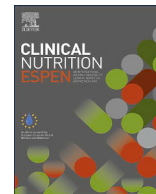




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Original article

Get a grip! – Baseline handgrip strength and sarcopenia as predictors of survival in patients with head and neck squamous cell carcinoma

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SUMMARY

Background: Low handgrip strength (HGS) and sarcopenia are common in patients with head and neck squamous cell carcinoma (HNSCC). This study aimed to explore associations between baseline HGS, fat-free mass index (FFMI), nutritional indices, and survival.

Methods: This was a prospective observational sub-study of a randomized nutritional intervention trial, including 50 male patients with HNSCC undergoing curative-intent treatment (surgery and/or (chemo)radiotherapy). Sarcopenia was defined as low HGS (<27 kg) and FFMI (<17 kg/m²). Chi-square, Kaplan–Meier, and Cox analyses were used.

Results: Low HGS was observed in 16%, low FFMI in 46%, and sarcopenia in 12%. Patients with low HGS had lower body weight, BMI, and FFMI, alongside more malnutrition, elevated CRP, and heavy smoking. Low HGS and sarcopenia were associated with shorter overall survival (HR 3.7, [95% CI 1.5–9.1] and 5.5, [2.2–14.5], respectively); FFMI was not. Adjustment removed significance. Findings should be interpreted cautiously due to the small, all-male cohort size.

Conclusion: In this small exploratory cohort HGS may serve as a simple screening surrogate for sarcopenia and survival.

Clinical trial registration: www.ClinicalTrials.gov, identifier NCT02159508.

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1. Introduction

In patients with head and neck cancer (HNC), the tumor itself, its location, and treatment may cause significant symptoms which interfere with eating and give rise to malnutrition [1]. In previous work, we have reported a Global Leadership Initiative on Malnutrition (GLIM)-based malnutrition prevalence of 37% in patients with HNC [2]. This is slightly higher than the prevalence rates of 25%–30% observed in other published HNC cohorts [1,3]. Related

loss of muscle mass is seen in 6%–73% of patients with HNC and sarcopenia using EWGSOP2 criteria in 21%–48% [1,3–11].

A shared feature in diagnosing malnutrition and sarcopenia is the assessment of muscle mass. In many earlier studies, loss of muscle mass was referred to as sarcopenia [12], but, as early as 2010, the European Working Group of Sarcopenia for Older Persons criteria (EWGSOP1) highlighted the fact that defining sarcopenia only in terms of muscle mass may be of limited clinical value [13]. The new EWGSOP2 criteria emphasize that low muscle strength should be used as the primary variable for sarcopenia assessment since muscle strength has been shown to better predict adverse outcomes and survival than muscle mass, while muscle quantity or quality are needed for verification of sarcopenia and for research purposes [14].

Handgrip strength (HGS) detected by a handheld dynamometer is a potentially valuable and practical tool in the assessment of muscle function with low HGS indicating probable sarcopenia [14,15]. HGS is, however, a multifactorial index dependent on sex, age, height, body weight, mid-upper arm circumference, body composition, cancer stage, comorbidities, and smoking [16–18]. These confounding factors explain the controversy surrounding the use of HGS alone for predicting nutritional status, and sarcopenia. However, only a limited number of prospective studies have evaluated EWGSOP2 -defined sarcopenia in patients with head and neck squamous cell carcinoma (HNSCC) receiving curatively intended treatment [6,8,19–21]. Furthermore, because normative values for HGS and FFMI are sex-specific, mixed-sex analyses may be methodologically challenging particularly in studies with limited sample sizes.

According to the GLIM criteria, the assessment of low muscle mass within these criteria includes Bioelectrical Impedance Analysis (BIA), alongside other established techniques such as Dual-Energy X-ray Absorptiometry and Computed Tomography [22]. Crucially, HGS is not a component of the GLIM diagnostic algorithm and should not be used as a surrogate for objective muscle mass assessment.

Features of sarcopenia affect survival in HNC [1,7,10,12,23,24]. The meta-analyses of Wong et al. [25] showed that radiologically defined sarcopenic patients have 2-fold higher mortality compared with non-sarcopenic patients with HNC. Furthermore, associations between low muscle mass, surgical complications, treatment breaks, and dose-limiting toxicities during chemo(-radio)therapy have been demonstrated [1,10,11,23,26]. Patients with malnutrition have more unplanned hospital admissions, longer hospital stays, and shorter overall survival (OS) compared with patients without any nutrition-related conditions [1]. Therefore, early identification of these phenomena in newly diagnosed patients with HNC is important in order to reduce the risk of complications and to improve treatment outcomes. According to existing literature, no previous studies have used GLIM and sarcopenia frameworks together in patients with HNC.

The aim of this study was to explore the relationship between HGS, with or without low muscle mass (i.e., sarcopenia), patient and tumor characteristics, nutritional indices, and survival among male patients with HNSCC undergoing either major surgery, with or without postoperative therapy, or definitive (chemo)radiotherapy.

