Understanding the Complexities of Cancer-Associated Cachexia

A clinical update on diagnosis, pathophysiology & management

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Cachexia, from the Greek *kakos hexis* ('bad condition'), has been recognised since the time of Hippocrates as a syndrome of profound wasting and inevitable mortality.[1] Modern definitions describe cachexia as a complex, multifactorial metabolic syndrome characterised by progressive loss of skeletal muscle mass, with or without loss of fat mass, that cannot be fully reversed by conventional nutritional support.[2]

Introduction

Although cancer-associated cachexia (CAC) is the most extensively studied form, the condition manifests in various inflammatory conditions, including chronic heart failure, chronic kidney disease, chronic obstructive pulmonary disease and rheumatoid arthritis.[3] CAC affects approximately 60–80% of individuals with advanced malignancy and contributes to more than 25% of cancer-related deaths, often due to respiratory or cardiac failure secondary to severe muscle depletion.[4]

In spite of these stark figures, CAC remains under-recognised and undertreated, representing one of the most significant unmet needs in oncology.[5] While its designation as a Cancer Research UK Grand Challenge reflects growing scientific interest, awareness among healthcare professionals remains limited.[6-8] This is compounded by a reluctance to address CAC directly; dietitians, for example, fear such discussions encroach on sensitive end-of-life issues, yet evidence suggests patients and carers value clear explanations.[9, 10] In the author's experience, when dietitians do not initiate conversations about CAC, it often remains entirely overlooked.

This article provides a clinical update on CAC, aiming to support dietitians to diagnose and manage CAC confidently and compassionately within routine practice.

Diagnosis

The international consensus definition proposed by Fearon *et al.*[2] remains the most widely cited framework for diagnosing CAC (**Table 1**). The framework also describes a continuum from precachexia to refractory cachexia, reflecting increasing metabolic derangement and diminishing reversibility.

Whilst a useful clinical tool, a key limitation of this definition is the absence of systemic inflammation as a diagnostic requirement, despite its central role in cachexia pathophysiology.[5] Additionally, the term 'simple starvation,' as used within their diagnostic criteria, is not defined, allowing for varying interpretations. More recently, the European Society for Medical Oncology (ESMO) recommends use of the Global Leadership Initiative on Malnutrition (GLIM) criteria (**Table 2**), conceptualising CAC as 'disease-related malnutrition with inflammation'.[11]

GLIM requires the presence of at least one phenotypic criterion and one aetiologic criterion (such as systemic inflammation) to diagnose malnutrition.[12] A modified Glasgow Prognostic Score (mGPS) of ≥1 satisfies GLIM criteria for systemic inflammation, and is associated with poorer functional status, reduced treatment response and inferior survival[13-15] (**Table 3**). Therefore, in contemporary dietetic practice, CAC is diagnosed in patients with ≥1 phenotypic criteria along with an mGPS of ≥1.

The GLIM criteria are now a vital asset in oncology nutrition as they offer a unified approach to identify both cachexia and malnutrition more broadly in patients with cancer. GLIM criteria have demonstrated strong prognostic validity in oncology, reliably predicting treatment tolerance, quality of life and survival.[17]

However, a practical framework to identify precachexia is lacking. Nutritional screening tools used to trigger GLIM assessment are insufficiently sensitive to detect early muscle loss, meaning opportunities for early intervention are frequently missed.[18, 19] Neglecting precachexia is comparable to delaying oncology treatment until the onset of symptoms – by which point the disease is already advanced.[13] With the expansion of prehabilitation, there is hope that more cases will be managed inadvertently, at least until a more direct framework is established.

Underlying pathophysiology

The aetiology of CAC is highly multifactorial, driven by complex tumour-host interactions involving tumour-derived factors, systemic inflammation, metabolic disturbance, and dysregulated immune and endocrine processes.[20] While inflammation is a fundamental component of host defence, tumours exploit inflammatory pathways to create a microenvironment that promotes angiogenesis, immune suppression and malignant progression.[21]

Although initiated locally, tumour-derived signals amplify into a systemic host inflammatory response via cytokine spill-over, hepatic acute-phase activation and neuroendocrine reprogramming.[21] Pro-inflammatory cytokines – particularly interleukin-6 (IL-6), tumour necrosis factor-α (TNF-α), and interleukin-1β (IL-1β) – produced by both tumour and host immune cells are central to the development of CAC, and drive disease progression and metastasis.[21]

