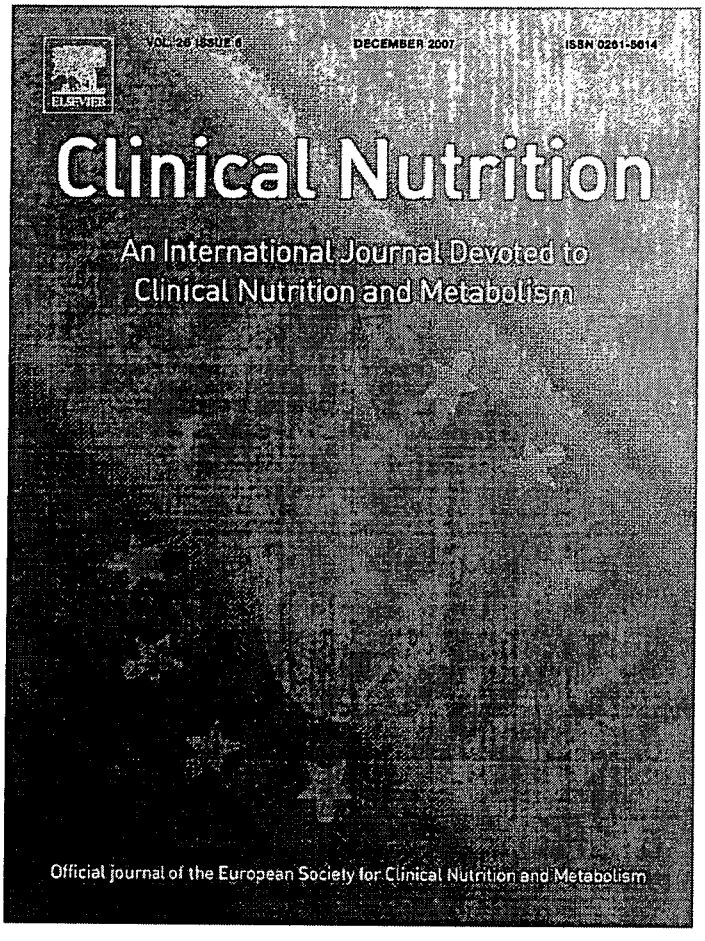
[12] Burton EJ, McKeigh IG, Burn DJ, et al. Cerebral atrophy in Parkinson's disease with and without dementia: a comparison with Alzheimer's disease, dementia with Lewy bodies and controls. Brain 2004;127:791–800.
[13] Camus V, Kraehenbuhl H, Preisig M, et al. Geriatric depression and vascular diseases: what are the links? J Affect Disord 2004;81:1–16.
[14] Carmelli D, Swan GE, Reed T, et al. Midlife cardiovascular risk factors and brain morphology in identical older male twins. Neurology 1999;52:1119–24.
[15] Chen P, Yu ES, Liu WT, et al. ADL dependence and medical conditions in Chinese older persons: a population-based survey in Shangai, China. J Am Geriatr Soc 1995;43(4):378–83.
[16] Convit A, Wolf OT, Tarshish C, et al. Reduced glucose tolerance is associated with poor memory performance and hippocampal atrophy among normal elderly. Proc Natl Acad Sci USA 2003;100:2019–22.
[17] Craft S, Watson GS. Insulin and neurodegenerative disease: shared and specific mechanisms. Lancet Neurol 2004;3:169–78.
[18] Craft S. Insulin resistance and cognitive impairment. Arch Neurol 2005;62:1043–4.
[19] Davison KK, Ford ES, Cogswell ME, et al. Percentage of body fat and body mass index are associated with mobility limitations in people aged 70 and older from NHANES III. JAGS 2002;50:1802–9.
[20] De La Torree JC. Alzheimer's disease is a vasocognopathy: a new term to describe its nature. Neurol Res 2004;26(5):517–24.
[21] De Leeuw FE, de Groot JC, Achten E, et al. Prevalence of cerebral white matter lesions in elderly people: a population-based magnetic resonance imaging study. The Rotterdam Scan Study. J Neurol Neurosurg Psychiatry 2001;70(1):9–14.
[22] Depp CA, Jeste DV. Definitions and predictors of successful aging: a comprehensive review of larger quantitative studies. Am J Geriatr Psychiatry 2006;14(1):6–20.
[23] Di Legge S, Piero VD, Altieri M, et al. Usefulness of primitive reflexes in demented and non-demented cerebrovascular patients in daily clinical practice. Eur Neurol 2002;45:104–10.
[24] Duncan BB, Schmidt MI, Polanczyk CA, et al. High mortality rates among Brazilian adult populations—an international comparison. Rev Assoc Med Br 1992;38:138–44 [Portuguese].
[25] Eguchi K, Kario K, Shimada K, et al. Greater impact of coexistence of hypertension and diabetes on silent cerebral infarcts. Stroke 2003:2471–4.
[26] Farkas E, De Jong G, Apro E, et al. Similar ultrastructural breakdown of cerebrocortical capillaries in Alzheimer's disease, Parkinson disease, and experimental hypertension. What is the functional link? Ann NY Acad Sci 2000;903:72–82.
[27] Ferrand J. Essai sur l'hémiplégie des veillards: les lacunes de désintégration cerébrale. Paris These, 1902 [French].
[28] Firbank M, O'Brien JT, Pakrasi S, et al. White matter hyperintensities and depression—preliminary results from the LADIS study. Int J Geriatr Psychiatry 2005;20:674–9.
[29] Folstein MF, et al. Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. J Psychiatry Res 1975;12:189–98.
[30] Ford ES, Giles WH, Dietz WH, et al. Prevalence of the metabolic syndrome among US adults: findings from the third national health and nutrition examination survey. JAMA 2002;287:356–9.
[31] Fundação Instituto Brasileiro de Geografia e Estatistica (IBGE). (Portuguese) [On-line] available at: http://www.ibge.gov.br [accessed 30-11-2005].
[32] Furukawa S, Fujita T, Shimabukuro M, et al. Increased oxidative stress in obesity and its impact on metabolic syndrome. J Clin Invest 2004:1752–61.
[33] Gardner JP, Li S, Srinivasan SR, et al. Rise in insulin resistance is associated with escalated telomere attrition. Circulation 2005;111: 2171–7.
[34] Geroldi C, Frisoni GB, Paolisso G, et al. Insulin resistance in cognitive impairment. The Inchanti study. J Am Geriatr Soc 2005;62: 1067–72.
