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Original Research

Temporal muscle thickness is associated with clinical frailty in patients with severe acute brain injury

Jonathan Tam [a,b,*], Annabelle Tosh [c], Patrick J Coppler [b], Laura Faiver [a], Nicholas Case [b], Clifton Callaway [b], Jonathan Elmer [a,b,d]

[a] Department of Critical Care Medicine, University of Pittsburgh School of Medicine, USA
[b] Department of Emergency Medicine, University of Pittsburgh School of Medicine, USA
[c] Biomedical Master's Program, University of Pittsburgh School of Medicine, USA
[d] Department of Neurology, University of Pittsburgh School of Medicine, USA

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ABSTRACT

Background/Objective: Frailty predicts adverse outcomes in critically ill patients. Characterizing and measuring frailty in the intensive care unit is challenging since most frailty scales are created for the outpatient population. Temporalis muscle thickness (TMT), measured on head imaging, decreases in patients with sarcopenia and may offer an objective index of frailty among patients with SABI. The objective of this study was to test the relationship between TMT and the Clinical Frailty Scale (CFS) in this population.

Methods: In this prospective, single-center, observational cohort study, we enrolled subjects with SABI who had routine head computed tomography within 48 h of hospital admission. We calculated Spearman and Pearson correlation coefficients between CFS, TMT, and age. We also tested the relationship between CFS and TMT using multivariable ordinal regression. Finally, we compared the values of TMT and CFS in this cohort with those observed in a post-cardiac arrest cohort.

Results: In 51 enrolled patients, TMT was negatively correlated with CFS ($\rho = -0.32$, $p = 0.02$) and age ($r = -0.45$, $p = 0.001$), while CFS was positively correlated with age ($\rho = 0.31$, $p = 0.03$). The relationship between TMT and CFS remained significant when adjusted for albumin level and body mass index (OR 0.63, 95%CI 0.40–0.98). Median TMT and CFS values did not differ between the SABI and post-arrest cohorts, though the shapes of their distributions did.

Conclusions: TMT demonstrated a moderate correlation with pre-morbid CFS in patients with SABI, suggesting it might aid in characterizing frailty in this population.

1. Introduction

Patients who sustain severe acute brain injury (SABI) often require emergent, resource-intensive treatments [1–4]. While potentially life-saving, these treatments should be targeted to patients who are likely to derive benefit. Therefore, early risk stratification is important for patient-centered decision-making. To inform these treatment decisions, clinicians and surrogates consider many factors, including patients' health and functional status, which are strongly influenced by baseline frailty [5–9].

Frailty, a syndrome of physiologic decline that increases vulnerability to acute health stressors, [10–12] is a known predictor of adverse outcomes in many diseases, including SABI [13–16]. How best to characterize frailty in the context of critical illness remains uncertain due to the multiple contributing and interacting factors that influence the syndrome, such as sarcopenia, age, comorbidity, cognition, and disability. Existing measures, which were created and validated for community-dwelling populations, are difficult to implement in a critical care setting [10,17] because they rely heavily on patient participation, functional assessments, or accumulated outpatient records [10,18,19]. Since patients with SABI are often unable to self-report or participate in assessments, traditional measures are difficult to apply. In the absence of objective data, clinicians and families may rely on subjective perceptions of frailty to inform decision-making [20–22].

Sarcopenia, a domain of physical frailty, is difficult to quantify in patients unable to perform strength and functional assessments [23–25].

* Corresponding author at: 3550 Terrace Street, Alan Magee Scaife Hall, Suite 600, Pittsburgh, PA 15213, USA.
E-mail address: tamjw@upmc.edu (J. Tam).