Clinically, this inflammatory milieu drives patients into negative energy balance through anorexia, early satiety, taste changes and nausea.[2, 3] In parallel, host metabolism is reprogrammed to favour energetically inefficient pathways, including the activation of so-called 'futile cycles' such as the Cori cycle, which consume more energy than they generate while preferentially supplying substrate to the tumour.[22] Browning of white adipose tissue further exacerbates hyper-metabolism and energy dissipation.[23]

Skeletal muscle wasting is driven by suppressed protein synthesis and accelerated proteolysis, mediated by the ubiquitin-proteasome system and autophagy.[23] Simultaneously, systemic insulin resistance and altered lipase activity drive adipose tissue depletion via pathologically accelerated lipolysis.[23] Collectively, these mechanisms ensure the continuous mobilisation of host energy stores to fuel tumour growth, explaining why dietary restriction merely accelerates the loss of host tissue rather than 'starving' the malignancy.[24]

Clinically, this inflammatory burden manifests as 'sickness behaviour'; a state of profound fatigue, apathy and anorexia. For families, CAC is best conceptualised as a 'chronic flu-like state', providing a relatable context for the patient's profound malaise and lack of interest in food.[25]

Resistance to nutritional support

In their seminal paper, Fearon *et al.* emphasised that conventional nutritional support cannot fully reverse the progressive skeletal muscle loss observed in CAC.[2] In practice, this means that *significant* reversal of muscle wasting is very unlikely, even in the context of an optimised dietary intake.[26] In the refractory phase, skeletal muscle loss may become unstoppable; prior to then, however, losses can be slowed, halted or even partially reversed.[26] Indeed, a window of anabolic potential seems to exist when survival is greater than 90 days.[27]

Thus, nutritional support in CAC is not futile, though its benefits are less pronounced than would be observed in malnutrition more broadly.[11] Invasive interventions like tube feeding, therefore, have a higher risk-to-benefit ratio in CAC.[28] It is important that patients with CAC and their families are aware that, in the refractory phase, even artificial feeding does not improve survival, and in the case of parenteral nutrition, generates more serious adverse events.[29]

Table 1: International consensus criteria on CAC[2]

Stage	Diagnostic criteria	Management
Precachexia	• Weight loss of ≤5% with anorexia and metabolic change (e.g. impaired glucose tolerance)	• Monitor • Preventive intervention
Cachexia	• Any one of the below: o Weight loss >5% within 6 months *(in absence of simple starvation)* o BMI <20 and any degree of weight loss >2% o Sarcopenia and any degree of weight loss >2%	• Multimodal management according to phenotype (with prioritisation of reversible contributory factors)
Refractory cachexia	• Variable degree of cachexia • Cancer disease both procatabolic and not responsive to anticancer treatment • Low performance status • <3 months expected survival	• Symptom palliation • Psychosocial support • Ethical discussion regarding nutritional support

Source: Fearon K, *et al.* (2011) Definition and classification of cancer cachexia: an international consensus. Lancet Oncol.; 12(5): 489-495.

Table 2: GLIM phenotypic and aetiologic criteria for the diagnosis of malnutrition[12]

Phenotypic criteria (≥1 required)	Aetiologic criteria (≥1 required)
Non-volitional weight loss • >5% within past 6 months or • >10% beyond 6 months	Reduced food intake • ≤50% energy requirements for >1 week, or • Any reduction for >2 weeks
Low BMI • <20 kg/m^2 if <70 years • <22 kg/m^2 in ≥70 years	Reduced food assimilation/absorption • Chronic gastrointestinal conditions causing malabsorption
Reduced muscle mass • Low fat-free mass index, appendicular skeletal muscle mass, calf circumference or handgrip strength	Disease burden/inflammatory condition • Acute disease or injury • Chronic disease with systemic inflammation

Source: Cederholm T, *et al.* (2019) GLIM criteria for the diagnosis of malnutrition - A consensus report from the global clinical nutrition community. Clin Nutr.; 38(1): 1-9.