[35] Guccione AG, Felson DT, Anderson JJ, et al. The effects of specifical medical conditions on the functional limitations of elders in the Framingham Study. Am J Public Health 1993;84:351–8.
[36] Gustafson D, Rothenberg E, Bjorkelung C, et al. An 18-year follow-up of overweight and risk of Alzheimer's disease. Arch Intern Med 2003;163:1524–8.
[37] Hachinsk V. Preventable senility: a call for action against the vascular dementias. Lancet 1992;340:645–8.
[38] Hachinski VC, Lassen NA. Multi-infarct dementia: a cause of mental deterioration in the elderly. Lancet 1974:2207–10.
[39] Haslam DW, James WPT, et al. Obesity. Lancet 2005;366:1197–209.
[40] Hausdorff JM, Yogev G, Springer S, et al. Walking is more like catching than tapping: gait in the elderly as a complex cognitive task. Exp Brain Res 2005;164:541–8.
[42] Hickman S, Howieson DB, Dame A, et al. Longitudinal analysis of the effects of the aging process on neuropsychological test performance in the healthy young-old and oldest-old. Dev Neuropsychol 2000;17:323–37.
[43] Hyung-Min K, Kim BJ. Metabolic syndrome as an independent risk factor of silent brain infarction in healthy people. Stroke 2006;37:466–72.
[44] Ishii N, Nishihara Y, Imamura T. Why do frontal lobe symptoms predominate in vascular dementia with lacunes? Neurology 1986;36:340–5.
[45] James WPT, Leach RJ, Mhurch CN, et al. Overweight and obesity (high body mass index). Geneva, Switzerland: World Health Organization; 2004. p. 497–596.
[46] Jensen GL. Obesity and functional decline: epidemiology and geriatric consequences. Clin Geriatr Med 2005;21:677–87.
[47] Kahn R, Buse J, Ferrannini E, et al. The metabolic syndrome: time for a critical appraisal—joint statement from the American Diabetes Association and the European association for the Study of Diabetes. Diabetes Care 2005;28:2289–304.
[48] Kalaria R. Similarities between Alzheimer's disease and vascular dementia. J Neurol Sci 2002;203/204:29–34.
[49] Kalmijn S, Foley D, White L, et al. Metabolic cardiovascular syndrome and risk of dementia in Japanese-American elderly men: the Honolulu-Asia aging study. Arterioscler Thromb Vasc Biol 2000;20: 2255–60.
[50] Kario K, Matsuo T, Kobayashi BA, et al. Hyperinsulinemia and haemostatic abnormalities are associated with silent lacunar cerebral infarcts in elderly hypertensive subjects. J Am Coll Cardiol 2001;37:871–7.
[51] Kobayashi S, Okada K, Koide H, et al. Subcortical silent brain infarction as a risk factor for clinical stroke. Stroke 1997;28:1932–9.
[52] Koyano W, Shibata H, Nakazato K, et al. Measurement of competence: reliability and validity of the TMIG-index of competence. Arch Gerontol Geriatr 1991;13:103–16.
[53] Kumari M, Brunner E, Fuhrer R, et al. Minireview: mechanisms by which the metabolic syndrome and diabetes impair memory. J Gerontol A: Biol Sci Med Sci 2000;55:B228–32.
[54] Kuo H-K, Lipsitz LA. Cerebral white matter changes and geriatric syndromes: is there a link? J Gerontol: Biol Sci Med 2004:M818–26.
[55] Kuusisto J, Koivisto K, Mykkanen L, et al. Association between features of the insulin resistance and Alzheimer's disease independently of apolipoprotein E4 phenotype: crosssectional population-based study. BMJ 1997;315:1045–9.
[56] Kwon HM, Kim BJ, Lee SH, et al. Metabolic syndrome as an independent risk factor of silent brain infarction in healthy people. Stroke 2006;37:466–72.
[57] Laakso MP, Partanen K, Riekkinen P, et al. Hippocampal volumes in Alzheimer's disease, Parkinson's disease with and without dementia, and in vascular dementia: an MRI study. Neurology 1996;46:678–81.
[58] Lesser IM, Boone KB, Mehringer CM, et al. Am J Psychiatry. Cognition and white matter hyperintensities in older depressed patients 1996;153:1280–7.
[59] Lotufo PA. Stroke in Brazil: a neglected disease. Sao Paulo Med J 2005;123:3–4.