2. Materials and methods

2.1. Design

The patient series was based on a prospective randomized single-institution study on the efficacy of intensive nutrition counselling in patients with histologically proven HNSCC (NCT02159508) [27]. The cases were identified by the

multidisciplinary head and neck tumor board, and managed at the Departments of Otorhinolaryngology - Head and Neck Surgery and/or Oncology at the Helsinki University Hospital (Helsinki, Finland) between November 2009 and December 2011. This sub-study investigates the associations between baseline characteristics, nutritional indices, and outcomes in male patients with HNSCC. Females were excluded due to their small number ($n = 15$) and the observed differences in anthropometry between sexes. The institutional Research Ethics Committee at the Helsinki University Hospital approved the study design, and a research permission was granted (322/E9/05).

2.2. Inclusion and exclusion criteria

Included patients had histologically proven HNSCC and received curative-intent treatment with either surgery alone, a combination of surgery and postoperative (chemo)radiotherapy, or definitive (chemo)radiotherapy. Patients with impaired renal function (creatinine >1.5 times the upper limit of normal (ULN)), liver function (serum bilirubin >1.5 times ULN), heart failure, or palliative intent of treatment were excluded.

2.3. Data collection

Data on patient-, tumor-, and nutrition-related factors, follow-up, and death, including causes, were collected from hospital records. Plasma C-reactive protein (CRP), albumin, prealbumin, body composition analysis, body weight, and HGS were measured, and smoking and alcohol consumption were evaluated on the first outpatient visit. Nutritional measurements were done by a clinical nutritionist (HO). The criterion for hypoalbuminemia was set at plasma albumin <35 g/L and for hypoprealbuminemia at plasma prealbumin <160 mg/L. Glasgow prognostic score (GPS) was 2 when elevated CRP (>10.0 mg/L) and hypoalbuminemia (<35 g/L) were noted, and 1 if either was abnormal.

2.4. Body composition

Height was documented from hospital charts or by patient recall. Actual body weight was measured to the nearest 0.1 kg with a portable calibrated digital scale (Tanita, Illinois, USA). Patients were measured in indoor clothing without shoes and 1.0–1.5 kg, based on season, was deducted. Body weight six months before the HNSCC diagnosis was documented by recall and the subsequent weight loss then calculated. Critical weight loss was determined as $>10\%$ during the previous six months. Body mass index (BMI) was calculated and classified as underweight (<18.5 kg/m²), normal (18.5–24.9 kg/m²), or overweight (≥ 25.0 kg/m²).

Body composition was analyzed with BIA using a single frequency (50 kHz) two-terminal bioimpedance meter (Bodystat Ltd®, Isle of Man, UK), and the measurement was performed according to standard procedure. Fat-free mass index (FFMI) was used as a proxy for body muscle mass and low FFMI was defined as <17 kg/m² (i.e., <10 percentile from median age and sex-adjusted value) [22,28].

2.5. Nutritional status assessment and diagnosis of malnutrition

Nutritional status assessment was performed with the patient-generated subjective global assessment (PG-SGA) [29]. The allocation was performed according to changes in body weight, food intake, nutrition impact symptoms, ability to function, disease severity, metabolic demand, and body composition. Patients who had either SGA class B or C were allocated as malnourished and patients with SGA A as well-nourished. A research dietitian (HO)

performed the full nutritional assessment for all patients, which included completing both the patient and the professional components of the PG-SGA.

Diagnosis of malnutrition was defined according to Global Leadership Initiative on Malnutrition (GLIM) criteria (without screening), and at least one phenotypic and one etiologic criterion had to be present for diagnosis [22]. The phenotypic criterion was met if body weight loss was >5% within the past 6 months, or if BMI <20 kg/m² and age <70 years, or <22 kg/m² and age ≥70 years, or if FFMI <17 kg/m². The etiologic criterion was either reduced food intake defined as ≤50% of the usual intake (i.e. low food intake) or disease burden/inflammation defined as CRP >5 mg/L [22,30].

2.6. Handgrip strength and sarcopenia

Muscle function was assessed by measuring HGS for both arms with a Jamar handgrip dynamometer (Sammons Preston Rolyan, Chicago, USA), according to the American Society of Hand Therapists protocol [31]. The same equipment was used in all measurements. The patients performed the test while sitting comfortably with shoulder adducted and forearm neutrally rotated, elbow flexed to 90°, and forearm and wrist in neutral position. The hand dynamometer was adjusted for hand size and position three was used for males. Patients performed a maximal isometric contraction three times, with a 30-s rest between the contractions. The average of three measurements of the dominant hand was used for the analysis [32].

Sarcopenia was defined as a combination of low muscle strength, i.e., HGS <27 kg, and low muscle mass, i.e., FFMI <17 kg/m² [14,33]. Sarcopenic obesity was defined as sarcopenia combined with BMI ≥25.0 kg/m² [34].