Table 3: Modified Glasgow Prognostic Score[13]

Biochemistry*	mGPS
CRP ≤10 mg/L	0
CRP >10 mg/L and albumin ≥35 g/L	1
CRP >10 mg/L and albumin <35 g/L	2

*Serial measurements of CRP are recommended to distinguish acute inflammation from persistent cancer-related inflammation.[16]
Source: McGovern J, *et al.* (2024) Global Leadership Initiative on Malnutrition cachexia: an inflammation-first approach for the diagnosis of disease-related malnutrition. Curr Opin Clin Nutr Metab Care.; 27(5): 393-396

Cachexia & anticancer treatment: A poor match-up

Oncologists generally prescribe anticancer treatment based on the body surface area (BSA) – the total skin surface of the body – with limited consideration of skeletal muscle status.[30] Skeletal muscle, however, plays important roles in drug distribution and clearance, meaning that BSA-based dosing in CAC can result in suboptimal drug exposure, whereby treatment is less effective and more toxic.[30] Indeed, CAC promotes earlier time to treatment failure and higher mortality after first-line chemotherapy.[31] Moreover, greater handgrip strength (HGS) at the start of palliative chemotherapy is associated with significantly better survival in older patients with advanced cancer.[32]

Differentiating cachexia from malnutrition

In practice, differentiating cachexia from non-inflammatory malnutrition can be challenging. Patients may meet diagnostic criteria for CAC with weight loss driven by non-inflammatory factors such as mechanical obstruction, dysphagia, treatment-related toxicity, psychological distress or pancreatic exocrine insufficiency.[11] Misattributing weight loss to cachexia may lead practitioners to assume nutritional interventions will be less effective and therefore withhold beneficial treatment.[33] Indeed, guidelines highlight CAC as a contraindication to artificial nutrition in advanced cancer.[28] Where cachexia status is uncertain, further assessment of systemic inflammation may help contextualise weight loss and guide expectations of nutritional response. An mGPS of 2 strongly indicates an underlying cachectic process and is recognised by ESMO as a contraindication to home parenteral nutrition.[11, 14] The neutrophil-to-lymphocyte ratio (NLR) is another marker of systemic inflammation, with values greater than 3 considered indicative.[15, 34] Serum lactate dehydrogenase (LDH) provides further insight; levels above 250 U/L are associated with increased tumour glycolysis and lactate production, reflecting energy-inefficient metabolic cycling and disease progression.[34] In equivocal cases, a time-limited trial of nutritional support with predefined outcome measures, such as improvements in HGS, may be necessary.[11, 28]

Not just an end-of-life issue

Recent evidence shows that skeletal muscle wasting can begin up to two years prior to a pancreatic cancer diagnosis – even in patients with localised, curable disease.[35] Crucially, this muscle loss occurs regardless of tumour location, supporting a systemic metabolic aetiology rather than being a mere consequence of pancreatic exocrine insufficiency. Indeed, in a cohort of 911 patients with newly diagnosed pancreatic cancer, approximately two-thirds presented with CAC; this prevalence was not dictated by tumour stage or location.[36]

In a cohort of 313 non-small cell lung cancer patients with only stage I disease, 66 (21%) had CAC at diagnosis.[37] Overall, cachexia is thought to affect about one third of patients with early-stage cancer.[38] These findings highlight that CAC can manifest early in the cancer trajectory, not just in the terminal phase.

Treating cancer-associated cachexia

Intervention data in CAC remain limited, not only by the small number of clinical trials but also by heterogeneity in cachexia definitions and outcome measures used to assess intervention effectiveness.[39, 40] This variability hampers comparison between studies and underscores the urgent need for global consensus on trial design and clinically meaningful endpoints.

Among nutritional supplements, only omega-3 fatty acids are recommended in major clinical guidelines. ESMO suggests omega-3-enriched oral nutritional supplements (ONS) for patients with CAC undergoing anticancer treatment, to attenuate loss of lean body mass.[11] Similarly, ESPEN suggests omega-3 supplements for those with advanced cancer with, or at risk of, malnutrition to improve appetite and lean body mass.[41] While these recommendations are graded as 'weak' due to low-certainty evidence, supporting trials typically utilise doses of approximately 2–2.2 g of eicosapentaenoic acid (EPA) per day. Combined intakes of EPA and docosahexaenoic acid (DHA) up to 5 g daily are considered safe, though fish oil should be avoided in patients receiving ibrutinib due to an increased risk of bleeding.[42]

A recent review found limited evidence supporting the effectiveness of exercise-only interventions (including resistance training) in mitigating skeletal muscle loss.[43] In CAC, meaningful tissue accretion may be difficult or impossible to achieve without physical activity, however.[11]