[60] Luichsinger J, Tang M, SheaS, et al. Hyperinsulinemia and risk of Alzheimer disease. Neurology 2004;63:1187–92.
[61] Marie P. Des Foyers lacunaires de désintégration et de différents autres états cavitaires du cerveau. Rev Med 1901;21:281–98 [French].
[62] McLaren D. Is insulin resistance becoming a global epidemic? Nutrition 1997;13:64–6.
[63] Milionis HJ, Rizos MHJ, Goudevenos J, et al. Components of the Met.S and risk for first-ever acute ischemic non-embolic stroke in elderly subjects. Stroke 2005;36:1372–6.
[64] Monteiro CA. The nutrition transition in Brazil. Eur J Clin Nutr 1995;49:105–13.
[65] Morrison JH, Hof PR. Life and death of neurons in the aging brain. Science 1997;278:412–9.
[66] Murray CJL, Lopez AD. Alternative projections of mortality and disability by cause 1990–2020: Global Burden of Disease Study. Lancet 1997;349:1498–504.
[67] Murray CJL, Lopez AD. Global mortality, disability, and the contribution of risk factors: Global Burden of Disease Study. Lancet 1997;349:1436–42.
[68] National System of Health Information, Brazil. (Portuguese) [On-line] available at: http://tabnet.datasus.gov.br [accessed 8-11-2005].
[69] Ninomiya J, L'Italien F, Criqui MH, et al. Association of the metabolic syndrome with history of myocardial infarction and stroke in the Third National Health and Nutrition Examination Survey. Circulation 2004;13:42–6.
[70] O'Brien J, Ames D, Schwietzer I, et al. White matter changes in depression and Alzheimer's disease: a review of magnetic resonance imaging studies. Int J Geriatr Psychiatry 1996;11:681–94.
[71] Olijhoek J, van der Graaf Y, Jan-Dirk B, et al. The metabolic syndrome is associated with advanced vascular damage in patients with coronary heart disease, stroke, peripheral arterial disease or abdominal aortic aneurysm. Eur Heart J 2004;25:342–8.
[72] Osler W. The principle and practice of medicine. New York: D. Appleton; 1892.
[73] Otiniano ME, Du XL, Maldonado MR, et al. Effect of metabolic syndrome on heart attack and mortality in Mexican-American elderly persons: findings of a 7-year follow-up from the Hispanic established population for the epidemiological study of the elderly. J Gerontol A: Biol Sci Med Sci 2005;60:466–70.
[74] Palloni A, Pinto-Aguirre, Martha P. Demographic and health conditions of ageing in Latin America and the Caribbean. Int J Epidemiol 2002;31:762–71.
[75] Pantoni L, Basile AM, Pracucci G, et al. Impact of age-related cerebral white matter changes on the transition to disability: the LADIS study—rationale, design and methodology. Neuroepidemiology 2005;24(1–2):51–62.
[76] Pantoni L, Gracia JH. The significance of cerebral white matter abnormalities 100 years after Biswanger report. A review. Stroke 1995;26:1293–301.
[77] Perennou D, Decavel P, Manckoundia P, et al. Evaluation of balance in neurologic and geriatric disorders. Ann Readapt Med Phys 2005;48(6):317–35 [Francês].
[78] Petrella RJ, Lattanzio CN, Desmeray A, et al. Can adoption of regular exercise later in life prevent metabolic risk for cardiovascular disease? Diabetes Care 2005;28:694–701.
[79] Plaschke K. Aspects of ageing in chronic cerebral oligaemia. Mechanisms of degeneration and compensation in rat models. J Neural Transm 2005;112:393–413.
[80] Podsiadlo D, Richardson S. The timed "Up and Go": a test of basic functional mobility for frail elderly persons. JAGS 1991;39:142–3.
[81] Prins ND, van Dijk EJ, der Heijer T, et al. Cerebral small-vessel disease and decline in information processing speed, executive function and memory. Brain 2005;128:2034–41.
[82] Pugh KG, Lipsitz LA. The microvascular frontal-subcortical syndrome of aging. Neurobiol Aging 2002;23:421–31.
[83] Rao R, Jackson S, Howard R, et al. Primitive reflexes in cerebrovascular disease: a community study of older people with stroke and carotid stenosis. Int J Geriatr Psychiatr 1999;14:964–72.
[84] Rasgon NL, Kenna HA. Insulin resistance in depressive disorders and Alzheimer's disease: revising the missing link hypothesis. Neurobiol Aging 2005;26(Suppl. 1):23–7.
[85] Roberts EL, Chich CP. Age-related alterations in energy metabolism contribute to the increased vulnerability of the aging brain to anoxic damage. Brain Res 1995;678:83–90.
[86] Rowe J, Kahn RL. Human aging: usual and successful. Science 1987;237:143–9.
[87] Saposnik G, Caplan LR, Gonzalez LA, et al. Stroke in South America: a systematic review of incidence, prevalence, and stroke subtypes. Stroke 2003:2103–7.
[88] Snowdon DA, Greiner LH, Mortimer JA, et al. Brain infarction and the clinical expression of Alzheimer's disease. The Nun Study. J Am Med Assoc 1997;277:813–7.
[89] Tinnetti ME, de Leon CFM, Doucette JT, et al. Fear of falling and fall-related efficacy in relationship to functioning among community-dwelling elders. J Gerontol 1990;45:239–43.
[90] Touboul PJ, Labreuche J, Vicaut E, et al. Carotid intima-media thickness, plaques, and Framingham risk score as independent determinants of stroke risk. Stroke 2005:1741–5.
[91] Trindade IS, Heineck G, Machado JR, et al. Prevalence of arterial hypertension in the population of Passo Fundo (Brazil) metropolitan area. Arq Bras Cardiol 1998;71:127–30 [Portuguese].
[92] Vanneste JAL. Diagnosis and management of normal-pressure hydrocephalus. J Neurol 2000;247:5–14.
[93] Veemer SE, Koudstaal PJ, Oudkerk M, et al. Prevalence and risk factors of silent brain infarcts in the population-based Rotterdam Scan Study. Stroke 2002:21–5.
[94] Verguese J, Lipton RB, Hall CB, et al. Abnormality of gait as a predictor of non-Alzheimer's dementia. N Engl J Med 2002;347:1761–8.
[95] Watson GS, Craft S. The role of insulin resistance in the pathogenesis of Alzheimer's disease. Implications for treatment. CNS Drugs 2003;17:27–45.
[96] Wolff SP, Jiang ZY, Hunt JV. Protein glycation and oxidative stress in diabetes mellitus and ageing. Free Radic Biol Med 1991;10:339–52.
[97] World Health Organization. Research protocol for measuring the prevalence of neurological disorders in developing countries. Neurosciences Program, Geneva: WHO; 1981.
[98] Yaffe K, Kanaya A, Lindquist K, et al. The metabolic syndrome, inflammation, and risk of cognitive decline. J Am Med Assoc 2004;292:2237–42.
[99] Zunker P. Hyperinsulinism and cerebral microangiopathy. Stroke 1996;27:219–23.