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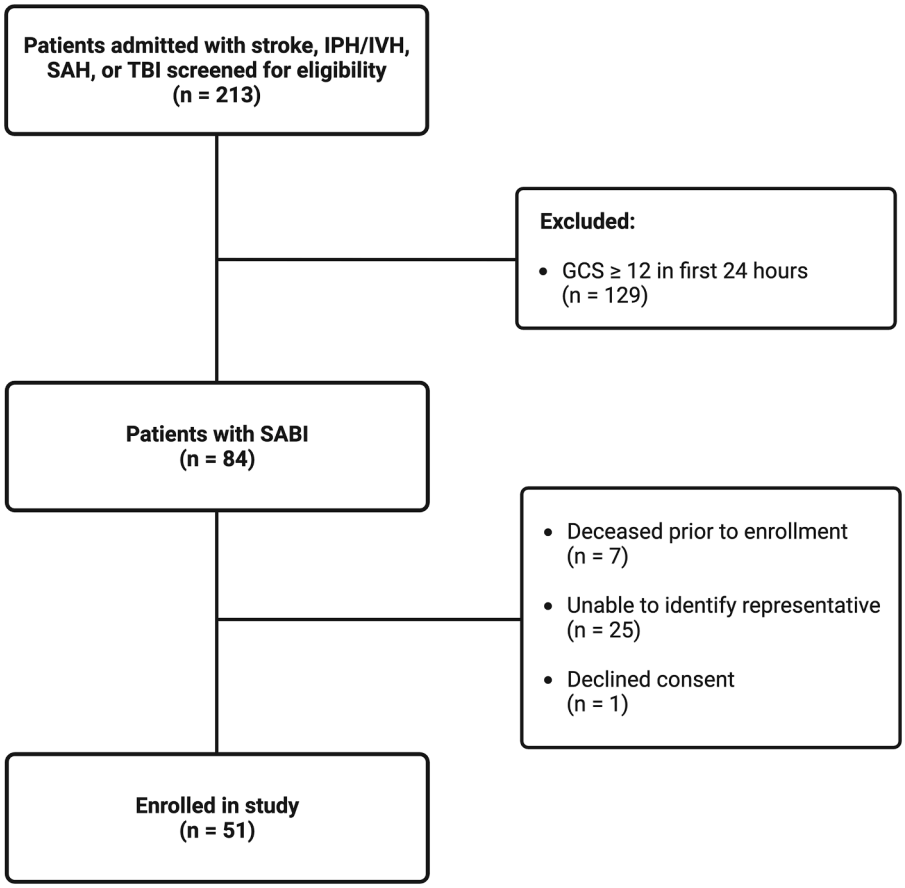

Fig. 1. Study enrollment diagram.
GCS: Glasgow coma scale. IPH/IVH: intraparenchymal hemorrhage. SABI: severe acute brain injury. SAH: subarachnoid hemorrhage. TBI: traumatic brain injury.

However, certain quantifiable aspects of sarcopenia (e.g., temporal wasting) are associated with frailty [26,27]. Previous work in a post-cardiac arrest population showed temporalis muscle thickness (TMT), measured on routinely obtained computed tomography (CT), may be a radiographic biomarker that allows for objective characterization of baseline frailty [26]. It is unknown if these findings generalize to other forms of SABI, which occurs in distinct patient populations with different cohort characteristics. Determining the validity of these findings in other types of SABI would be important for generalizability and eventual implementation across other populations prone to routine cross-sectional head imaging. Improving objective frailty phenotyping may serve not only prognostic purposes, but it may also support system-level monitoring of recovery trajectories after SABI such as length of stay, disposition, and rehabilitation priorities, positioning TMT as a potential "performance monitoring" biomarker [28].

The primary objective of this study was to test the relationship between TMT and clinical frailty measured by the Clinical Frailty Scale (CFS)[10] in patients hospitalized after SABI. We hypothesized that TMT would be correlated with premorbid CFS in patients with SABI. A secondary objective was to compare frailty characteristics of this cohort with previous data from post-arrest patients (a specific subgroup of SABI).

