutrients. 2024 Oct 10;16(20):3428. doi: 10.3390/nu16203428. PMID: 39458423; PMCID: PMC11510680.
11. Dong X, Luo W, Wang Y, et al. Role and mechanism of myonectin in severe acute pancreatitis: a crosstalk between skeletal muscle and pancreas. Skelet Muscle. 2024 Dec 3;14(1):29. doi: 10.1186/s13395-024-00363-1. PMID: 39627746; PMCID: PMC11613728.
12. Dutta AK, Goel A, Kirubakaran R, Chacko A, Tharyan P. Nasogastric versus nasojejunal tube feeding for severe acute pancreatitis. Cochrane Database Syst Rev. 2020 Mar 26;3(3):CD010582. doi: 10.1002/14651858.CD010582.pub2. PMID: 32216139; PMCID: PMC7098540.
13. Evans L, Rhodes A, Alhazzani W, Antonelli M, Coopersmith CM, French C, Machado FR, Mcintyre L, Ostermann M, Prescott HC, Schorr C, Simpson S, Wiersinga WJ, Alshamsi F, Angus DC, Arabi Y, Azevedo L, Beale R, Beilman G, Belley-Cote E, Burry L, Cecconi M, Centofanti J, Coz Yataco A, De Waele J, Dellinger RP, Doi K, Du B, Estenssoro E, Ferrer R, Gomersall C, Hodgson C, Møller MH, Iwashyna T, Jacob S, Kleinpell R, Klompas M, Koh Y, Kumar A, Kwizera A, Lobo S, Masur H, McGloughlin S, Mehta S, Mehta Y, Mer M, Nunnally M, Oczkowski S, Osborn T, Papathanassoglou E, Perner A, Puskarich M, Roberts J, Schweickert W, Seckel M, Sevransky J, Sprung CL, Welte T, Zimmerman J, Levy M. Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock 2021. Intensive Care Med. 2021 Nov;47(11):1181-247. doi: 10.1007/s00134-021-06506-y. Epub 2021 Oct 2. PMID: 34599691; PMCID: PMC8486643.
14. Farquhar R, Matthews S, Baxter N, et al. Sarcopenia and sarcopenic obesity on body composition analysis is a significant predictor of mortality in severe acute pancreatitis: a longitudinal observational study. World J Surg. 2023 Nov;47(11):2825-33. doi: 10.1007/s00268-023-07122-1. Epub 2023 Aug 4. PMID: 37541981; PMCID: PMC10545625.
15. Gao Y, Arfat Y, Wang H, Goswami N. Muscle atrophy induced by mechanical unloading: mechanisms and potential countermeasures. Front Physiol. 2018 Mar 20;9:235. doi: 10.3389/fphys.2018.00235. PMID: 29615929; PMCID: PMC5869217.
16. Kilic GS, Tahtaci M, Yagmur F, Akin FE, Tayfur Yurekli O, Ersoy O. Influence of sarcopenia as determined by bioelectrical impedance analysis in acute pancreatitis. Medicine (Baltimore). 2024 Dec 6;103(49):e40868. doi: 10.1097/MD.0000000000040868. PMID: 39654169; PMCID: PMC11630989.
17. Kirzhner A, Rossels A, Sapojnik D, Zaharoni H, Cohen R, Lin G, Schiller T. Psoas muscle index and density as prognostic predictors in patients hospitalized with acute pancreatitis. J Clin Med. 2024 Oct 22;13(21):6314. doi: 10.3390/jcm13216314. PMID: 39518454; PMCID: PMC11547049.
18. Kolck J, Hosse C, Leimbach A, et al. Opportunistic screening for long-term muscle wasting in critically ill patients: insights from an acute pancreatitis cohort. Eur J Med Res. 2024 May 22;29(1):294. doi: 10.1186/s40001-024-01884-7. PMID: 38778361; PMCID: PMC11110383.
19. Kolosovych I, Hanol I, Bystrytska M, Uzun H. Changes in vitamin D and calcium-phosphorus metabolism in patients with severe acute pancreatitis. Turk J Surg. 2022 Dec 20;38(4):327-33. doi: 10.47717/turkjsurg.2022.5669. PMID: 36875274; PMCID: PMC9979554.
20. Knoedler S, Schliermann R, Knoedler L, et al. Impact of sarcopenia on outcomes in surgical patients: a systematic review and meta-analysis. Int J Surg. 2023 Dec 1;109(12):4238-4262. doi: 10.1097/JS9.0000000000000688. PMID: 37696253; PMCID: PMC10720826.
21. Le KDR, Patel H, Downie E. A systematic review on the prognostic role of radiologically-proven sarcopenia on the clinical outcomes of patients with acute pancreatitis. PLoS One. 2025 Apr 29;20(4):e0322409. doi: 10.1371/journal.pone.0322409. PMID: 40299837; PMCID: PMC12040213.
22. Leppäniemi A, Tolonen M, Tarasconi A, Segovia-Lohse H, Gamberini E, Kirkpatrick AW, Ball CG, Parry N, Sartelli M, Wolbrink D, van Goor H, Baiocchi G, Ansaloni L, Biffl W, Coccolini F, Di Saverio S, Kluger Y, Moore E, Catena F. 2019 WSES Guidelines for the Management of Severe Acute Pancreatitis. World J Emerg Surg. 2019 Jun 13;14:27. doi: 10.1186/s13017-019-0247-0. PMID: 31210778; PMCID: PMC6567462.
23. Li N, Chen S, He Y, et al. Effects of oral supplementation of β -hydroxy-β -methylbutyrate on muscle mass and strength in individuals over the age of 50: a meta-analysis. Front Nutr. 2025 Apr 3;12:1522287. doi: 10.3389/fnut.2025.1522287. PMID: 40248035; PMCID: PMC12003145.