2.7. Smoking and alcohol consumption

Information on the number of cigarettes smoked per day and daily alcohol consumption was obtained from the patients. Smoking was classified as never smokers (smoked <100 cigarettes in their lifetime and not currently smoking), former smokers (smoked ≥100 cigarettes in their lifetime and currently a non-smoker), and current smokers (smoked ≥100 cigarettes in their lifetime and currently a smoker). Cumulative smoking exposure in former and current smokers was determined in terms of pack-years by multiplying the number of years smoked with the average number of packs per day (i.e., 20 cigarettes/day = 1 pack) and classified as never smoker (0 pack-years), light smoker (0.1–20.0 pack-years), moderate smoker (20.1–40.0 pack-years), and heavy smoker (>40 pack-years) [35].

Alcohol consumption history was recorded as servings of alcohol (1 serving = 12 g ethanol = 1 drink) per week, and male patients were categorized as heavy drinkers in cases of 23 or more drinks per week, i.e., alcohol abuse or high-risk alcohol use according to Finnish recommendations [36].

2.8. Statistics

Due to the small number of patients, all statistical analyses were performed with non-parametric tests. Descriptive data are reported as proportions or medians with range or interquartile range (IQR). Between-group comparisons were analyzed with Chi-square, Fisher's exact test, the Mann-Whitney U test, or the Kruskal-Wallis test, as appropriate. Associations were analyzed with odds ratios (OR) with 95% confidence interval [95% CI] with logistic regression or Spearman's rho. Follow-up time was calculated with reverse Kaplan-Meier analyses. Kaplan-Meier

estimates for OS were calculated from the time of diagnosis to death from any cause or censored at last date of follow-up (18th March 2015), and for disease-free survival (DFS) to date of cancer recurrence or death from any cause or censored at last date of follow-up. Associations were studied using univariate and multivariable Cox regression models. Covariates selected for adjustment in the multivariable model were identified based on preliminary bivariate analysis, using a significance threshold of $p < 0.25$. Given the limited number of events and the risk of overfitting, we pre-specified parsimonious Cox models restricted to clinically essential covariates (HGS, sarcopenia, GLIM malnutrition, and CRP) as a sensitivity analysis complementary to the fully adjusted models. As an additional sensitivity analysis, HGS and sarcopenia were adjusted with disease stage. The assumption of proportionality of risks was assessed using the Schoenfeld test, confirming $p > 0.05$ for all variables. Statistical analyses were carried out using SPSS software (version 29.0; SPSS Inc, Chicago, IL). A two-tailed p -value less than 0.05 was considered statistically significant and no correction for multiple testing was performed.

3. Results

3.1. Patient demographics

Patient characteristics are shown in Table 1. The median age was 61 years (range 33–77) and 22% were ≥65 years. Most of the patients (70%) had Stage IV disease. A total of 35 patients (70%) had positive regional neck nodes, and one patient had distant metastases. The most prevalent tumor sites were oropharynx (30%), hypopharynx (20%), and larynx (20%).

3.2. Handgrip strength and nutritional indices

Median HGS was 39 kg (range 6–63) among all patients (Table 2). Patients with low HGS had a median HGS of 23 kg (range 6–26) and those with normal HGS had a median HGS of 42 kg (range 27–63; Table 2). Low HGS was observed in 8 (16%) patients. Patients with low HGS had a median body weight of 64 kg (range 46–81) and patients with normal HGS had a median body weight of 81 kg (52–118 kg, $p = 0.007$). Median height (176 cm in both groups, $p = 0.510$) or weight change (−5.5% vs. −1%, $p = 0.265$) did not vary according to HGS. The median HGS for underweight patients was 25 kg, 34 kg for normal weight, and 47 kg for overweight patients ($p < 0.001$).

A total of 23 (46%) patients had low FFMI, and 6 (12%) had sarcopenia, while none had sarcopenic obesity (Table 1). Malnutrition according to GLIM criteria was diagnosed in 17 (34%) patients and 15 (30%) patients were malnourished according to PG-SGA. Thirty-five (70%) patients had lost body weight during the previous 6 months, 2 (4%) were weight stable, and 13 (26%) had gained weight. Six (12%) patients had critical weight loss.

Malnutrition with GLIM, poor nutritional status according to PG-SGA, BMI <18.5 kg/m², GPS 2, elevated CRP, hypoprealbuminemia, and heavy smoking with >40 pack-years were more prevalent in patients with low HGS than in patients with normal HGS, whereas age, FFMI, critical weight loss, heavy drinking and low food intake were similar between the groups (Table 1).

Median HGS was statistically significantly lower in patients with malnutrition, poor nutritional status, sarcopenia, BMI <18.5 kg/m², critical weight loss, low FFMI, hypoprealbuminemia, and heavy smoking (Table 2). Associations with HGS were not observed in patients ≥65 years, with elevated CRP, GPS 2, stage IV disease, or heavy alcohol drinking.

Table 1
Characteristics of 50 male patients stratified according to handgrip strength.