2. Methods

2.1. Patients and setting

The University of Pittsburgh Human Research Protection Office approved this prospective, observational cohort study (STUDY24010027). We prospectively screened and enrolled a convenience sample of subjects at an urban academic hospital with trauma and comprehensive stroke capabilities from October 2024 to April 2025 to achieve our target sample size. We included patients aged ≥18 years admitted with SABI who had a non-contrasted head CT obtained within 48 h of hospital admission as part of routine clinical care. CT scans were acquired using a GE Lightspeed VCT 64-channel scanner (120 kVp, 225 mA, 5 mm slice thickness). We included SABI patients with ischemic stroke, intraparenchymal/intraventricular hemorrhage (IPH/IVH), non-traumatic subarachnoid hemorrhage (SAH), or traumatic brain injury (TBI) admitted to the hospital with a best Glasgow Coma Scale (GCS) ≤ 12 in the first 24 h after injury [29]. We obtained written informed consent from the participant or legal representative identified by the clinical team to obtain CFS. We excluded SABI patients with hypoxic-ischemic brain injury (HIBI) after cardiac arrest, as this population was the focus of a prior study. Population details of the HIBI cohort were previously published [26]. In brief, the cohort includes 50 consecutively screened adult subjects enrolled in 2024 following cardiac arrest, all of whom had routine head CT within 48 h of arrest.

2.2. Measures

To describe our cohort, we extracted participant characteristics including age, sex, race, ethnicity, injury etiology, GCS, 30-day survival, albumin level, height, weight, and 30-day modified Rankin Scale (mRS) from the electronic health record. Study investigators approached patients or their representatives to administer the CFS regarding the patients' pre-injury health state using a standard structured classification tree [30]. We recorded all data using REDCap [31,32].

A study investigator, blinded to clinical characteristics, CFS, and outcomes, measured TMT on CT scan bilaterally 5 mm above the

Table 1
Cohort characteristics.

	Severe acute brain injury	Post-cardiac arrest *
N	51 (50.5%)	50 (49.5%)
Age (median, [IQR])	63 [49 - 72]	58 [50 - 67]
Sex (n, %)		
Male	23 (45.1%)	31 (62.0%)
Female	28 (54.9%)	19 (38.0%)
Race (n, %)		
American Indian or Alaskan Native	0 (0.0%)	1 (2.0%)
Asian	1 (2.0%)	0 (0.0%)
Black/African American	4 (7.8%)	10 (20.0%)
Native Hawaiian or Pacific Islander	5 (9.8%)	0 (0.0%)
White	41 (80.4%)	34 (68.0%)
Unknown	0 (0.0%)	5 (10.0%)
Ethnicity (n, %)		
Non-Hispanic or Latino	51 (100.0%)	41 (82.0%)
Unknown	0 (0.0%)	9 (18.0%)
Etiology (n, %)		
Ischemic stroke	10 (19.6%)	0 (0.0%)
IPH/IVH	22 (43.1%)	0 (0.0%)
Subarachnoid hemorrhage	11 (21.6%)	0 (0.0%)
Traumatic brain injury	8 (15.7%)	0 (0.0%)
Hypoxic-ischemic brain injury	0 (0.0%)	50 (100.0%)
GCS (median, [IQR])	10 [8 - 11]	—
PCAC (median, [IQR])	—	3 [2 - 4]
CFS (median, [IQR])	4 [2 - 4]	4 [2 - 6]
TMT (millimeters; median, [IQR])	6.1 [5.2 - 7.1]	6.6 [5.0 - 8.9]
Albumin (g/dL; median, [IQR])	4.0 [3.6 - 4.2]	—
BMI (kg/m^2; median, [IQR])	26.2 [23.9 - 33.0]	—
30-day survival (n, %)	41 (80%)	—
30-day mRS (median, [IQR])	4 [4 - 6]	—

BMI: Body mass index.
CFS: Clinical frailty scale.
GCS: Glasgow coma scale.
IPH/IVH: intraparenchymal hemorrhage / intraventricular hemorrhage.
mRS: Modified Rankin scale.
PCAC: Pittsburgh cardiac arrest categories.
TMT: Temporalis muscle thickness.
* This cohort was previously published and fully described in a previous study.[26].

superior orbital rim adjacent to the Sylvian fissure [26,33,34]. Interrater reliability for this method was excellent in a prior study [26]. We used the average TMT of both sides as the TMT measurement for each participant.