24. Lin C, Zhang J, Wang C, Lian W, Liu Y. The impact of skeletal muscle index at the third lumbar spine on nosocomial deterioration and short-term prognosis in acute pancreatitis: a retrospective observational study. PeerJ. 2024 Apr 30;12:e17283. doi: 10.7717/peerj.17283. PMID: 38708354; PMCID: PMC11067894.
25. McClave SA, Taylor BE, Martindale RG, Warren MM, Johnson DR, Braunschweig C, McCarthy MS, Davanos E, Rice TW, Cresci GA, Gervasio JM, Sacks GS, Roberts PR, Compher C; Society of Critical Care Medicine; American Society for Parenteral and Enteral Nutrition. Guidelines for the Provision and Assessment of Nutrition Support Therapy in the Adult Critically Ill Patient: Society of Critical Care Medicine (SCCM) and American Society for Parenteral and Enteral Nutrition (A.S.P.E.N.). JPEN J Parenter Enteral Nutr. 2016 Feb;40(2):159-211. doi: 10.1177/0148607115621863. Erratum in: JPEN J Parenter Enteral Nutr. 2016 Nov;40(8):1200. doi: 10.1177/0148607116670155. PMID: 26773077.
26. Malmstrom TK, Morley JE. SARC-F: a simple questionnaire to rapidly diagnose sarcopenia. J Am Med Dir Assoc. 2013 Aug;14(8):531-2. doi: 10.1016/j.jamda.2013.05.018. Epub 2013 Jun 25. PMID: 23810110.
27. Matuszczak Y, Viires N, Allamedin H, Aubier M, Desmonts JM, Dureuil B. Alteration in diaphragmatic function induced by acute necrotizing pancreatitis in a rodent model. Am J Respir Crit Care Med. 1999 Nov;160(5 Pt 1):1623-8. doi: 10.1164/ajrccm.160.5.9806004. PMID: 10556131.
28. Wang YT, Lang JK, Haines KJ, Skinner EH, Haines TP. Physical rehabilitation in the ICU: A systematic review and meta-analysis. Crit Care Med. 2022 Mar 1;50(3):375-88. doi: 10.1097/CCM.0000000000005285. PMID: 34406169.
29. Pathak A, Blackwell S, Sealock RJ. Should an NPO order be placed for my patient with acute pancreatitis? Cleve Clin J Med. 2024 Jun 3;91(6):341-3. doi: 10.3949/ccjm.91a.24009. PMID: 38830703.
30. Schweickert WD, Pohlman MC, Pohlman AS, et al. Early physical and occupational therapy in mechanically ventilated, critically ill patients: a randomised controlled trial. Lancet. 2009 May 30;373(9678):1874-82. doi: 10.1016/S0140-6736(09)60658-9. Epub 2009 May 14. PMID: 19446324; PMCID: PMC9906655.
31. Singer P, Blaser AR, Berger MM, Calder PC, Casaer M, Hiesmayr M, Mayer K, Montejo-Gonzalez JC, Pichard C, Preiser JC, Szczeklik W, van Zanten ARH, Bischoff SC. ESPEN Practical and Partially Revised Guideline: Clinical Nutrition in the Intensive Care Unit. Clin Nutr. 2023 Sep;42(9):1671-89. doi: 10.1016/j.clnu.2023.07.011. Epub 2023 Jul 15. PMID: 37517372.
32. Singh T, Azim A, Gutte S, Gurjar M, Poddar B, Niyaz Z, Ghosh NK. Impact of sarcopenia on the outcome of patients of severe acute pancreatitis in a critical care setting: a retrospective observational study. Indian J Crit Care Med. 2024 Mar;28(Suppl 1):S263-5. doi: 10.5005/jaypee-journals-10071-24667.97. Epub 2024 Mar 7. PMCID: PMC11886205.
33. Stevens RD, Dowdy DW, Michaels RK, Mendez-Tellez PA, Pronovost PJ, Needham DM. Neuromuscular dysfunction acquired in critical illness: a systematic review. Intensive Care Med. 2007 Nov;33(11):1876-91. doi: 10.1007/s00134-007-0772-2. Epub 2007 Jul 17. PMID: 17639340.
34. Su H, Zhou H, Gong Y, et al. The effects of β-hydroxy-β-methylbutyrate or HMB-rich nutritional supplements on sarcopenia patients: a systematic review and meta-analysis. Front Med (Lausanne). 2024 Jul 12;11:1348212. doi: 10.3389/fmed.2024.1348212. PMID: 39071082; PMCID: PMC11272589.
35. Tournadre A, Vial G, Capel F, Soubrier M, Boirie Y. Sarcopenia. Joint Bone Spine. 2019 May;86(3):309-14. doi: 10.1016/j.jbspin.2018.08.001. Epub 2018 Aug 8. PMID: 30098424.
36. Tseng PT, Zeng BY, Zeng BS, et al. Omega-3 polyunsaturated fatty acids in sarcopenia management: A network meta-analysis of randomized controlled trials. Ageing Res Rev. 2023 Sep;90:102014. doi: 10.1016/j.arr.2023.102014. Epub 2023 Jul 11. PMID: 37442370.
37. Van Grinsven J, van Vugt JLA, Gharbharan A, Bollen TL, Besselink MG, van Santvoort HC, van Eijck CHJ, Boerma D; Dutch Pancreatitis Study Group. The association of computed tomography-assessed body composition with mortality in patients with necrotizing pancreatitis. J Gastrointest Surg. 2017 Jun;21(6):1000-8. doi: 10.1007/s11605-016-3352-3. Epub 2017 Mar 15. PMID: 28299618; PMCID: PMC5443861.
38. Wang L, Zhong X, Yang H, et al. When can we start early enteral nutrition safely in patients with shock on vasopressors? Clin Nutr ESPEN. 2024 Jun;61:28-36. doi: 10.1016/j.clnesp.2024.03.007. Epub 2024 Mar 12. PMID: 38777444.

