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Sarcopenic Obesity in Cancer Patients: A Comprehensive Review of Prevalence, Clinical Impact, and Therapeutic Implications

Jihane Bouziane *, Wissal Hassani, Samia Khalfi, Kaoutar Soussy, Fatima Zahraa Farhane, Zenab Alami and Touria Bouhafa

Department of Radiation Oncology, Hassan II University Hospital, Fez, Morocco.

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Abstract

Sarcopenic obesity (SO) is a clinical entity characterized by the coexistence of low muscle mass (sarcopenia) and excess fat mass (obesity). In cancer populations, this condition is often overlooked, as it is masked by a normal or elevated body mass index (BMI). This literature review synthesizes current data on the prevalence, prognostic implications, and potential mechanisms of SO in oncology. The average prevalence of SO is approximately 9.3% (range: 2.3–14.6%) in patients with solid tumors and reaches 24.7% in obese patients (BMI ≥ 30 kg/m^2). SO is independently associated with reduced survival, an increased risk of postoperative complications, and dose-limiting toxicity from systemic chemotherapy. Assessment by computed tomography (CT) at the level of the third lumbar vertebra (L3) is the gold standard for diagnosis. Despite growing evidence, standardized diagnostic criteria and specific management strategies are still lacking. Pharmacokinetic studies and dose-modulation trials based on lean body mass are needed to optimize treatment safety and efficacy in these high-risk patients.

Keywords: Sarcopenic Obesity; Cancer; Body Composition; Computed Tomography; Survival; Toxicity; Surgical Complications

1. Introduction

The assessment of nutritional status and body composition in cancer patients traditionally relies on weight and body mass index (BMI). However, these parameters do not provide information on body composition, i.e., the relative distribution of muscle and adipose tissue. It is now established that cancer patients can exhibit divergent body composition trajectories, with muscle loss potentially occurring even in the setting of stable or increasing weight, leading to an often invisible phenotype: sarcopenic obesity (SO) [1,2].

Roubenoff referred to the "convergence of two epidemics" in modern populations: obesity and sarcopenia [3]. Sarcopenic obesity (SO) represents the extreme manifestation of this conjunction, characterized by the simultaneous accumulation of high fat mass and a severe reduction in muscle mass [4]. The advent of secondary analysis of routine staging CT scans now allows for precise and specific quantification of muscle and fat mass, revealing considerable heterogeneity not captured by BMI [2,5].

This review aims to: 1) summarize the definitions and prevalence of SO in oncology; 2) synthesize data concerning its impact on survival, surgical complications, and systemic treatment toxicity; and 3) discuss potential pathophysiological mechanisms and management perspectives.

* Corresponding author: Jihane Bouziane

2. Methods

This narrative review synthesizes current evidence on the definition, epidemiology, and clinical impact of sarcopenic obesity (SO) in oncology.

The analysis was structured around three core objectives, guiding the selection and interpretation of the literature:

- **Definition and Diagnostic Evolution:** We examined key consensus statements and critical reviews to trace the evolving definition of SO. Particular emphasis was placed on the 2022 joint consensus of the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) [10], which provides a modern diagnostic framework integrating low muscle function, low muscle mass, and high adiposity.
- **Prognostic and Clinical Impact:** The review prioritized original research employing objective, imaging-based body composition analysis, primarily computed tomography (CT) at the third lumbar vertebra, to evaluate the association between SO and critical oncology outcomes. This includes foundational studies, such as that by Prado et al. [5], and subsequent investigations establishing SO as an independent risk factor for reduced survival, increased postoperative complications, and heightened toxicity from systemic therapies.
- **Pathophysiological and Mechanistic Insights:** To explain the observed clinical risks, we integrated pharmacokinetic and body composition studies that elucidate the disconnect between standard drug dosing metrics and the actual volume of distribution in patients with SO. This mechanistic perspective underpins the discussion on toxicity and informs potential management strategies.

This approach aims to provide a factual and critical overview of SO as a relevant factor in the management of cancer patients (Figure 1).

Figure 1 Flowchart of Study Selection and Analysis

3. Results

3.1. Definition and Diagnostic Variability

There is no universal consensus for defining SO, leading to considerable heterogeneity in the literature that hinders research comparability and clinical practice [6]. In oncology, sarcopenia has most often been operationally defined by a skeletal muscle index (SMI, muscle area at L3/height^2) below sex-specific thresholds associated with mortality risk, such as <52.4 cm^2/m^2 for men and <38.5 cm^2/m^2 for women in gastrointestinal and respiratory cancers [5]. A critical limitation is that this and similar diagnostic approaches are based solely on low muscle mass, without accounting for muscle strength, which is considered the primary diagnostic parameter in geriatric consensus definitions like EWGSOP2 [7]. Furthermore, body composition assessment varies widely, with studies analyzing different muscle compartments and applying different normalization factors [6].