2.3. Statistical analysis

We targeted a sample size of 50 subjects to yield 80% power for a two-tailed test of a Spearman coefficient of 0.5 based on prior data [26,35]. We summarized baseline characteristics using descriptive statistics. We calculated Spearman's or Pearson's coefficients to evaluate the strength and direction of the correlations between CFS, age, and TMT when appropriate. To further test the relationship between TMT and CFS, we used an ordinal regression model adjusting for age, body mass index (BMI), and serum albumin level as potential confounders. We then compared median CFS scores and TMT measurements in this cohort with SABI to previously collected data from a cohort of 50 post-cardiac arrest patients[26] using Wilcoxon rank sum tests and compared distribution of measurements using a Kolmogorov-Smirnov test.

As an exploratory analysis, we quantified the relationship between TMT and 30-day outcomes (survival and mRS) in patients with SABI using logistic regression for binary outcomes and ordinal regression for ordinal outcomes. We performed both univariate and multivariate analyses, adjusting for age and GCS given their established association with outcome [36,37].

We were concerned that enrolling a convenience sample and relying on identification of a representative for inclusion could result in selection bias. To address this, we tabulated the screening TMT measurements of patients who did not survive to enrollment. We used STATA Version 18 (College Station, Texas, USA) for statistical analyses and R (R Foundation for Statistical Computing, Vienna, Austria) to generate figures [38].

3. Results

We screened 213 patients with acute neurological injury, of whom 84 met criteria for SABI (Fig. 1). Of these, 33 otherwise eligible patients were not enrolled: one patient representative declined consent, 32 either died before approach or we could not identify a surrogate representative. We included 51 subjects with SABI for the primary analysis.

Median age was 63 [IQR 49, 72] years, 28 (55%) were female, and the most common SABI subgroup was IPH/IVH (22; 43%) (Table 1). Median best GCS in the first 24 h after injury was 10 [IQR 8, 11]. TMT was negatively correlated with CFS (Spearman's $\rho = -0.32$, $p = 0.02$; Fig. 2**a & Figure S1**). TMT was also negatively correlated with age (Pearson's $r = -0.45$, $p = 0.001$; Fig. 2**c**), while CFS was positively correlated with age (Spearman's $\rho = 0.31$, $p = 0.03$; Fig. 2**b**). In our adjusted ordinal regression model, TMT was still independently associated with CFS (OR 0.63, 95%CI 0.40–0.98).

When comparing with the prior post-arrest cohort, we found no differences in median CFS or TMT among patients with SABI. The median CFS was 4 [IQR 2, 6] in the post-arrest cohort and 4 [IQR 2, 4] in the SABI cohort ($p = 0.07$). Similarly, median TMT was 6.6 mm [5.0, 8.9] in the post-arrest cohort and was 6.1 mm [IQR 5.2, 7.1] in the SABI cohort ($p = 0.27$) [26]. Although the medians were not different, the distributions of CFS differed between the SABI and post-arrest cohorts ($p = 0.002$). Similarly, TMT distributions differed between groups ($p = 0.03$). Post-arrest patients had a bimodal distribution of frailty and a broader distribution of TMT (Fig. 3**& Figure S2**).

