Assessing cachexia in obesity: contradiction or perfectly possible?

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Purpose of review
Existing definitions of clinically important weight loss in patients with cancer do not specifically address weight loss in patients who are obese at presentation. This review explores the clinical impact of weight loss and depletion of the skeletal muscle mass (i.e., criteria defining cancer cachexia), in patients with obesity.

Recent findings
Overweight and obese BMI values are shown by many recent studies to pose a survival advantage in patients with cancers of advanced stage, when compared with BMI in normal and underweight ranges. The classification of cancer-associated weight loss has evolved, and current grading schemes evaluate the impact of weight across the range of BMI values. Weight loss is associated with mortality in patients with BMI more than 30 kg/m^2, however this is to a much lesser degree than in patients with lower BMI values. Diagnostic imaging permits the precise assessment of skeletal muscle index (SMI) in patients with cancer, and it has been clearly shown that while usually quite muscular, obese patients can have profound muscle depletion (i.e., sarcopenia), independent of the presence of weight loss. Muscle depletion associates strongly with mortality in obese patients, as well as with complications of cancer surgery and systemic therapy.

Summary
It would seem contradictory to diagnose concurrent obesity and cachexia, as these terms represent opposite ends of the weight spectrum. Weight loss can occur in anyone with cancer, however its priority for clinical management may be lesser in obese versus low body weight individuals. Sarcopenic obesity is strongly associated with a poor clinical outcome and deserves further research, diagnosis in clinical practice, and new strategies for mitigation.

Keywords
cachexia, cancer, obesity, weight loss

INTRODUCTION

There were nearly 20 million new cases of cancer and 9.7 million deaths from cancer in the year 2022. Obesity is extremely widespread globally and is driving up the population attributable fraction of obesity-related cancers [1,2]. The impact of these trends is that in many countries, patients with a diagnosis of cancer are increasingly likely to be obese at presentation. This situation is associated with a variety of conundrums. One difficult and confusing question is that while obesity and associated metabolic disorders are clearly risk factors for cancer in the first place, there is a widely discussed obesity 'paradox' wherein for patients with a diagnosis of cancer, obese patients may have a survival benefit. Another issue is that the upward shift in body weight renders definitions of clinically significant weight loss increasingly unclear. Cancers of advanced stage are associated with involuntary weight loss leading to progressive depletion of body energy (fat) and protein (muscle) reserves. Weight loss is the cardinal criterion defining cancer cachexia; however, the impact of weight loss in individuals having a very large fat reserve at the start of a cancer journey is unclear. May we label an obese patient with cancer as 'cachexic' if they are losing weight? Sarcopenic obesity is an unusual

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KEY POINTS

- Obesity confers increased duration of survival in patients with advanced cancer. Studies on classification of cancer-associated weight loss reveal that the risk of mortality associated with weight loss scales inversely with BMI, with the lowest mortality in patients of the highest body weights.
- Sarcopenic obesity is a prevalent body habitus associated with high risks of morbidity and mortality. While research evidence continues to build, sarcopenic obesity is not yet being detected in clinical practice.
- Developing the knowledge base for best practices in the clinical management of obese patients with cancer should be a priority.