The definition of obesity in SO also varies considerably. While body mass index (BMI) ≥ 30 kg/m^2 is commonly used in oncology studies [8], other thresholds (25 or 27 kg/m^2) or direct measures like fat mass percentage and waist circumference are also applied [6]. Waist circumference is a notable indicator as it reflects visceral abdominal adiposity, which may directly contribute to impaired muscle mass and function [9]. The reliance on BMI alone is a significant limitation, as it cannot differentiate between fat and lean mass.

3.2. Towards a Standardized Diagnostic Framework: The ESPEN/EASO Consensus

To address these inconsistencies, the 2022 joint consensus of the European Society for Clinical Nutrition and Metabolism (ESPEN) and the European Association for the Study of Obesity (EASO) proposed a standardized definition and a structured diagnostic algorithm [10]. The consensus defines SO as a condition characterized by the coexistence of adiposity-based obesity (high body fat percentage) and sarcopenia (low muscle mass *and* function). It underscores that SO is a unique clinical phenotype requiring the concomitant evaluation of both parameters, as the interaction between excess fat and deficient muscle drives its adverse outcomes [10]. A key advancement is the emphasis on muscle function as a necessary diagnostic component, aligning with the EWGSOP2 approach [7]. The consensus also highlights that standard population reference ranges for muscle mass may be inadequate for individuals with obesity, indicating a potential need for body mass-adjusted thresholds to avoid underestimating sarcopenia [11, 12, 13].

The ESPEN/EASO framework is based on a three-step process: screening, diagnosis, and staging [10].

Screening aims for maximum sensitivity. It should target all individuals with a high BMI (using WHO thresholds: ≥ 30 kg/m^2 for non-Asians, ≥ 27.5 kg/m^2 for Asians) or high waist circumference (≥ 102 cm for men, ≥ 88 cm for Caucasian women with cardiometabolic risk), combined with risk factors for sarcopenia. These include age ≥ 70 years, the presence of chronic diseases, or any clinical suspicion of functional decline [10, 14, 15].

Figure 2 presents a visual summary of the clinical indications for sarcopenic obesity screening, mapping clinical categories to their specific diagnostic indicators.