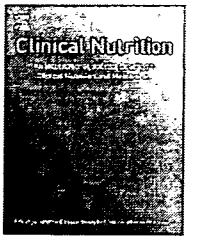


Clinical Nutrition (2007) 26, 764–770

Available at www.sciencedirect.com

ScienceDirect

http://intl.elsevierhealth.com/journals/clnu



ORIGINAL ARTICLE

Lack of body weight measurement is associated with mortality and hospitalization in community-dwelling frail elderly

Sachiko Izawa[a], Hiromi Enoki[a], Yoshihisa Hirakawa[a], Yuichiro Masuda[a], Mitsunaga Iwata[b], Jun Hasegawa[a], Akihisa Iguchi[a], Masafumi Kuzuya[a,*]

[a]*Department of Geriatrics, Nagoya University Graduate School of Medicine, 65 Tusruma-cho, Showa-ku, Nagoya 466-8550, Japan*
[b]*Emergency Department, Nagoya Ekisaikai Hospital, 4-66 Shonen-cho, Nakagawa-ku, Nagoya 454-8502, Japan*

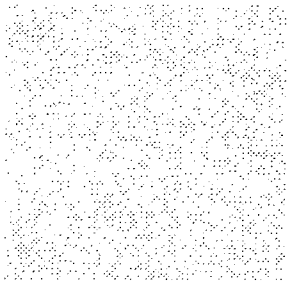
Received 12 May 2007; accepted 30 August 2007

KEYWORDS
Anthropometric measurement;
Weight;
Mortality;
Hospitalization;
Elderly

Summary
Background & aims: Although it is not uncommon for there to be frail older people living in the community, who do not know their weight and/or height, the health-related outcomes of those older remains unknown. We examined whether missing these anthropometries are a predictor of mortality or hospitalization during a 2-year follow-up period in community-dwelling older people using various community-based services.
Methods: This study was a prospective cohort analysis of 952 community-dwelling elderly. Data included the clients' demographic characteristics, basic activities of daily living (ADL), comorbidity, and anthropometric measurements at baseline. Analysis of mortality and hospitalization over the 2-year period was conducted using multivariate Cox proportional hazards models.
Results: Among the 952 participants, 342 and 292 had missing data for height and weight at baseline, respectively. Multivariate Cox proportional hazards models adjusting for potential confounders showed that the lack of data on weight was associated with 2-year mortality (hazard ratio, HR:1.54, 96% CI:1.09–1.79) as well as hospitalization (HR:1.34,

*Corresponding author. Tel.: +81 52 744 2364; fax: +81 52 744 2371.
E-mail address: kuzuya@med.nagoya-u.ac.jp (M. Kuzuya).

0261-5614/$ - see front matter
doi:10.1016/j.clnu.2007.08.007



95% CI:1.01–1.79) during the 2-year follow-up, although the lack of height measurement was not associated with these adverse outcomes.
Conclusions: Older people living in the community with unavailable weight data appear to be more likely to have a high risk of mortality and hospitalization.

Introduction

Height and weight are two of the most commonly used anthropometric measurements to assess nutritional status and overall health in clinical practice and research.[1–3] Changes in weight or body mass index (BMI), the most widely used anthropometric index, during certain periods are frequently used to determine whether nutritional status or overall health status has changed. In fact, a number of studies have demonstrated that weight loss or BMI reduction is an independent risk factor of adverse outcomes for older people, including high mortality and functional decline.[4–8]

In addition, low weight or low BMI levels themselves have been demonstrated to be a predictor of adverse outcomes including mortality or prolonged hospital stays in the older people.[9–11]

However, it is not uncommon for there to be frail and non-ambulatory older people, especially older people living in the community, who do not know their weight and/or height, or cannot be weighed or measured for height due to disabilities or postural changes. In fact, when subjects living at home have severe functional disabilities, it is essential to have special equipment such as beds or wheelchair scales to measure their weight. In addition to weight measurements, height measurements are also essential in the calculation of BMI levels. It is also not uncommon for it to be difficult to measure the height for the older people with postural changes, including muscle and arterial contracture and kyphosis. Thus, for older people with disabilities, data on anthropometry such as height and weight are likely to be missing, and therefore these individuals are likely to be under-represented in many comparisons. Whereas BMI that is generally regarded as the most widely used anthropometric index, in most previous studies those elderly with missing data for height, body weight, and BMI are excluded from the analysis. Analysis of only the available data leads to findings that apply only to a subgroup of the original population of interest, and that subgroup cannot be prospectively identified. However, the health-related outcomes of those people who cannot measure these essential anthropometries at home or do not know recent their anthropometries remains unknown.