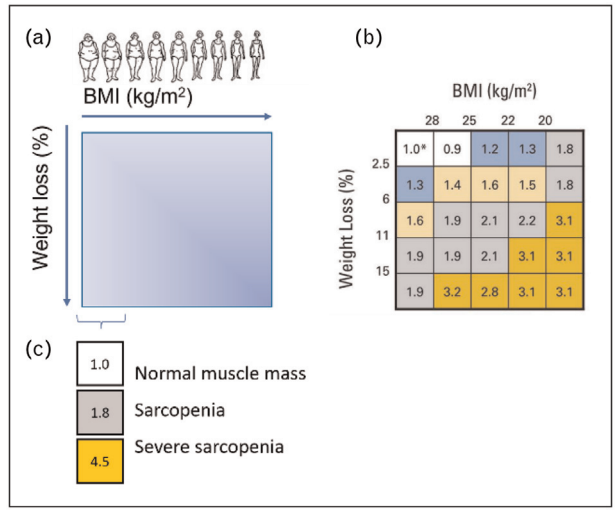

FIGURE 1. Weight loss has muted prognostic impact, but sarcopenia has a high prognostic impact in patients with obesity. (a) Landscape of BMI and weigh loss. Patients with cancers present with a wide array of BMI and weight loss history. (b) Weight Loss Grades according to Hazard Ratio for death. Color coding reflects Grade 0 (white), Grade 1 (blue), Grade 2 (yellow), Grade 3 (grey), and Grade 4 (orange) weight loss based on adjusted ∗Hazard Ratio for mortality [5]. (c) Patients with obesity are at risk for mortality according to the concurrent presence of reduced, or severely reduced skeletal muscle mass. Hazard ratio for mortality are presented for the patients with high BMI >28 kg/m^2 [7].

body habitus, characterized by muscle depletion which can be very profound, in individuals at the opposite extreme of the fat mass distribution. Loss of muscle deeply undermines the survival advantage of obesity, and sarcopenic obesity also bodes badly for treatment tolerance. Is a sarcopenic obese patient 'cachexic' if the wasting is exclusive to their muscle mass? To gain a clearer understanding of these many questions, the purpose of this review was to explore the clinical impact of weight loss and depletion of the skeletal muscle mass (i.e., criteria defining cancer cachexia) in patients with obesity.

WEIGHT LOSS CAN OCCUR IN PATIENTS OF ANY BMI, BUT THE IMPACT OF WEIGHT LOSS IS LESSER IN PATIENTS WITH OBESITY, COMPARED WITH NORMAL WEIGHT

Weight loss is the cardinal diagnostic criterion of cancer cachexia [3]. Percentage weight loss is an index of severity, but there had been marked inconsistencies as to what % loss oncologists considered clinically important until more clearly defined criteria for the classification of cancer-associated weight loss emerged [4▪▪,5]. Weight loss grading in cachexia is based on the concept reached by international consensus [3] that severity can be classified according to degree of depletion (of body reserves) in combination with the rate of ongoing weight loss, that is, a fall of 5 kg/m^2 in BMI from an initial value of 22 kg/m^2 has more severe implications than the same loss from an initial value of 35 kg/m^2. Based on this premise, analyses were conducted to assess the landscape of BMI and weight loss (Fig. 1a) across five strata of BMI and five strata of weight loss (Fig. 1b). What is evident from this analysis is that there is a risk of mortality associated with weight loss in heavier individuals; however, this risk is considerably lower than in patients with any lower starting BMI [5]. For example, a 10% weight loss in a person of BMI 30 is Grade 2 weight loss, whilst a 10% weight loss in a person of BMI 19 is Grade 4 weight loss, with corresponding differences in the hazard ratio for death (1.6 versus 3.1) [5].

These weight loss grades depicted have been validated in populations of more than 16 000 patients from Asia [4▪▪] and more than 12 000 patients from Europe/Canada [6] suggesting that these associations between mortality are generalizable to different populations. While most results were from samples with mixed primary tumors [5,6], the same relationships exist in specific primary tumor sites such as head and neck cancer [7]. Xie *et al.* [8] described the effect in simple terms, stating that patients with obesity must lose nearly twice as much weight as normal weight patients, to have the same hazard ratio for mortality.

The simple conclusion that weight loss is a less significant risk for patients with high body weights is confirmed by an ever-increasing number of studies that map to the term 'obesity paradox'. These are studies of the simple association of BMI with survival, usually without consideration of weight loss.