		All	Normal HGS	Low HGS	p-value	OR (95% CI)
		N = 50 (%)	≥27 kg n = 42 (84%)	<27 kg n = 8 (16%)		
Age, years	<65	39 (78)	33 (79)	6 (75)	0.823	
	≥65	11 (22)	9 (21)	2 (25)		
Tumor site	Oral cavity	8 (16)	6 (14)	2 (25)	0.106	
	Oropharynx	15 (30)	12 (29)	3 (38)		
	Hypopharynx	10 (20)	8 (19)	2 (25)		
	Larynx	10 (20)	10 (24)	0 (0)		
	Nasopharynx	6 (12)	6 (14)	0 (0)		
	Unknown primary	1 (2)	0 (0)	1 (13)		
Stage	I-III	9 (18)	11 (26)	4 (50)	0.841	
	IV	35 (70)	31 (74)	4 (50)		
Treatment plan	Surgery + chemoradiotherapy	7 (14)	7 (17)	0 (0)	0.057	
	Surgery + radiotherapy	1 (2)	1 (2)	0 (0)		
	Chemoradiotherapy	35 (70)	30 (71)	5 (62)		
	Radiotherapy	5 (10)	2 (5)	3 (38)		
	Surgery	2 (4)	2 (5)	0 (0)		
Malnutrition	No malnutrition (GLIM)	33 (66)	31 (74)	2 (25)	0.008	1.00
	Malnutrition (GLIM)	17 (34)	11 (26)	6 (75)		8.4 (1.5–48.3)
Nutritional status	Well-nourished (PG-SGA A)	35 (70)	32 (76)	3 (38)	0.029	1.00
	Malnourished (PG-SGA B or C)	15 (30)	10 (24)	5 (62)		5.3 (1.1–26.4)
Muscle mass	Normal (FFMI ≥17 kg/m ²)	27 (54)	25 (60)	2 (25)	0.079	
	Low (FFMI <17 kg/m ²)	23 (46)	17 (40)	6 (75)		
Sarcopenia	No	44 (88)	42 (100)	2 (25)	<0.001	1.00
	Yes	6 (12)	0 (0)	6 (75)		22.0 (6–85)
Body mass index	<18.5 kg/m ² , underweight	3 (6)	1 (2)	2 (25)	0.030	1.00
	18.5–25 kg/m ² , normal weight	22 (44)	18 (43)	4 (50)		9.2 (1.3–66.6)
	>25 kg/m ² , overweight	25 (50)	23 (55)	2 (25)		0.6 (0.1–2.5)
Critical weight loss	No (≤10% per 6 month)	44 (88)	38 (90)	6 (75)	0.217	
	Yes (>10% per 6 month)	6 (12)	4 (10)	2 (25)		
Hypoprealbuminemia	No (≥160 mg/L)	41 (82)	37 (88)	4 (50)	0.010	1.00
	Yes (<160 mg/L)	9 (18)	5 (22)	4 (50)		7.4 (1.4–39.4)
Hypoalbuminemia	No (≥35 g/L)	40 (40)	35 (83)	5 (62)	0.177	
	Yes (<35 g/L)	10 (20)	7 (17)	3 (38)		
Inflammation	No (CRP ≤5 mg/L)	23 (46)	22 (52)	1 (13)	0.038	1.00
	Yes (CRP >5 mg/L)	27 (54)	20 (48)	7 (88)		7.7 (0.9–68.1)
Glasgow prognostic scale	GPS 0–1	43 (86)	38 (90)	5 (62)	0.037	1.00
	GPS 2	7 (14)	4 (10)	3 (38)		5.7 (1.0–33.3)
Smoking status	Never smoker	8 (16)	7 (17)	1 (13)	0.337	
	Former smoker	16 (32)	15 (36)	1 (13)		
	Current smoker	26 (52)	20 (48)	6 (75)		
Smoking	Non-heavy smoker (≤40 pack-years)	31 (16)	29 (69)	2 (25)	0.019	1.00
	Heavy smoker (>40 pack-years)	19 (36)	13 (31)	6 (75)		6.7 (1.2–37.7)
Alcohol drinking	Non-heavy drinker (≤23 drinks/week)	42 (84)	35 (83)	5 (62)	0.932	
	Heavy drinker (>23 drinks/week)	8 (16)	7 (17)	3 (38)		
Food intake <50% ^a	No	39 (78)	34 (81)	5 (62)	0.248	
	Yes	11 (22)	8 (19)	3 (38)		

CRP, C-reactive protein, FFMI fat-free mass index, GLIM Global Leadership Initiative on Malnutrition, GPS Glasgow prognostic scale (CRP>10 mg/L and/or Albumin <35 g/L), HGS handgrip strength, PG-SGA patient-generated global assessment.

^a food intake ≤50% of usual intake.