In our exploratory analysis, TMT was not associated with 30-day survival (univariate OR 1.46; 95%CI 0.85–2.50, multivariate OR 1.30; 95%CI 0.69–2.41). Similarly, we found no association between TMT and mRS at 30 days (univariate OR 0.76; 95%CI 0.57–1.08, multivariate OR 0.98; 95%CI 0.68–1.43). In the 7 patients who were screened eligible but died before enrollment, the median TMT was 4 mm [IQR 3.6–5.5].

4. Discussion

In this study, TMT demonstrated a moderate negative correlation with premorbid frailty. After adjusting for BMI and albumin level in ordinal regression, we found the relationship remained significant. TMT has been identified as a prognostic indicator across a range of neurologic conditions including neuro-oncology, Parkinson's disease, SAH, and ischemic stroke [39–43]. These findings suggest TMT may serve as a practical, radiologic biomarker reflecting a component of frailty in patients with SABI. Complex treatment decisions are shaped not only by the acute severity of illness, but also by pre-illness health characteristics, which can heavily influence the anticipated recovery course. Various tools have been developed to describe frailty across different clinical settings, without clear consensus as to which best captures the construct [44]. Each has distinct strengths and limitations, such as dependence on clinical history or patient participation, and none have been rigorously evaluated in populations with critical illness. This study lays groundwork for research to improve the ability to characterize phenotypes of frailty, support prognostication, and guide more individualized treatment decisions in this heterogenous cohort. Future work will focus on assimilating biomarkers—including radiographic measurements of sarcopenia—to quantify frailty across multiple dimensions using objective clinical data in critically ill patients, providing granular data to supplement historical data. Additionally, to establish TMT as a frailty biomarker in SABI, future directions involve defining clinically meaningful reference ranges, establishing reliability across raters and