This collective of findings spans an ever-larger number of patients and (systemic) treatment plans in which obese subsets usually classified according to the WHO categories (BMI >30 kg/m^2) are seen to survive longer than those in the normal body weight ranges (BMI 18.5–24.9 kg/m^2). Ge *et al.* [9▪▪] provided evidence from two large prospective studies from the United States ($n = 4393$) and China ($n = 9486$), including patients with cancer in the US National Center for Health Statistics National Health and Nutrition Examination Survey (NHANES) and from the Investigation on Nutrition Status and Clinical Outcome of Common Cancers (INSCOC) in China. These studies demonstrate that across the span of BMI categories underweight, normal weight, overweight, and obese, the risk of mortality falls in a continuous fashion. This appears to be the case across countries in which rates of obesity and cancer prevalences differ. In the INSOC cohort, the fully adjusted hazard ratio was 0.69 [95% confidence interval (95% CI) 0.61–0.78] in patients with BMI more than 30 kg/m^2 compared to the normal-weight range. The finding that obesity is associated with reduced risk of mortality is repeatedly demonstrated in cohorts of patients within specific primary tumor sites and treatment plans. In a retrospective study of Asian patients who were treated with immune checkpoint inhibitors for advanced non-small cell lung cancer, BMI more than 28 kg/m^2 was associated with longer overall survival, independent of several clinical covariates (hazard ratio, 0.64; 95% CI 0.52–0.80) [10]. Perhaps there is no paradox. Obesity is rarely understood to confer an advantage. However, if one considers that body mass contains 'reserves' in the sense of stored fuel(s) held in contingency for conditions of reduced food availability and/or increased metabolic demand, it makes sense that high body reserves would be advantageous. In cancer, both nutritional deficit and alterations in metabolism in varying degrees contribute to drawing down each patient's reserves.

Increasingly, researchers are evaluating the association of adipose tissue mass and specific sub-compartments thereof, in the association of obesity with enhanced survival. In a study of patients with non-small cell lung cancer, Tao *et al.* [11] explored association of mortality with specific radiological measures of subcutaneous fat and pericardial fat, rather than overall BMI. In multivariable Cox regression analysis, after adjusting for clinical variables, increased subcutaneous fat index (hazard ratio = 0.56, 95% CI 0.47–0.66) and increased pericardial fat index (hazard ratio = 0.47, 95% CI 0.40–0.56) were associated with longer overall survival. This was true across cancer stages 1–3 and Stage 4, and in patients who had surgery or no surgery. Many questions have been raised about the importance of visceral obesity, as distinct from subcutaneous fat or weight/BMI-based measures of obesity. On the one hand, a survival benefit of high VAT values has been suggested, as for other anatomic sites of adipose tissue. Matsui *et al.* [12▪] produced a systematic review on the association of visceral adipose tissue with postoperative outcome in upper gastrointestinal cancer, noting 24 studies with $n = 3407$ patients. They concluded that compared with low visceral adipose tissue, high visceral adipose tissue assessed by CT may improve overall survival (hazard ratio: 0.69; 95% CI 0.55–0.87). On the other hand, some specifically deleterious characteristics of excess visceral fat cannot be forgotten, that is, its inappropriate secretion of adipokines, proinflammatory cytokines, and growth factors, fostering the carcinogenesis of obesity-related tumors.

IS SARCOPENIC OBESITY A FORM OF CACHEXIA?

Diagnostic imaging has provided a remarkable opportunity to reveal deficits of muscle mass that can be very profound. Such deficits had previously gone undetected, especially when overlaid by a considerable mantle of adipose tissue. For about 15 years, researchers have been taking advantage of cancer imaging acquired during routine cancer care to detect sarcopenic obesity. Jurdana and Cemazar [13] provide an updated review and perspective on sarcopenic obesity in oncology. While noting that different definitions of sarcopenic obesity make it difficult to determine its exact prevalence, it is reported to be present in 15–36% of patients with a BMI more than 30 kg/m^2. These authors also discuss some of the biological pathways leading to sarcopenic obesity and the metabolic dysfunction which may occur in the interplay among tumor, muscle, and adipose tissue.

Accumulated evidence supports the contention that severe skeletal muscle depletion undoes most of the survival benefit of high BMI. This is illustrated in Fig. 1c, in which hazard ratio for mortality is illustrated for the patients with BMI more than 28 kg/m^2, according to the degree of skeletal muscle depletion. Notably, about 11% of patients with high BMI and limited or no weight loss have severe sarcopenia, associated with an increased risk of death (hazard ratio, 4.54; 95% CI 2.92–7.06) [7].