3.3. Smoking

Eight patients (16%) were never smokers, 16 (32%) were former smokers, and 26 (52%) were current smokers, of which 12 had quit more than 1 year and 4 less than a year before diagnosis. The median HGS was 51 kg (IQR 44–54) in non-smokers, 44 kg (range 34–54) in light smokers, 37 kg (range 35–43) in moderate smokers, and 33 kg (range 26–41) in heavy smokers (p = 0.024). Heavy smokers with >40 pack-years had lower HGS compared with non-heavy smokers (43 vs. 33 kg; OR 6.7 [95% CI: 1.2–38.0]; Tables 1 and 2).

Heavy smokers had lower FFMI than non-heavy smokers, median 16.0 (range 12.8–19.7) vs. 18.0 (range 15.5–24.4; OR 1.9, [95% CI: 1.2–2.8]; p = 0.003).

3.4. Survival

The median follow-up time was 77 months (95% CI: 71–82). In this study of 50 men with HNSCC, 24 men died during follow-up

with a corresponding 5-year OS rate of 58%. The cause of death was HNSCC in 17 patients, other cancer in 6, and malnutrition in 1. Of the 24 who died, 7 had low HGS. Males with low HGS had shorter OS compared with those with normal HGS (HR 3.7 [95% CI: 1.5–9.2]; p = 0.004), with corresponding 5-year OS rates of 13% vs. 67%, respectively (Fig. 1A).

During the follow-up time, 18 patients experienced a recurrence of HNSCC and 17 died (1 curatively treated local relapse in jaw), with a 5-year DFS rate of 52%. Of those 25 patients with either recurrence or death, 7 had low HGS. Men with low HGS had worse DFS than those with normal HGS (HR 3.2; [95% CI: 1.3–7.7]; p = 0.010) with 5-year DFS rates of 13% vs. 60%, respectively (Fig. 1B).

Men with low FFMI did not have statistically significantly shorter OS compared with those with normal FFMI (HR 2.3; [95% CI: 0.99–5.2], p = 0.053), with corresponding 5-year OS rates of 43% vs. 70%, respectively (Fig. 1C), or shorter DFS (HR 1.8; [95% CI: 0.8–3.9], p = 0.144), with 5-year DFS rates of 39% vs. 63%, respectively (Fig. 1D).

Table 2
Median (range) handgrip strength in 50 males according to nutritional indices.

		HGS in kg median (range)	95% CI	p-value
HGS	Normal	42 (27–63)	38–48	<0.001
	Low	23 (6–26)	14–25	
Age	<65 years	39 (14–63)	36–47	0.907
	≥65 years	39 (6–59)	29–49	
Stage	I–III	37 (23–59)	27–53	0.478
	IV	41 (6–63)	37–47	
Malnutrition	No malnutrition (GLIM)	41 (23–63)	37–49	0.027
	Malnutrition (GLIM)	36 (6–54)	26–45	
Nutritional status	Well-nourished (SGA A)	42 (22–63)	39–51	0.005
	Malnourished (SGA B or C)	31 (6–50)	26–41	
Muscle mass	Normal (FFMI ≥17 kg/m ²)	43 (23–63)	39–53	0.016
	Low (FFMI <17 kg/m ²)	34 (6–54)	29–41	
Sarcopenia	No	41 (23–63)	38–47	<0.001
	Yes	22 (6–26)	14–25	
Body mass index	<18.5 kg/m ² , underweight	25 (14–36)	14–36	<0.001
	18.5–25 kg/m ² , normal weight	34 (6–54)	31–39	
	>25 kg/m ² , overweight	47 (23–63)	41–54	
Critical weight loss	No (≤10% per 6 months)	41 (6–63)	37–48	0.036
	Yes (>10% per 6 months)	31 (14–45)	30–45	
Hypoprealbuminemia	No (≥160 mg/L)	41 (14–45)	38–49	0.004
	Yes (<160 mg/L)	30 (6–63)	22–41	
Hypoalbuminemia	No (≥35 g/L)	40 (22–63)	37–48	0.119
	Yes (<35 g/L)	35 (6–54)	21–47	
Inflammation	No (CRP ≤5 mg/L)	40 (25–59)	37–49	0.340
	Yes (CRP >5 mg/L)	38 (6–63)	35–47	
Glasgow prognostic scale	GPS 0–1	40 (22–63)	37–47	0.061
	GPS 2	30 (6–54)	14–54	
Smoking	Non-heavy smoker (≤40 pack-years)	43 (21–63)	38–53	0.004
	Heavy smoker (>40 pack-years)	33 (6–49)	26–41	
Alcohol	Non-heavy drinker (≤23 drinks/week)	41 (6–63)	37–48	0.181
	Heavy drinker (>23 drinks/week)	37 (20–53)	25–43	
Food intake <50%	No	41 (21–63)	37–48	0.053
	Yes	36 (6–50)	25–41	

CRP, C-reactive protein, GLIM global leadership initiative on malnutrition, GPS Glasgow prognostic scale, HGS, handgrip strength, PG-SGA patient-generated global assessment.

p-values between normal and low HGS were calculated by the Mann–Whitney's U-test.