In the present prospective cohort study we tried to determine whether lack of height or body weight measurement is a predictor of mortality or hospitalization during a 2-year follow-up period in community-dwelling older people using various community-based services.

Methods

Subjects

The present study employed baseline data of the subgroup of participants in the Nagoya Longitudinal Study for Frail Elderly (NLS-FE) and data on the mortality and hospitalization of these participants during the 2-year follow-up period. Details of participants and the NLS-FE have been published elsewhere.[12,13] The study population consisted of 952 community-dwelling frail elderly (355 men and 597 women, age 65 years or older) who were eligible for long-term care insurance (LTCI) program,[14–16] lived in Nagoya City, and were provided visiting nurse services from the Nagoya City Health Care Service Foundation for Older People, which has 17 visiting nursing stations associated with care-managing centers. The LTCI system covers care for both the elderly aged 65 and older. Under the LTCI program, care levels (levels 0–5) are determined according to eligibility criteria. The elderly in the community who are eligible for LTCI are disabled and chronically ill, have physical and mental problems, and are easy to admit acute hospital or institute care setting.[14–16] These NLS-FE participants, who were enrolled between 1 December 2003 and 31 January 2004, were scheduled to undergo comprehensive in-home assessments by trained nurses at the baseline, and at 6, 12, and 24 months. At 3-month intervals, data were collected about any important events in the lives of the participants, including admission to the hospital and mortality. Written informed consent for participation, according to procedures approved by the institutional review board of Nagoya University Graduate School of Medicine, was obtained from the patients or, for those with substantial cognitive impairment, from a surrogate (usually the closest relative or legal guardian).

Data collection

The data were collected at the clients' homes from standardized interviews with patients or surrogates and caregivers, and from care-managing center records taken by trained nurses. The data included clients' demographic characteristics, depressive symptoms as assessed by the short version of the Geriatric Depression Scale (GDS-15),[17] and a rating for 10 basic activities of daily living (ADL) (feeding, mobility on bed, bathing, grooming, dressing, using the toilet, walking inside and outside, transferring, and using stairs) using summary scores ranging from 0

(total disability) to 20 (no disability). The interview with participants also included questions about the utilization of care services, including the day-care service and home-help service programs, as well as medical services. Information obtained from care-managing center records included data on the following physician-diagnosed chronic conditions: ischemic heart disease, congestive heart failure, cerebrovascular disease, diabetes mellitus, dementia, cancer, neurodegenerative disorders including Parkinson's disease, and other diseases comprising the Charlson Comorbidity Index,[18] which represents the sum of weighted indexes, taking into account the number and seriousness of preexisting comorbid conditions. The data also included the number of prescribed medications. Of these 952 participants, 276 could not complete the GDS-15 because of severe cognitive impairment or communication impairment.

Height and weight data were generally measured at home and collected by nurses. The visiting nurses were asked to measure the height or weight of participants at home as much as possible. In the case that body weight measurements could not be taken at home for some reason, recorded or self-reported weight data obtained sometime within the last month was used. Weight was measured in light clothing without shoes using a portable weight scale at home. Height was generally measured in an upright position using a tape measure attached to the wall. However, when participants could not persist in an upright position, height measurements were obtained in a prone position. Height measurements were unavailable for subjects with severe kyphosis (defined as any subject whose kyphosis made it impossible for the visiting nurse to make a convenient or reliable height measurement) or severe muscle and arterial contracture.

Measurement of the triceps skin fold (TSF) thickness (to the nearest 0.1 mm) was made using skin-fold calipers and mid-upper arm circumference (AC) (to the nearest 0.1 cm) using a flexible measuring tape on the right side of the participant's body, unless affected by disability or disease. Arm area (AMA) was calculated using standard formulas: $AMA = (AC(cm) - 0.3142 \times TSF(mm))^2/4\pi$.

Of the 342 participants for whom height data were unavailable, 328 (95.9%) and 326 (95.3%) were available for their TSF and AC, respectively. Of the 292 participants for whom weight data were unavailable, 280 (95.9%) and 277 (94.9%) were available for their TSF and AC, respectively.

Statistical analysis

The Student's *t*-test and Chi-squared test were used to compare differences between participants with available and not available height data or between those with available and not available weight data. To evaluate the risk of participants with missing height or weight data, which was expressed as an odds ratio (OR) with a corresponding 95% confidence interval (CI), logistic regression models were used. The models included factors that differed significantly between participants with incomplete and complete anthropometric measurements.

Survival curves describing mortality and hospitalization over the 2 years after enrollment in participants with or without missing weight or height data at baseline were conducted using the Kaplan–Meier method and compared with the log-rank statistic. Cox proportional hazard models were used to assess the association of lack of height or weight measurement at baseline with 2-year mortality or hospitalization during a 2-year period. To create an ideal model for a multivariate Cox proportional hazards models, we selected covariates as follows. We first evaluated the univariate association between each covariate at baseline using the chi-square test for categorical and the *t*-test for continuous variables. Next, we evaluated the association between each covariate and 2-year mortality or hospitalization during the 2-year period using the univariate Cox proportional hazards model. We then sequentially evaluated the impact of each of the remaining covariates on the overall model fit through a series of Cox proportional hazards models. The risk of a variable was expressed as a hazard ratio (HR) with a corresponding 95% CI.