Studies on sarcopenic obesity in cancer spans many specific tumor groups and treatment plans and continue to add to the body of evidence. The complexity of the relationships between sarcopenic obesity and outcome is evident (Table 1). Three recent meta-analyses concerned sarcopenic obesity

Table 1. Recent literature on sarcopenic obesity in patients with cancer

Ref.	Population	Outcome of sarcopenic obesity Hazard ratio (HR) or odds ratio (OR) [95% confidence interval (CI)] from multivariable analyses. Comparison sarcopenic obese versus all other body types (reference), unless indicated otherwise
Meta-analyses		
Li *et al.* [14] $n=11\,970$	Primary liver cancer	HR 2.87, CI 2.23–3.70, overall survival HR 2.28, CI 1.54–3.35, recurrence-free survival
Wang *et al.* [15] $n=8729$	Gastrointestinal cancer, surgery	OR 1.30, CI 1.03–1.64 total complications OR 2.15, CI 1.39–3.32 complications Clavien-Dindo ≥IIIa HR 1.73, CI 1.46–2.06, overall survival HR 1.41, CI 1.20–1.66, disease-free survival
Gao *et al.* [16] $N=10\,004$	Any cancer	HR 1.83, CI 1.41–2.38, overall survival OR 3.01, CI 2.08–4.33, postoperative complications OR 5.69, CI 2.76–11.7, prolonged stay in hospital OR 5.54, CI 1.12–27.4 chemotoxicity
New studies		
Chilioro *et al.* [17]	Rectal cancer, locally advanced, radiation therapy	HR 2.24, CI 0.69–7.21, overall survival
Wagner *et al.* [18]	Colon cancer, metastatic, surgery	HR 3.41, CI 2.45–7.65, disease-free survival
Medici *et al.* [19]	Cervical cancer, stage 1–2–3; definitive RT and chemotherapy	HR 5.29, CI 1.30–21.55, disease-free survival HR 2.65, CI 1.28–5.49, overall survival
Sohal *et al.* [20]	Pancreatic cancer, resectable, neoadjuvant chemotherapy	No effect of sarcopenic obesity, overall survival
Guarneri *et al.* [21]	Pancreatic cancer, resectable	No effect of sarcopenic obesity, overall survival
Bawaji *et al.* [22]	elective colon resection for nonmetastatic colon cancer	OR 2.15, CI 1.14–3.69, overall morbidity OR 5.07, CI 1.22–20.93, 30-day mortality OR 2.95, CI 1.41–6.18, anastomotic leak
Kalid *et al.* [23]	spinal metastases, surgical treatment Case-control comparison of obese patients ± sarcopenia	OR 6.00, CI 1.69–21.26, nonhome discharge OR 3.27, CI 1.01–10.62, 30-day readmission OR 4.85, CI 1.29–18.26, 90-day mortality OR 3.78, CI 1.17–12.2, 1-year mortality
Yamagishi *et al.* [24]	Gastric cancer, gastrectomy	OR 3.95, CI 1.39–11.2, overall survival
Hayashi *et al.* [25]	Hepatocellular carcinoma, hepatectomy	HR 4.08, CI 1.37–12.1, posthepatectomy bile leakage
Li *et al.* [26]	Hepatocellular carcinoma, trans-arterial chemoembolization	HR 8.35, CI 4.96–14.1, sarcopenic visceral obesity, overall survival HR 5.23, CI 3.41–8.02, sarcopenic obesity, overall survival
Surov *et al.* [27]	Multiple myeloma	HR 2.30, CI 0.90–5.63, overall survival
Juez *et al.* [28]	Gastrectomy, gastric cancer	OR 2.82, CI 1.1–7.1, Clavien-Dindo complications ≥IIIb

in primary liver cancer [14], gastrointestinal cancers [15], and any cancer [16]. These attest to consistent risks for mortality as well as serious complications after cancer surgery, and the fact that patients characterized for sarcopenic obesity in research now number in the tens of thousands. A caveat on the interpretation of available data is strongly emphasized by the authors of the meta-analyses that sarcopenic obesity is defined inconsistently. Obesity is not only defined by BMI more than 30 kg/m^2, but also by more than 25 kg/m^2 especially in Asian populations, or by total adipose tissue (area or index) or by visceral adipose tissue alone (area or index). The skeletal muscle index threshold values for sarcopenia also vary widely, selected from the literature in some cases, or determined empirically within the data set, and while sarcopenia is most often diagnosed on total abdominal muscle index, in some cases only a single muscle (e.g., psoas) is used. Wang *et al.* [15] discussed high heterogeneity in the classification criteria for sarcopenic obesity, noting that more stringent criteria (resulting in <10% prevalence) were associated with increased hazard ratios and increased statistical significance

for adverse outcomes compared to the high prevalence (>20%) based on less stringent criteria.