Males with both low HGS and low FFMI, i.e., sarcopenia, had shorter OS compared with those without sarcopenia (HR 5.5; [95% CI: 2.1–14.5], $p = 0.001$), with 5-year OS rates of 0% vs. 66%, respectively (Fig. 1E). Men with sarcopenia had poorer 5-year DFS compared with those without (HR 3.98; [95% CI: 1.5–10], $p = 0.004$) with 5-year DFS rates of 0% vs. 59%, respectively (Fig. 1F).

After adjusting OS with BMI, CRP, GLIM-based malnutrition, FFMI, and smoking status, the association with either HGS or sarcopenia was no longer statistically significant (Tables S1 and S2). Similarly, after adjusting DFS for BMI, CRP, food intake, GLIM-based malnutrition, FFMI, and smoking status, the association with HGS and sarcopenia was no longer statistically significant (Tables S3 and S4). In parsimonious models including HGS, GLIM malnutrition, and CRP, low HGS and sarcopenia remained associated with mortality (e.g., HR 3.55, 95% CI 1.32–9.56; and HR 5.41, 95% CI 1.53–19.11), whereas the association for FFMI attenuated and was not statistically significant (HR 1.93, 95% CI 0.66–5.62). As an additional sensitivity analysis, after adjustment for disease stage, low HGS and sarcopenia remained significantly associated with higher mortality risk (HR 4.15, 95% CI 1.57–11.0; and HR 5.51, 95% CI 2.06–14.69).

3.5. Correlations of HGS, FFMI, sarcopenia, and malnutrition with survival

Figure 2 shows the association between HGS and FFMI (Spearman rho 0.589, $p < 0.001$). Patients with low HGS and low FFMI, i.e. sarcopenia, had poor outcomes as all patients died within

55 months, whereas 67% (8 out of 12, 4 died of HNSCC) of those in the best HGS and FFMI tertiles (FFMI ≥18.6 kg/m² and HGS 47.3 kg) survived. Malnutrition with GLIM criteria appears to identify patients with low FFMI better (OR 3.0, [95% CI: 1.5–5.7], $p = 0.001$) than patients with low HGS (OR 1.07, [95% CI: 1.01–1.13]; $p = 0.021$).

4. Discussion

In this cohort of men with HNSCC receiving curatively intended treatment, low HGS was present in 16% and EWGSOP2-defined sarcopenia in 12%. Low HGS associated with poor nutritional status, systemic inflammation, and heavy smoking, but not heavy alcohol use. Patients with low HGS or sarcopenia had worse survival, whereas low muscle mass alone showed only a non-significant trend, and statistical significance was lost after adjustment.

The prevalence of low HGS in this study was lower than the 22%–45% reported by Meerkerk et al., [6,8] where patients were typically 10–20 years older and had less stage IV disease (43%–44% vs. 70% in our study). The median HGS aligns with previous reports for male patients with HNC (36–38 kg), while studies including females report substantially lower values (22–29 kg), reflecting differences also in sex, ethnicity, and disease stage [3,37–41]. Patients without low HGS, sarcopenia, or malnutrition had median HGS values of 41–42 kg, comparable to healthy Finnish males [42].

Few studies have applied EWGSOP2 criteria, combining low muscle strength and low muscle quantity, in HNC populations [6–8,14,21]. Reported sarcopenia prevalence ranges from 5% to 8%