All analyses were performed using the Statistical Package for the Social Sciences (SPSS) Version 14.0. A probability value of 0.05 or less was considered significant.

Results

Baseline characteristics

Among the 952 participants, 342 (35.9%) and 292 (30.7%) had missing data for height and weight at baseline, respectively. Among the 610 participants with height data available, 96 (15.7%) had missing data for weight, and among the 660 participants with weight data available, 146 (22.1%) had missing data on height. As such, BMI was unavailable in 438 participants (46.0%).

Table 1 shows the comparisons of baseline characteristics of participants having or missing data for height or weight. Participants missing these anthropometric measurements were significantly more likely to be women, to be older, to have lower ADL scores, to have lower day-care service use, a lower rate to 3 and more regular prescribing medication, lower AC, and a higher prevalence of dementia and pressure sores compared with participants with anthropometric measurements at baseline. Participants with missing weight measurements were significantly more likely to have higher comorbidity, to have a lower rate of living alone, and to have lower AMA than those with available weight measurements.

Factors related to lack of measurement

To identify the factors associated with missing height and weight data, two different multivariate logistic regression models were conducted (Table 2). Model 1 is based on the inclusion of factors that differed significantly between participants with incomplete and complete anthropometric measurements. Model 2 includes the presence or absence of cerebrovascular disease, dementia, or pressure sores instead of the Charlson comorbidity index. We obtained comparable results with these two models, suggesting that age and ADL levels are determinants of the presence or absence of height and weight measurements: older and lower ADL function are associated with missing these anthropometric measurements. In addition, those utilizing

Table 1 Base line characteristics of the 952 care recipients.

	Total	Height		p	Weight		p
	$n = 952$	Available $n = 610$	Not available $n = 342$		Available $n = 660$	Not available $n = 292$	
Men/women, *n* (% of men/total)	355/597 (37.3)	254/356 (41.6)	101/241 (29.5)	<0.001	263/397 (39.8)	92/200 (31.5)	0.014
Age (years), mean (SD)*	80.5 (7.9)	79.8 (7.9)	81.6 (7.8)	<0.001	79.7 (7.8)	82.2 (8.1)	<0.001
Basic ADL (range, 0–20), mean (SD)*	10.3 (6.9)	11.4 (6.7)	8.3 (6.9)	<0.001	12.2 (6.2)	6.0 (6.6)	<0.001
GDS-15 (range, 0–15), mean (SD)†	7.1 (3.6)	7.1 (3.6)	6.9 (3.5)	0.400	7.0 (3.6)	7.2 (3.6)	0.655
Charlson comorbidity index, mean (SD)*	2.3 (1.6)	2.3 (1.6)	2.4 (1.6)	0.120	2.2 (1.7)	2.6 (1.5)	<0.001
Day-care (service) use (% of total)	35.6	33.0	40.4	0.022	38.9	28.1	0.001
Home help service use (% of total)	51.7	49.5	55.6	0.073	50.5	54.5	0.255
Regular medical checkups (% of total)	73.4	74.5	71.3	0.283	72.7	75.0	0.456
Three or more regular prescription medications (% of total)	79.9	82.0	76.2	0.035	84.7	69.1	<0.001
Living alone (% of total)	19.0	20.6	16.0	0.086	21.6	12.9	0.002
Mid arm circumference (cm), mean (SD)	23.5 (4.4)	23.7 (4.6)	23.1 (4.1)	0.031	23.9 (4.6)	22.6 (3.8)	<0.001
Triceps skin fold (cm), mean (SD)	1.4 (0.9)	1.5 (0.9)	1.4 (0.8)	0.067	1.5 (0.9)	1.4 (0.9)	0.590
Arm muscle area (cm^2), mean (SD)	29.6 (11.7)	29.9 (11.8)	29.1 (11.5)	0.280	30.7 (11.9)	27.1 (10.7)	<0.001
Chronic diseases (% of total)							
Ischemic heart disease	11.6	13.0	9.3	0.117	11.9	11.0	0.702
Congestive heart failure	11.2	10.9	11.7	0.719	11.7	9.7	0.401
Cerebrovascular disease	40.7	39.4	43.0	0.326	38.3	46.4	0.033
Diabetes mellitis	13.1	13.5	12.4	0.640	11.9	16.0	0.114
Dementia	38.1	33.7	46.0	<0.001	30.7	56.1	<0.001
Cancer	10.5	11.0	9.6	0.525	11.7	7.6	0.080
Hypertension	23.1	23.6	22.2	0.627	23.5	22.3	0.679
Pressure sore	11.6	9.2	15.8	0.002	7.0	21.9	<0.001

*Student *t*-test, others were analyzed by χ^2 test (user vs. nonuser).
†GDS-15: geriatric depression scale, total: $n = 676$.

day-care services were more likely to have their weight measurement but not height measurement. Furthermore, model 2 indicated that the presence of dementia or pressure sores is associated with missing weight measurement but not missing height.

Prospective study

Participants with missing weight measurement had a higher mortality rate (having: 18.0%; missing: 38.4%, $p < 0.001$) and hospitalization (having: 32.6%; missing: 40.8%, $p = 0.015$) during the 2-year follow-up compared with those with available weight measurements. Participants with missing height measurement had higher mortality (having: 22.1%; missing: 28.1%, $p = 0.04$), but there was no difference in hospitalization (having: 33.3%; missing: 38.3%, $p = 0.119$).