In Table 1, recent new studies [17–27] are indicated, exploring various outcomes associated with sarcopenic obesity in a wide array of cancers and treatment settings. The studies all included multivariable analyses, to detect the independent prognostic value of sarcopenic obesity after taking other clinical and demographic factors into account. In most studies, the outcome of patients with sarcopenic obesity was compared with the outcome of all other body types combined (reference group). A single study [23] had a case–control design in which obese patients with and without sarcopenia were compared with each other. Of the recent studies in Table 1, three were negative for association of sarcopenic obesity and overall survival, in resectable pancreatic cancer, with [20] and without [21] neoadjuvant chemotherapy and in locally advanced rectal cancer treated with radiation [17]. Sarcopenic obesity was associated with poorer overall survival, disease-free survival or both, in all studies where this outcome was reported, and in surgical setting, sarcopenic obesity was associated with complications of surgery, including serious complications by Clavien–Dindo grade [28], anastomotic leak after colon cancer surgery [22] and posthepatectomy bile leakage [25], short term (30-day) mortality [22,23] as well as nonhome discharge [23]. Li *et al.* [26] noted that sarcopenic visceral obesity showed a higher hazard ratio for mortality than sarcopenic obesity based on BMI, in patients with hepatocellular carcinoma treated with trans-arterial chemoembolization; however, this specific body habitus has otherwise rarely been investigated.

Collectively, current findings point to multiple severe consequences of sarcopenic obesity with several of these associated with a relative risk greater than five-fold that of patients without sarcopenic obesity, in specific primary tumor sites and treatment plans. The authors of meta-analyses stress the need to improve the quality of evidence [12▪] have been eloquent concerning the limitations of this literature, noting inconsistency, sampling bias, and publication bias. Gao *et al.* [16] found results for the relationship between sarcopenic obesity and chemotherapy toxicity to be the most inconsistent and controversial.

INTERPRETATION/CONCLUSION

I return initially to the question raised by the title of this presentation 'Assessing cachexia in obesity: contradiction or perfectly possible?' Cachexia is a condition defined by progressive weight loss, which if intense or occurs over an extended period can culminate in exhaustion of the body reserves of energy and protein. Diagnostic criteria for cancer cachexia include weight loss and muscle depletion. Clearly, obese patients with cancer can have one or both, however it may not be appropriate or useful to label them as being affected by cachexia. The term cachexia has a widespread and popular understanding of weight loss leading to underweight (very low BMI) or emaciation. A concurrent diagnosis of both cachexia and obesity is contradictory, as these represent opposite ends of the spectrum of weight-related disorders, in most people's minds.

I suggest that we simply need to develop the knowledge base for best practices in the clinical management of obese patients with cancer. Obesity has many implications in this context, and a good example of effort taken to address the unique needs of patients with obesity is the American Society for Clinical Oncology Guideline for appropriate dosing of systemic antineoplastic agents in obese adults with cancer [29]. However, the unique situation of obese patients with cancer at risk for weight loss has not yet been addressed in any clinical practice guideline. The evidence presented in the preceding sections suggests that obesity is associated with a longer survival time, but any survival benefit is obliterated by underlying sarcopenia, and a host of other serious problems arise. The relative risk of poor outcomes related to sarcopenic obesity (Table 1) is often considerably greater than the risks associated with weight loss of a low grade. Thus, we may consider sarcopenic obesity a priority concern. More evidence of better quality is needed to assess its impact. To that end, it would seem important to use standardized metrics for the assignment of sarcopenic obesity status, permitting the aggregation and meta-analysis of data for more rigorous interpretation.

How might we improve the nutritional and metabolic care of obese patients with advanced stages of cancer? From the oncologist's perspective, and that of the cancer surgeon, the goal is to optimize cancer treatment. Complications of cancer, surgery, and toxicity of chemotherapy appear to be important outcomes of sarcopenic obesity, but because we operate blind to the presence of sarcopenic obesity in day-to-day clinical practice, these complications are not preventable. We must more rigorously quantify these risks and develop strategies for mitigation.

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Conflicts of interest

V. Baracos is a consultant for Pfizer and Nestle Health Science.

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- ■ of special interest
- ■■ of outstanding interest

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