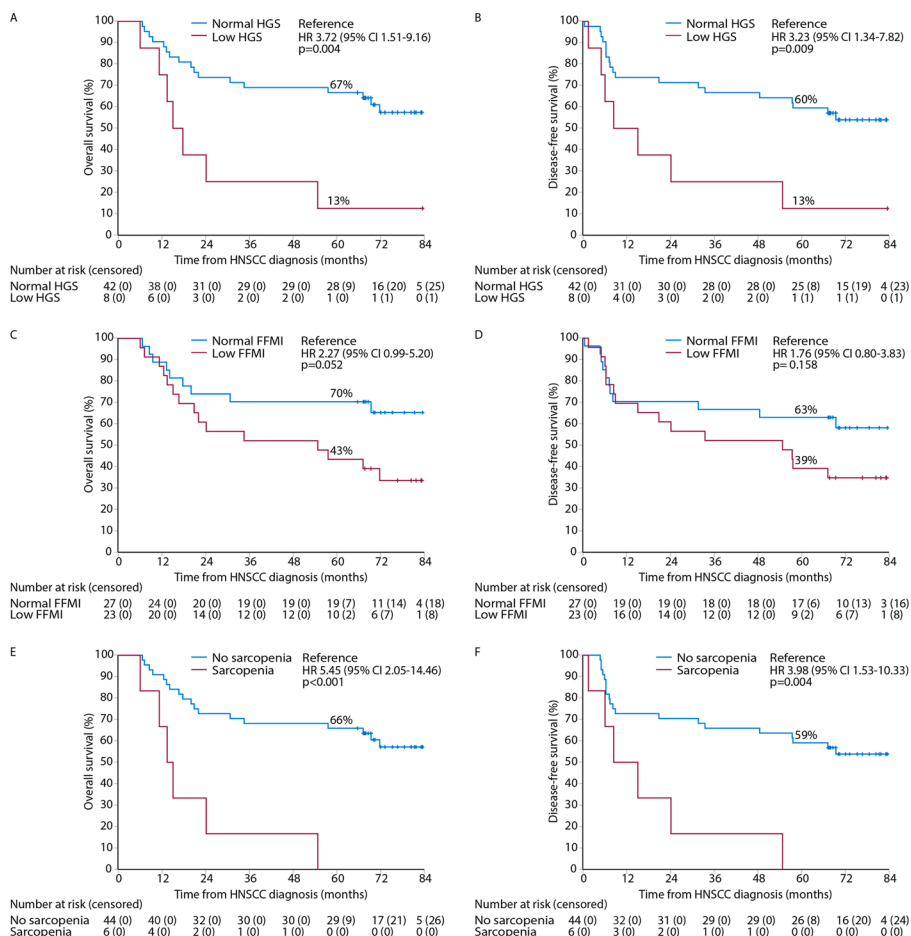


Fig. 1. Overall survival (OS; panels A, C, E) and disease-free survival (DFS; panels B, D, F) according to handgrip strength (A and B), muscle mass (C and D), and sarcopenia (E and F) in 50 males with HNC. Number of deaths: 7/8 in the low HGS group vs. 17/42 in the normal HGS group, 15/23 in the low muscle mass group vs. 9/27 in the normal muscle mass group, and 6/6 in the sarcopenia group vs. 18/44 in the non-sarcopenia group. Numbers at risk (numbers censored) are presented below the panels.

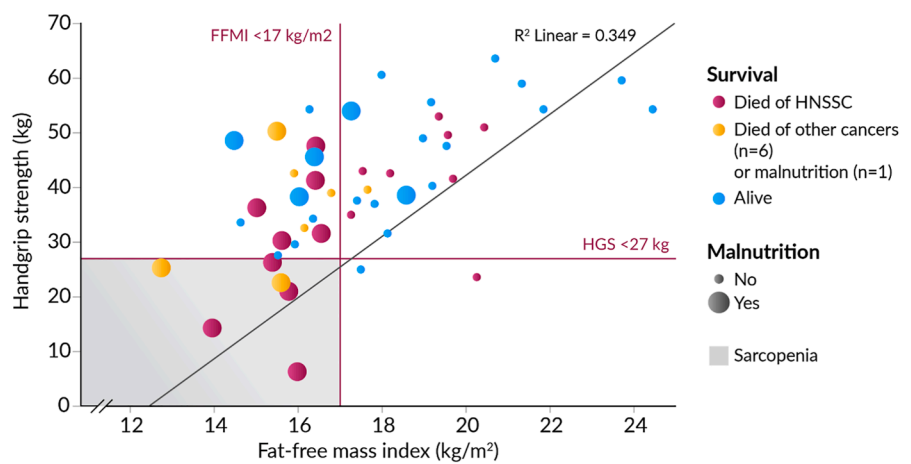


Fig. 2. Scatter plot of handgrip strength and fat-free mass index with cut-offs, and sarcopenia stratified according to malnutrition. Survival status with colors, malnutrition according to GLIM with circle size and sarcopenia according to EWGSOP2 in grey in 50 males with HNC.

in younger cohorts and 14%–15% in mixed histology groups, consistent with our finding, while rates rise to 33%–48% in older patients [6–8,21]. In contrast, low muscle mass alone shows much higher variability (6%–73%) across studies due to differences in patient populations and methods. In our cohort, low FFMI was observed nearly four times more than EWGSOP2-defined sarcopenia, mirroring findings by Meerkerk et al. (61% vs. 14%) [6].

Malnutrition and poor nutritional status were associated with low HGS in this study, consistent with prior evidence, while critical weight loss was not significantly linked with low HGS, although men with such loss had HGS values about 10 kg lower than men without critical weight loss [41,43]. HGS is not a consistent marker of nutritional status, as its association with malnutrition was weaker than that of FFMI, aligning with previous findings [17]. One possible explanation is that HGS may capture early nutritional changes before alterations in body composition occur [44].

FFMI showed only a weak association with HGS in our cohort, which contrasts with findings from the previous study conducted in younger population [39]. This discrepancy may be partly explained by myosteatosis, fat infiltration into muscle, which impairs muscle function and is more prevalent in advanced disease stages [38]. In addition, heavy smoking (>40 pack-years) was associated with lower HGS and FFMI, aligning with earlier research [9,45,46]. Smoking-related muscle loss is thought to result from impaired protein synthesis, alterations in muscle-regulatory gene expression, and mitochondrial dysfunction, all of which contribute to progressive muscle atrophy [44,47,48]. Notably, low HGS showed strong association with markers of systemic inflammation (CRP, GPS), supporting existing evidence that cancer-related inflammation drives muscle wasting and functional decline [49,50].