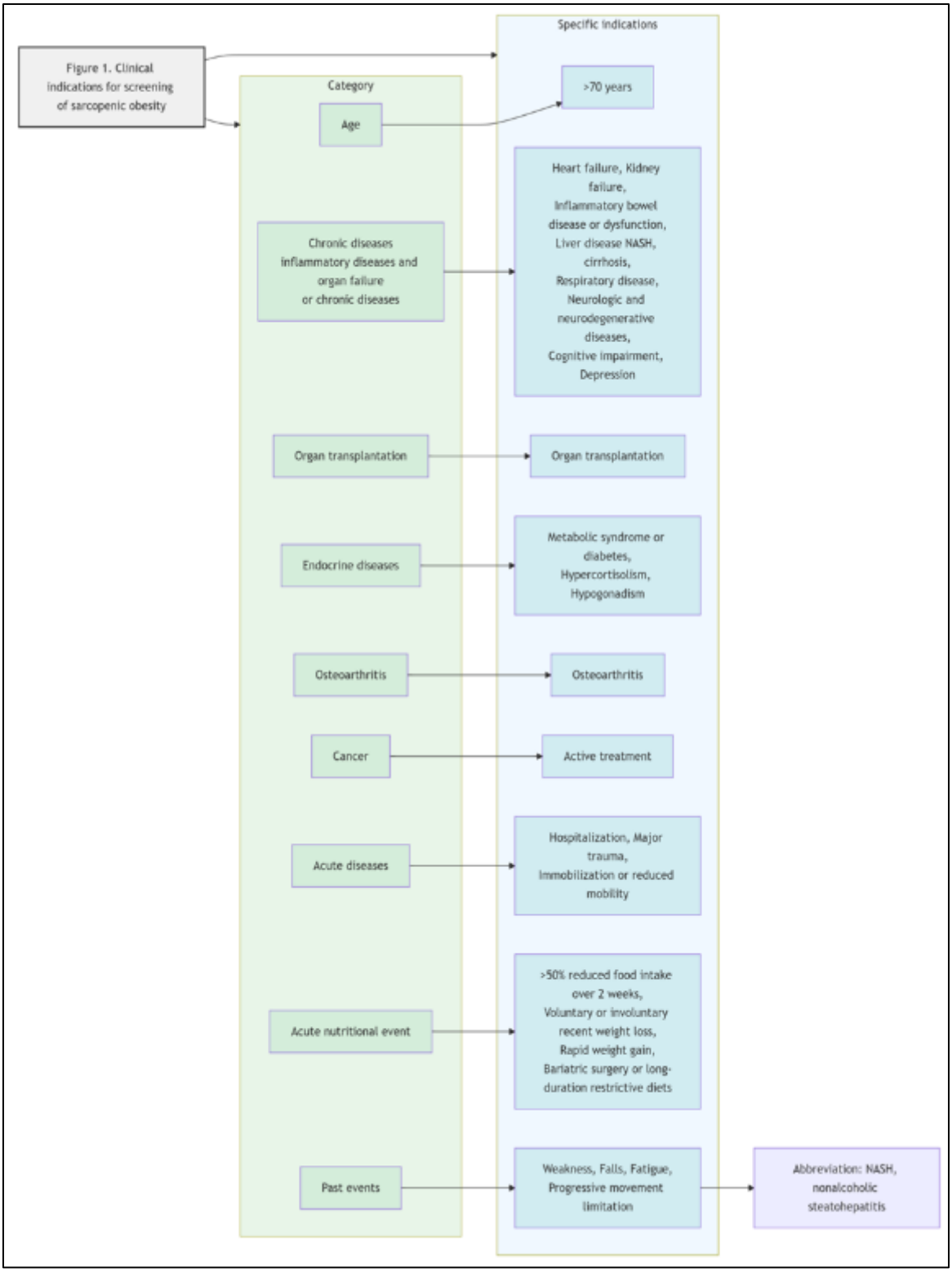

Figure 2 Clinical indications for screening of sarcopenic obesity

Diagnosis follows a two-step confirmation. First, reduced muscle strength must be documented using validated tests such as handgrip strength (< 27 kg for Caucasian men, < 16 kg for women) or the five-time sit-to-stand test (≥ 17 seconds). Second, if weakness is confirmed, a body composition analysis (via DXA, BIA, or CT) is required to simultaneously verify low muscle mass (using body weight-adjusted indices like SMM/W or ALM/W) and high fat mass [10, 12, 13].

Staging is recommended to stratify severity: Stage I for SO without overt complications and Stage II for SO associated with complications like impaired mobility or metabolic dysregulation [10].

Although the ESPEN/EASO criteria are recent and their application in oncology is still emerging—with preliminary studies in lung cancer and other comorbidities showing the need for further validation—they represent a crucial step toward standardizing the identification of this high-risk phenotype [10, 16].

3.3. Prevalence Estimates in Oncology

The reported average prevalence of SO in populations with advanced solid cancers is 9.3% (range: 2.3–14.6%), increasing to 17.9% (range: 2.3%–36.6%) when including all patients with BMI >25 kg/m^2 [17,18]. Prevalence is generally lower in early-stage cancers [17,18] and higher in aggressive malignancies such as locally advanced or metastatic pancreatic cancer [19,20,21]. It is important to note that these figures are derived from studies using the earlier, heterogeneous, and often mass-only definitions. The adoption of the newer, more comprehensive ESPEN/EASO criteria, which integrate functional assessment and specific adiposity thresholds, may impact future epidemiological estimates and significantly improve the clinical identification of SO in cancer patients.

3.3.1. Impact on Survival

SO is an independent prognostic factor for increased mortality in several cancers:

- Advanced lung and gastrointestinal cancers: In these patients, a low skeletal muscle index (SMI) defining SO was associated with a four times higher mortality, with a relative risk of 4.2 (95% CI: 2.7–7.2) after multivariate adjustment [5].
- Resectable pancreatic cancer: Nearly 7-fold increased risk of 60-day postoperative mortality (OR = 6.76; 95% CI: 2.41–18.99, P=0.001) linked to a high visceral fat to muscle ratio [22].
- Hepatocellular carcinoma: Associated with worse disease-free survival (DFS) (OR 5.26; 95% CI 2.03–13.8, P<0.001) and reduced overall survival (OR = 2.58; 95% CI: 1.17–5.52, P=0.019) [23].
- Locally advanced gastric cancer: Significantly shorter median survival in SO patients (6 vs. 25 months, log-rank test P=0.000) [24].
- However, some studies have not found a significant association, notably in metastatic breast cancer [25] or after esophagectomy [26].