Figure 1 shows the Kaplan–Meier curves describing mortality and hospitalization over the 2 years in participants with or without missing weight or height data at baseline. The missing weight and height data at baseline were significantly associated with lower survival rate during 2-years follow-up (Log rank test: $p < 0.0001$ and $p = 0.030$, respectively) (Figure 1A and C). Although the missing weight data at the baseline was associated with higher hospitalization during 2-years follow-up, the missing height data was not (Log rank test: $p = 0.001$ and $p = 0.067$, respectively) (Figure B and D).

Table 3 provides the results of the series of Cox proportional hazards models to examine the HRs of missing height and weight data at baseline for 2-year mortality and hospitalization during 2-year follow-up. In the unadjusted models, missing data for height were significantly associated with an elevated risk for 2-year mortality, but not for hospitalization during the 2-year follow-up. For 2-year mortality, sequential adjustment for the covariates lost the association between participants with missing data on height and 2-year mortality. In unadjusted models, missing weight data were significantly associated with elevated risks for 2-year mortality and for hospitalization during follow-up periods. The sequential adjustment lowered the hazard for mortality associated with missing weight data, but the association remained statistically significant even after the fully adjusted model. For hospitalization, the rather constant HR associated with participants with missing weight data was observed even after full adjustment.

Discussion

In the present study we demonstrated that approximately one-third of elderly participants who are older people living

Table 2 Logistic regression analysis to identify independent predictors of lack of anthropometric measurements.

Base line variables	Model 1*				Model 2†			
	Height		Weight		Height		Weight	
	OR‡	95% CI	OR	95% CI	OR‡	95% CI	OR	95% CI
Men (vs. women)	0.74	0.53–1.02	0.86	0.59–1.26	0.74	0.53–1.03	0.87	0.59–1.27
Age (continous variable)	1.03	1.01–1.05	1.03	1.01–1.06	1.03	1.00–1.05	1.03	1.01–1.05
ADL score (continous variable, range, 0–20)	0.94	0.91–0.96	0.87	0.84–0.89	0.94	0.91–0.97	0.88	0.85–0.91
Living alone (vs. with others)	1.16	0.76–1.76	1.55	0.94–2.56	1.15	0.75–1.75	1.54	0.93–2.56
Day-car service use (vs. nonuse)	1.33	0.98–1.82	0.57	0.39–0.82	1.35	0.98–1.85	0.55	0.37–0.80
No of prescription medications (vs. <3)								
3–5	1.07	0.70–1.64	0.74	0.46–1.17	1.08	0.71–1.66	0.80	0.50–1.29
≧6	0.86	0.56–1.35	0.65	0.40–1.07	0.87	0.55–1.36	0.72	0.43–1.18
Charlson comorbidity index (continous variable)	1.00	0.90–1.10	1.03	0.92–1.16				
Presence of chronic diseases (vs. absence)								
Cerebrovascular disease					0.96	0.69–1.33	1.00	0.69–1.46
Dementia					1.01	0.72–1.42	1.48	1.01–2.18
Pressure sore					1.19	0.74–1.92	2.05	1.21–3.46

*Model 1 includes gender, age, ADL score, living arrangement at baseline, use or nonuse of day-care services, number of prescribed medication, and Charlson comorbidity index.
†Model 2 includes the presence or absence of cerebrovascular disease, dementia, or pressure sores instead of the Charlson comorbidity index.
‡Odds ratio (OR).

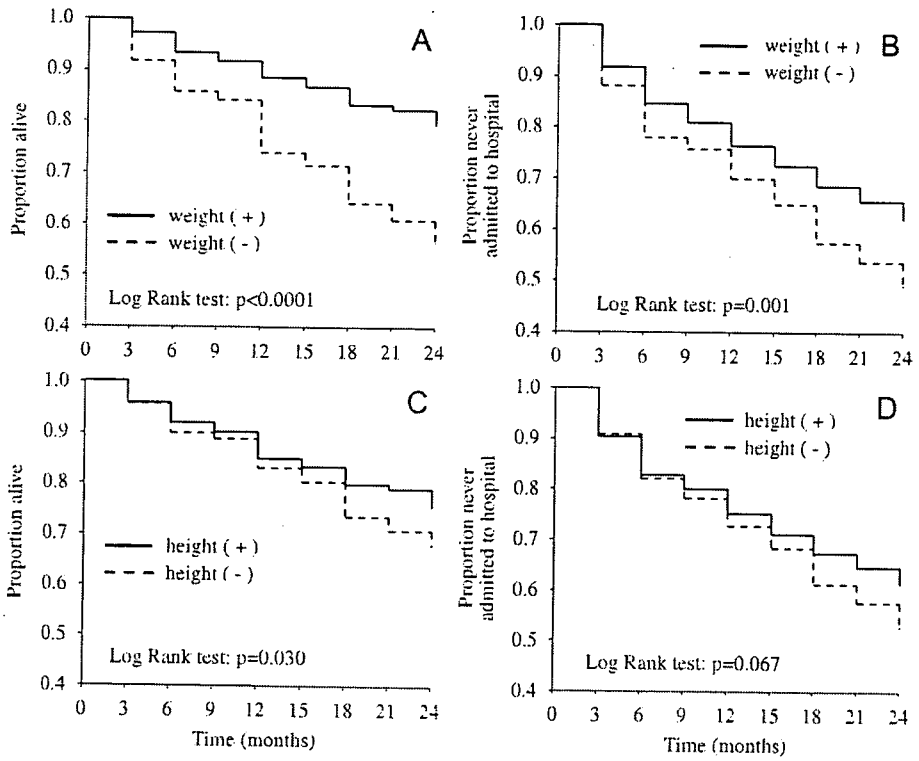


Figure 1 The Kaplan–Meier survival curves describing mortality (A, C) and hospitalization (B, D) over the 2-years after enrollment in participants with or without missing weight (A, B) or height (C, D) data at baseline.