39. Welch C, K Hassan-Smith Z, A Greig C, M Lord J, A Jackson T. Acute sarcopenia secondary to hospitalisation – an emerging condition affecting older adults. Aging Dis. 2018 Feb 1;9(1):151-64. doi: 10.14336/AD.2017.0315. PMID: 29392090; PMCID: PMC5772853.
40. Xu J, Wan CS, Ktoris K, Reijnierse EM, Maier AB. Sarcopenia is associated with mortality in adults: a systematic review and meta-analysis. Gerontology. 2022;68(4):361-76. doi: 10.1159/000517099. Epub 2021 Jul 27. PMID: 34315158.
41. Yee EJ, Maatman TK, Sood AJ, Gunderman L, Zyromski NJ. Dynamic frailty: Objective physiological assessment to guide management in necrotizing pancreatitis. Surgery. 2021 May;169(5):1078-85. doi: 10.1016/j.surg.2020.10.017. Epub 2020 Dec 15. PMID: 33339652.
42. Yoshihara I, Kondo Y, Okamoto K, Tanaka H. Sepsis-associated muscle wasting: a comprehensive review from bench to bedside. Int J Mol Sci. 2023 Mar 6;24(5):5040. doi: 10.3390/ijms24055040. PMID: 36902469; PMCID: PMC10003568.
43. Zhang L, Hu W, Cai Z, et al. Early mobilization of critically ill patients in the intensive care unit: A systematic review and meta-analysis. PLoS One. 2019 Oct 3;14(10):e0223185. doi: 10.1371/journal.pone.0223185. PMID: 31581205; PMCID: PMC6776357.
44. Zhang S, Hu C, Wang X, Huang Z, Xia Q, Deng L. Identification of biomarkers and immune-metabolic regulators in acute pancreatitis and sarcopenia: a multi-modal transcriptomics study. J Cell Mol Med. 2025 Aug;29(15):e70769. doi: 10.1111/jcmm.70769. PMID: 40785039; PMCID: PMC12336052.