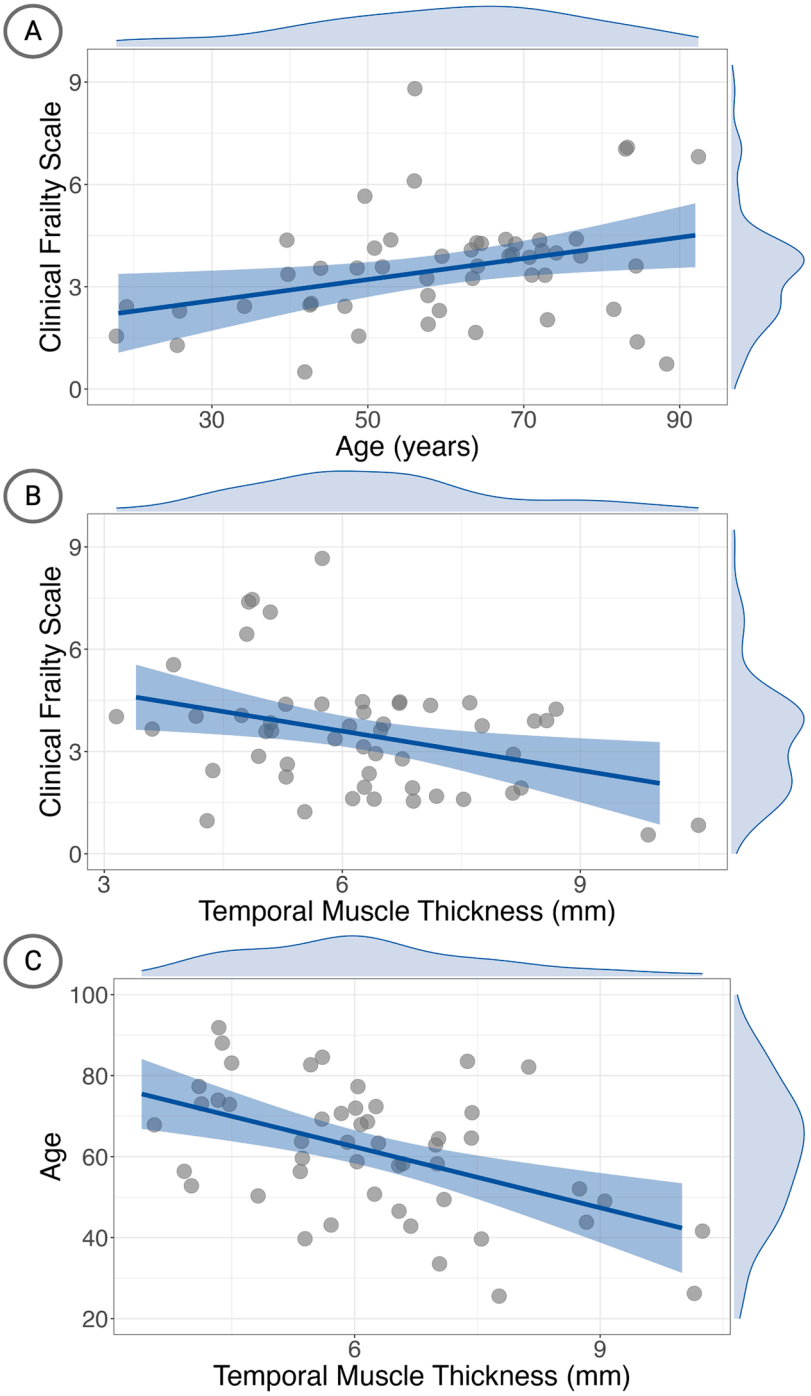

Fig. 2. Scatter plots with marginal density plots and best-fit lines describing correlations between A) CFS and TMT ($\rho = -0.32$, $p = 0.02$), B) CFS and age ($\rho = 0.31$, $p = 0.03$), and C) Age and TMT ($r = -0.45$, $p = 0.001$).

scanners, and validating whether TMT adds prognostic and system-level value beyond age and existing frailty tools.

The correlation between TMT and pre-morbid clinical frailty measured by CFS in patients admitted to the ICU after SABI, is notably lower than the correlation between TMT and CFS in a previous study of post-cardiac arrest patients ($\rho = -0.52$) [26]. We also found the median frailty in SABI patients did not differ from post-arrest patients with HIBI. Median CFS is 4 in both groups, which represents "living with very mild frailty," but CFS was skewed towards milder frailty in SABI patients. CFS in the post-cardiac arrest cohort frailty had a bimodal distribution, likely comprised of the relatively robust individuals who arrest due to sudden disease such as acute coronary events, and very frail individuals who arrest because of accumulating chronic disease. The distinction in the frailty distribution between cohorts might also result from our sampling strategy. Most subjects in this convenience sample were enrolled within 5 days of hospital admission; however, it is possible that the more frail had a higher likelihood of dying early in the hospital course due to illness severity and early WLST, which could explain the skewed distribution of frailty in patients with SABI. Median TMT in the was much lower in the screened patients who died before approach (4.1 mm). This suggests these patients may be more sarcopenic and potentially frailer than our study population (median TMT 6.1 mm), resulting in earlier death prior to enrollment.