3.4. Surgical Complications

SO is associated with a major increased risk of major postoperative complications:

- Gastrectomy: In patients undergoing surgery for gastric cancer, sarcopenic obesity (SO) multiplied the risk of major post-gastrectomy complications by 6 (OR = 6.07; 95% CI: 1.90–13.36, P=0.002) [27] and the risk of surgical site infection after laparoscopic surgery by 4.6 (OR = 4.59; 95% CI: 1.18–17.78, P=0.028) [28].
- Pancreaticoduodenectomy: Patients with sarcopenic obesity (SO) also had a higher prevalence of postoperative complications (abscesses, cardiac and pulmonary complications) after pancreaticoduodenectomy [29], compared to obese non-sarcopenic patients.
- Colorectal surgery: More major complications (P=0.019) and higher 30-day mortality (P<0.001) [18].

3.5. Systemic Treatment Toxicity

The main hypothesis is that the elevated body surface area (BSA) of SO patients leads to higher absolute doses of chemotherapy, which must then be distributed, metabolized, and eliminated by a very reduced lean body mass (LBM), resulting in an increased incidence of toxicity [5].

Esophageal cancer: A significantly higher risk (OR 5.54; 95% CI 1.12–27.44) of dose-limiting toxicity (DLT) was observed as early as the first cycle of neoadjuvant chemotherapy [30].

Locally advanced gastric cancer: 100% of SO patients had to interrupt adjuvant chemotherapy due to grade 3-4 toxicity versus 28% in the rest of the cohort [24].

Melanoma on anti-PD1 immunotherapy: The presence of SO was associated with a 12 times higher risk (OR 12.0; 95% CI 1.4–103, P=0.01) of early limiting acute toxicity requiring interruption of anti-PD1 immunotherapy [31].

Non-small cell lung cancer: In NSCLC, patients with a sarcopenic obesity phenotype (high BSA/lean tissue ratio) had a 5 times higher risk of dose-limiting hematological toxicity (OR 5.21; 95% CI 1.61–16.8; $p < 0.01$). Conversely, highly muscled patients (low BSA/lean tissue ratio) had a significantly reduced risk (OR 0.19; 95% CI 0.06–0.62; $p < 0.01$), suggesting better treatment tolerance [32].

The key findings on the association between sarcopenic obesity and treatment-related toxicity are summarized in Table 1.

Table 1 Association Between SO and Treatment-Related Toxicity

Cancer Type / Treatment	Type of Toxicity Studied	Effect Size (Odds Ratio, HR, or %)	Key Finding	Reference
Esophageal (Neoadjuvant CT)	Dose-Limiting Toxicity (Cycle 1)	OR 5.54 (1.12 – 27.44)	Significantly higher early DLT risk.	[30]
Gastric (Adjuvant CT)	Grade 3-4 Toxicity (Leading to stop)	100% vs. 28% (SO vs. Non-SO)	Severe toxicity in SO group.	[24]
Melanoma (Anti-PD1)	Early Limiting acute Toxicity	OR 12.0 (1.4 – 103)	Drastically higher risk of immunotherapy interruption.	[31]
NSCLC (Platinum-based)	Hematological DLT	OR 5.21 (1.61 – 16.8)	High BSA/LBM ratio predicts toxicity.	[32]
Colorectal (Oxaliplatin - LEANOX)	Grade≥2 Peripheral Neuropathy (OIPN)	HR 0.53 (0.34 – 0.84) for OIPN 67.2% vs 42.1% (Primary endpoint success)	LBM-based dosing (Arm 3) vs standard BSA-based (Arm 2): • Significantly reduced risk of OIPN (HR 0.53, P=0.01) • Higher rate of patients completing treatment without severe neuropathy • Fewer dose reductions (P<0.001) • Better quality of life • Equivalent RFS and OS.	LEANOX (NCT03255434) [33]

CT: Chemotherapy; DLT: Dose-Limiting Toxicity; BSA: Body Surface Area, OIPN: Oxaliplatin-Induced Peripheral Neuropathy.

3.5.1. Treatment

In the absence of established pharmacotherapy, the management of sarcopenic obesity is primarily based on multimodal interventions combining physical exercise and diet [34, 35, 36].