# Профілактика та лікування гострої вторинної саркопенії у пацієнтів з інфікованим некротичним панкреатитом. Огляд літератури

**В. Тетеріна**

Національний медичний університет імені О. О. Богомольця, Київ

Проведено огляд сучасної наукової літератури, присвяченої вивченню етіологічних чинників, патогенетичних механізмів, діагностичних підходів і методів профілактики та лікування гострої вторинної саркопенії у хворих на гострий некротичний панкреатит. Гостра вторинна саркопенія — тяжке ускладнення гострого панкреатиту, зумовлене поєднанням системного запалення, гіподинамії та нутритивної недостатності. У наукових дослідженнях доведено, що саркопенія та саркопенічне ожиріння асоціюються з вищою смертністю та частотою виникнення ускладнень гострого панкреатиту, а також із тривалішим перебуванням у стаціонарі. За даними літератури, поширеність вторинної саркопенії серед хворих із гострим панкреатитом становить від 18 до 70—80 %. Варіабельність показників зумовлена відмінностями в діагностичних підходах, критеріях оцінки та клінічних характеристиках досліджуваних когорт пацієнтів. У сучасних наукових роботах розглянуто різні підходи до діагностики зазначеного захворювання. Наголошено на важливості раннього виявлення вторинної саркопенії за допомогою функціональних тестів, візуалізаційних та інструментальних методів діагностики. Обґрунтовано, що модель профілактики й лікування вторинної саркопенії потребує залучення мультидисциплінарної команди лікарів і полягає в ефективній протизапальній терапії, оптимізації нутритивної підтримки (раннє ентеральне харчування з адекватним білково-енергетичним забезпеченням, корекція дефіцитів мікроелементів), застосуванні нутрієнтів з антикатаболічними та протизапальними властивостями (ω-3 поліненасичені жирні кислоти, β-гідрокси-β-метилбутират, креатин), а також ранній активізації хворого за індивідуальною програмою фізіотерапії. У клінічних спостереженнях підтверджено, що такі втручання асоціюються з кращими результатами збереження та відновлення м'язової маси й функціонального статусу пацієнтів, що безпосередньо впливає на виживаність хворих, тривалість госпіталізації, ризик ускладнень та інвалідизації. Огляд зарубіжних публікацій дав змогу узагальнити сучасні дані щодо діагностики, профілактики та лікування вторинної саркопенії у хворих із гострим некротичним панкреатитом. Невелика кількість праць, присвячених цій проблемі в контексті ускладненого гострого панкреатиту, свідчить про актуальність і необхідність проведення досліджень для уточнення та пошуку оптимальних профілактичних і лікувальних стратегій для цієї категорії пацієнтів.

**Ключові слова:** гострий панкреатит, некротичний панкреатит, вторинна саркопенія, саркопенічне ожиріння, діагностика, нутритивна підтримка, мультидисциплінарний підхід.

**FOR CITATION**

Teterina V. Prevention and treatment of acute secondary sarcopenia in patients with infected necrotizing pancreatitis. Literature review. General Surgery (Ukraine). 2025;(4);73-81. http://doi.org/10.30978/GS-2025-4-73.