Low HGS and EWGSOP2-defined sarcopenia were negative prognostic factors for survival, while low muscle mass alone showed only a non-significant trend. These findings align with Chergi et al. [7], who reported shorter survival in sarcopenic patients with HNC. Prior studies and meta-analyses on low muscle mass report 5-year OS rates of 20%–70% and HRs of 1.9–3.9, comparable to the current study [4,5,12,51,52]. This suggests that EWGSOP2-defined sarcopenia and low HGS identify patients at highest risk [25]. Although we did not find a direct link between HGS and cancer stage, likely due to excluding palliative cases.

4.1. Limitations and strength of the study

This study has several limitations. This single-centre study included a relatively small, male-only cohort ($n = 50$) and because all patients received curative-intent treatment and most had stage IV disease, the results mainly apply to relatively fit, treatment-eligible men with HNSCC, and may not extrapolate to women, very old patients, or palliative populations. Restricting analysis to men was necessary due to sex-specific cut-offs for HGS and muscle mass, and findings thus apply only to males. The small sample size reduces statistical power and may overestimate hazard ratios; results should be considered exploratory and hypothesis-generating. Selection bias is possible, as patients receiving palliative care, i.e., more extensive disease, were excluded, likely creating a healthier subgroup and potentially inflating HGS's prognostic value (spectrum effect). No correction for multiple testing was applied, which may increase the risk of Type I error; therefore, findings should be interpreted with caution. Our survival analysis did not incorporate time-dependent covariates or competing-risk models, which may have provided more accurate estimates for cause-specific mortality and accounted for changes in clinical variables over time. After adjustment for BMI, CRP, malnutrition, FFMI, and smoking, the associations of HGS and

sarcopenia with survival were no longer statistically significant. Given the substantial interrelationships among these variables, this attenuation likely reflects shared explanatory variance and suggests that unadjusted associations may overestimate independent effects. Furthermore, the very low number of events in the sarcopenia group produced wide confidence intervals and statistically unstable HR estimates that warrant cautious interpretation. To address potential overfitting arising from the limited number of deaths, we conducted prespecified parsimonious Cox models restricted to key clinically relevant covariates (HGS, sarcopenia, GLIM malnutrition, and CRP) and (HGS, sarcopenia and disease stage). In these reduced models, low HGS and sarcopenia remained associated with mortality, whereas the association for FFMI attenuated and was not statistically significant. Taken together, these sensitivity analyses support the robustness of the main findings while highlighting the need for larger, adequately powered studies to confirm these associations. HGS is effort-dependent and influenced by conditions such as arthritis or nerve injury, which we could not fully control. Historical weight loss relied on self-report, introducing recall bias, and energy intake data were unavailable, though low food intake was not associated with survival in this cohort. Finally, lack of muscle quality data (e.g., myosteatosis) limits interpretation.

A key strength of this study is its use of data from a prospective randomized trial with standardized nutritional counselling and treatment. Nutritional status was assessed using multiple validated methods, providing a comprehensive evaluation in a relatively homogeneous male cohort. We applied the updated EWGSOP2 criteria and used the average of three HGS measurements, which improves identification of probable sarcopenia compared with using the highest value [14,32]. All assessments were performed by a single dietitian (HO), ensuring consistency.

Future studies should include larger, more diverse cohorts including both sexes, different ethnicities, and all disease stages to validate these findings and improve generalizability. Incorporating CT-derived muscle quality metrics (e.g., muscle attenuation) alongside SMM assessments could offer a more nuanced view of body composition and for validation of cut-offs.

5. Conclusion

In this small exploratory cohort low HGS and EWGSOP2-defined sarcopenia appear to be associated with poorer survival, in contrast to low FFMI. Malnutrition, poor nutritional status, inflammation, low BMI, and heavy smoking associates with HGS. While HGS offers a practical, low-cost screening option, its application must be paired with careful clinical judgment and validation in larger, more diverse patient populations before it can be considered a definitive, standalone "ready for prime time" assessment tool across the full spectrum of HNSCC disease severity. HGS <27 kg may identify men at higher risk of adverse outcomes in patients with HNSCC; however, larger and gender-inclusive cohorts are required for validation.

Patient consent statement

Informed consent was obtained from all participants.

Data availability statement

All data generated or analyzed during this study are included in this article and in the supplementary material mentioned above. Further enquiries can be directed to the corresponding author.

Ethics approval statement

The institutional Research Ethics Committee at the Helsinki University Hospital approved the study design, and research permission was granted.

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Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work the author(s) used Web of Science Research Assistant to search references and AI for modifying sentences. After using these tools/services, the authors reviewed and edited the content as needed and took full responsibility for the content of the published article.

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Conflict of interest disclosure

None of the authors report any conflict of interest.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.clnesp.2026.103312>.

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