- **Physical Exercise**: A combination of resistance training (improving protein synthesis and muscle fiber size) and aerobic activity (reducing total and visceral fat mass) is recommended for optimal effects on body composition and muscle function [35, 37, 38, 39]. Recommendations should be individualized in terms of intensity and progression [34, 35].
- **Nutrition**: The dietary strategy typically combines moderate caloric restriction (a deficit of 200-750 kcal/day) aimed at progressive weight loss, with high protein intake (1-1.2 g/kg/day) to preserve lean mass [34 , 35]. Vitamin D supplementation is often considered, although evidence for its efficacy is inconsistent [34, 40].
- **Emerging Therapies**: Several pharmacological approaches (GLP-1 receptor agonists, selective androgen receptor modulators, anamorelin, myostatin inhibitors) or surgical ones (bariatric surgery) are under investigation [34 , 41 , 42]. However, their efficacy and safety, particularly in older adults, are not yet sufficiently supported by strong evidence for routine recommendation [34, 43, 44].

In conclusion, lifestyle modifications (combined exercise and diet) constitute the cornerstone of treatment, supported by the highest level of evidence [34]. The adoption of the standardized diagnostic criteria from the ESPEN/EASO consensus should facilitate future research to evaluate the efficacy of these interventions and new therapies.

4. Discussion

This review confirms that SO is an independent and significant risk factor in the oncological journey, associated with poorer survival, increased postoperative complications, and worse tolerance to systemic therapies. Historically, heterogeneity in diagnostic definitions has likely contributed to discordant findings in the literature and obscured its true prevalence. A pivotal advance has been achieved with the 2022 ESPEN/EASO joint consensus, which now provides a standardized diagnostic framework integrating muscle function and body composition [10]. This consensus is expected to homogenize future research and improve the clinical identification of this high-risk phenotype.

The adverse outcomes associated with SO are mechanistically supported by body composition pharmacology. The work of Prado et al. [5] revealed a weak correlation ($r^2 = 0.37$) between body surface area (BSA) and lean body mass (LBM) in obese cancer patients [45]. This implies that for a BSA-calculated chemotherapy dose, the effective volume of drug distribution can vary up to threefold between individuals based on their LBM. This pharmacokinetic mismatch explains the heightened toxicity risk, as supported by studies showing LBM determines a significant portion of drug clearance variability [46]. Consequently, the most immediate and evidence-based strategy to mitigate this risk is personalized dosing. The phase II LEANOX trial [33] demonstrated that adjusting oxaliplatin dose based on LBM significantly reduced severe neuropathy and improved quality of life without compromising efficacy, providing a critical model for safer chemotherapy delivery in SO.

Despite this diagnostic and pharmacological progress, significant therapeutic challenges persist. While a multimodal approach combining resistance exercise and high-protein nutritional support forms the current cornerstone of management [36], these are extrapolations from cachexia or sarcopenia guidelines. No pharmacological agents are specifically approved for SO, and high-quality interventional trials to reverse the phenotype in the cancer context are urgently needed. The feasibility of significantly modifying body composition within narrow therapeutic windows remains a key unanswered question.

In conclusion, the management of SO in oncology is at a transition point: moving from inconsistent identification to standardized diagnosis [10], and from generalized toxicity risk to actionable dose personalization [33]. Future efforts must prioritize the clinical implementation of diagnostic standards and the development of specific, evidence-based therapeutic strategies through dedicated trials, translating growing pathophysiological understanding into improved clinical outcomes for this vulnerable population.

5. Conclusion

Sarcopenic obesity (SO) is a critical, underdiagnosed phenotype in cancer patients, independently linked to worse survival, increased surgical complications, and higher treatment toxicity. It remains hidden by normal BMI, necessitating routine body composition analysis via CT scans.

The new ESPEN/EASO consensus standardizes diagnosis by integrating muscle function with mass and fat assessment. Current management relies on multimodal lifestyle interventions (personalized exercise and nutrition). Crucially, personalizing chemotherapy dosing based on lean body mass (as in the LEANOX trial) offers a direct, evidence-based strategy to reduce toxicity.

Future priorities are implementing these diagnostic standards, developing integrated prehabilitation programs, and expanding pharmacokinetic research to personalize all cancer therapies, advancing toward precision oncology for high-risk SO patients.

Compliance with ethical standards

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Disclosure of conflict of interest

The authors declare no competing interests.

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Author contributions

All authors contributed to the conception, literature search, analysis, and manuscript drafting.

Data availability

All data are contained within the article and its references.

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