Given the complexity of CAC, it is logical that a multimodal approach combining nutritional, physical and pharmacological treatment is necessary for its effective management. However, despite strong theoretical rationale for multimodal interventions, clinical evidence supporting their efficacy remains limited.[22]

Recently, ESMO published clinical practice guidelines for CAC in adults.[11] Key nutritional recommendations are summarised in **Table 4**. Currently, no pharmacological treatment for CAC is licensed. Short courses of corticosteroids may transiently improve appetite and well-being, albeit with limited effect on body weight and survival.[11] Effective management of CAC primarily relies on successful treatment of the underlying malignancy, though targeted therapies addressing key metabolic pathways are under active investigation and appear promising.[44]

Table 4: Key ESMO recommendations on the nutritional management of cancer-associated cachexia[11]

	Recommendation
Nutritional screening	Implement regular nutritional screening for all patients receiving anticancer treatment and in those with an expected survival of more than 3-6 months.
Nutritional support	In patients with inadequate food intake, nutritional interventions are recommended. In patients with an expected survival of more than 3-6 months and in those receiving anticancer therapy, these interventions should be escalated, as required. In other situations, low-risk interventions (e.g. counselling and ONS) are preferred.
Multimodal management	Nutritional intervention should be combined with physical activity (resistance exercise 2-3 times per week as well as moderate aerobic training), symptom control and psychosocial support.
Energy requirements	Total energy expenditure is often normal (25-30 kcal/kg/bodyweight/day) but may be unpredictably low or high in some patients.
Protein requirements	A protein intake of 1.2, and possibly up to 2 g/kg/bodyweight/day, may be required to balance protein synthesis.
Macronutrient balance	Regimens with fat accounting for half of the non-protein calories are recommended – fat is often metabolised more efficiently than carbohydrate.
Meal pattern	Dietary counselling should emphasise a 'little-and-often' approach.

Source: Arends J, *et al.* (2021) Cancer cachexia in adult patients: ESMO Clinical Practice Guidelines. ESMO Open.; 6(3): 100092.

Monitoring interventions

In monitoring CAC, ESMO recommends regular reassessment of nutritional status that extends beyond body weight, with emphasis on skeletal muscle mass and physical function.[11]

In research settings, skeletal muscle index and skeletal muscle density can be quantified using routine CT images at the level of the third lumbar vertebra, allowing accurate identification of sarcopenia and myosteatosis without additional patient burden.[45] Wider clinical adoption of this approach will improve monitoring and evaluation of cachexia-directed interventions. A more accessible, albeit less accurate, means of body composition analysis is possible with bioimpedance analysis.[11]

Functionally, HGS remains a useful tool in clinical practice as it indicates muscle strength as well as the general health status.[39] It is thought that a change in HGS of 5-6.5 kg is likely to be clinically meaningful.[46] Eastern Cooperative Oncology Group (ECOG) performance status offers another useful functional metric.[39]

Reliance on body weight alone has important limitations. Weight stability appears satisfactory on the surface, though it may mask ongoing skeletal muscle loss (with concurrent fat gain).[18] As muscle loss is the defining pathological feature of CAC, assessment of muscle mass and function should be prioritised where feasible to evaluate treatment response and guide management.[11] Nevertheless, preventing further unintentional weight loss remains an important therapeutic goal, except in refractory cachexia where losses may be unavoidable.[11, 26]

Conclusion

CAC is a debilitating, multifactorial syndrome that remains significantly under-recognised and undertreated despite its substantial death toll. Driven by systemic inflammation and metabolic reprogramming, CAC prioritises tumour fuel supply at the expense of host muscle and fat stores, sometimes long before cancer is diagnosed. As CAC progresses, it becomes increasingly resistant to nutritional support. While nutritional support offers meaningful benefit in earlier stages, its impact is considerably diminished in refractory cachexia.

The GLIM criteria allows clinicians to conceptualise CAC as disease-related malnutrition with inflammation. Dietitians are well-placed to diagnose CAC using this framework and should take opportunities to openly discuss this distressing and clinically significant, yet often unmentioned, syndrome with patients and families. Effective management requires treatment of both the cancer and the host: alongside optimal anticancer therapy, multimodal management of CAC should be implemented combining nutritional support (± omega-3s), physical activity, symptom control and psychosocial care.

Advances in research and wider access to objective muscle assessment tools offer hope for improved monitoring and more effective treatment pathways, revolutionising dietetic care in oncology in the not-too-distant future.

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