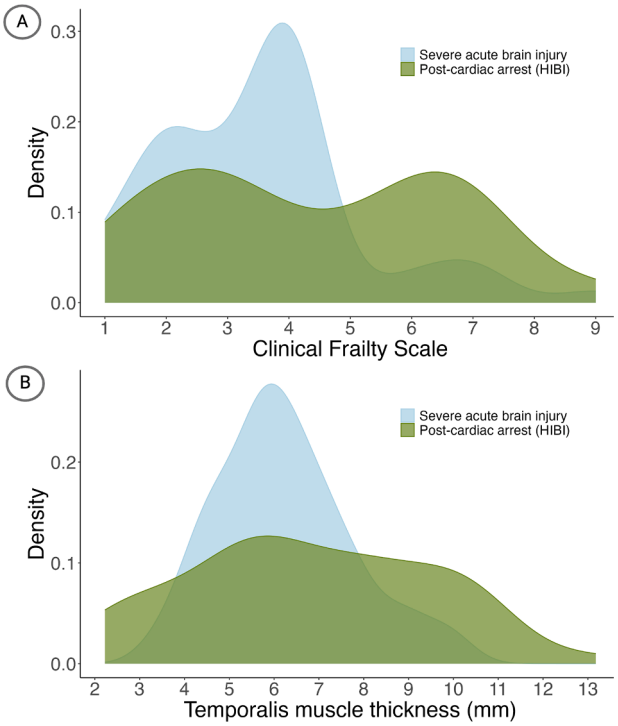

Fig. 3. Density plots visualizing distinct distributions of A) CFS in subjects hospitalized after SABI (n = 51) compared to those hospitalized after HIBI due to cardiac arrest (n = 50). Kolmogorov-Smirnov test p-value = 0.002. B) Illustrates distinct distributions of TMT in the two cohorts. Kolmogorov-Smirnov test p-value = 0.03.

4.1. Limitations

Our study has several limitations. First, we enrolled a convenience sample, which introduces the potential for selection bias. Additionally, we use a rather strict definition of SABI for study eligibility based on modified criteria from Kiker et al [29]. The relationships between TMT, frailty, and outcomes in patients with less severe acute brain injury is unknown. We enrolled subjects whose best GCS in the first 24 h after brain injury was $\leq$ 12. This sample represents patients who would generally be unable to participate in interviews to determine pre-morbid frailty status from the moment they enter the hospital up to hour 24 after injury—a timeframe in which many major medical decisions are made. We also enrolled a heterogenous SABI population, and it is possible each etiology of SABI has a distinct frailty distribution. However, our sample size limits the ability to detect these differences. We use the CFS because it is the most used frailty measure in critical care research due to its validity in the general population and ease of administration, but it is neither the most comprehensive nor the most reliable measure [11]. We used the classification tree method recommended by Theou et al. to remain consistent throughout our data collection [30]. Finally, our analyses comparing TMT with patient outcomes and comparing frailty between cohorts were underpowered and exploratory—intended to inform the design of future, adequately powered studies rather than establish predictive validity in isolation.

5. Conclusion

TMT is moderately correlated with premorbid clinical frailty in patients with SABI. TMT may serve as a routinely available radiologic biomarker to characterize the sarcopenic dimension of frailty in this population. This study represents an early step in refinement and development of multidimensional frailty measures, while highlighting the need for future studies of reliability, generalizability, and validation in combination with other clinically relevant biomarkers.

Disclosure of potential conflicts of interest

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The authors declare that they have no additional conflicts of interest.

Declaration of the use of generative AI and AI-assisted technologies in scientific writing and in figures, images and artwork

did not use AI

Data statement

The data supporting the findings of this study are available upon reasonable request from the corresponding author. The data are not publicly available due to privacy and/or ethical restrictions.

CRediT authorship contribution statement

Jonathan Tam: Writing – review & editing, Writing – original draft, Visualization, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Annabelle Tosh:** Writing – review & editing, Investigation, Formal analysis, Data curation. **Patrick J Coppler:** Writing – review & editing, Methodology, Formal analysis, Conceptualization. **Laura Faiver:** Writing – review & editing, Visualization, Investigation, Conceptualization. **Nicholas Case:** Writing – review & editing, Visualization, Validation. **Clifton Callaway:** Writing – review & editing, Validation, Supervision, Methodology, Conceptualization. **Jonathan Elmer:** Writing – review & editing, Validation, Supervision, Methodology, Formal analysis, Conceptualization.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Jonathan Tam reports financial support was provided by National Institutes of Health. Laura Faiver reports financial support was provided by National Institutes of Health. Jonathan Elmer reports financial support was provided by National Institutes of Health. Patrick Coppler reports financial support was provided by National Institutes of Health. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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None

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jarlif.2026.100070.

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