at home and receiving informal or formal assistance under the LTCI program lack height or weight measurements. Among the 952 participants, 438 (46.0%) were missing data for height, weight, or both, indicating nearly half of the participants did not know their own BMI. Exact information regarding the reasons for the lack of height or weight

Table 3 Hazards risk of lack of anthropometric measurements according to Cox proportional hazard model to identify independent predictors of adverse events.

Variable		Measurements			
		Height		Weight	
		HR*	95% CI	HR	95% CI
Mortality					
Unadjusted		1.33	1.02–1.72	2.38	1.84–3.08
Adjusted†	Model 1	1.31	1.01–1.71	2.29	1.76–2.98
	Model 2	1.00	0.73–1.35	1.55	1.10–2.19
	Model 3	0.97	0.71–1.32	1.54	1.09–2.18
Hospitalization					
Unadjusted		1.22	0.98–1.52	1.44	1.15–1.80
Adjusted‡	Model 1	1.27	1.02–1.59	1.53	1.22–1.92
	Model 2	1.18	0.92–1.52	1.34	1.00–1.78
	Model 3	1.17	0.90–1.51	1.34	1.01–1.79

*HR: hazard risk.

†Model 1 includes age and gender; Model 2 includes age, gender, ADL score, living arrangement at baseline, number of regular medical check per week, number of prescribed medication, use or nonuse of day-care services, and presence or absence of chronic diseases (dementia, cancer, hypertension, or pressure sore); Model 3 includes factors in Model 2 and mid arm circumference.

‡Model 1 includes age and gender; Model 2 includes age, gender, ADL score, number of regular medical check per week, number of prescribed medication, presence or absence of cancer or pressure sore; Model 3 includes factors used in Model 2 and mid arm circumference.

measurement or lack of knowledge of these values at the baseline is not available in the present study. However, the association between older age or lower ADL function, and missing either anthropometric data may suggest that the height and weight of older people with ADL impairment are more likely to not be measured at home or to not regularly be measured in the community. It should be noted that a lack of weight measurement is strongly associated with ADL functional status of the subjects compared with that of height measurement, suggesting that participants with missing weight measurement are more functionally dependent older people. In addition, we showed that the presence of dementia and pressure sores is associated with a lack of weight measurement but not of height measurement. In fact, it is not unusual for the frail elderly to be unable to be weighed at home without hoist or wheelchair scales due to severe ADL impairment or the presence of advanced dementia or pressure sore. In contrast, it seems that there are obviously individuals whose height cannot be measured because of the presence of kyphosis or other postural problems. Thus there are different groups of community-dwelling elderly for whom either height or weight or both cannot be measured.

We observed that those utilizing day-care services are more likely to have their weight measurement, suggesting that clients' weight is often measured at the day-care (service) center. It is well known that older people, especially those with functional limitations, are particularly vulnerable to undernutrition, which may further impair functional ability and increase the incidence of morbidity and mortality.[9,10] Nutritional screening is, therefore, crucial for not only hospitalized elderly people but also for those living in the community. However, the results regarding the unexpected high rate of older people with missing major anthropometric measurements in the community indicate that regular nutritional screening is not conducted for older people receiving community-based services under the LTCI program by health care professionals.

We observed that a lack of weight data is associated with 2-year mortality as well as hospitalization during the 2-year follow-up, even after adjusting for potential confounders, including age, ADL status, and comorbidity. A lack of height measurements is not associated with these adverse outcomes, suggesting again that there are different groups of community-dwelling elderly in the community for whom height and weight cannot be measured. The mechanisms underlying the association between lack of weight measurement and mortality and hospitalization are unclear in the present study, but could be related to several factors. A large number of studies have shown that lower levels of weight, BMI, or weight loss is an important predictor of mortality in elderly peoples.[4–11] Although we do not know whether their weight or BMI was lower than that of subjects who had been weighed at the baseline and whether they had weight loss during the study periods, it is possible that the association with adverse outcomes might be due to the undernutrition of participants with missing data on weight, since AC and AMA levels, potential markers of nutritional status, of the subjects with missing data on weight were significantly lower than those of the subjects who were weighed. However, we observed that the association between a lack of weight measurement and mortality or hospitalization persisted even after adjusting for AC, suggesting that undernutrition alone at baseline cannot explain this association. However, we cannot exclude the possibility that our participants lacking weight measurements might

have experienced weight change during the study period. Weight loss frequently goes unrecognized in older people and may lead to undernutrition and poor clinical outcomes.[4,19,20] The lack of weight measurement or a lack of knowledge of weight change by patients themselves, care givers, or health care professionals, including visiting nurses and physicians, results in a lost chance to detect early changes in patients' health conditions and nutritional status, and to respond appropriately to their weight changes.

The present study cannot reveal whether lack of knowledge of their own weight, weight change itself, or other background factors related to nonmeasurement of weight beyond what we could measure and control for may contribute to the 2-year mortality and hospitalization during a 2-year follow-up period. Future study is needed to examine whether measuring the weight of these older people with functional limitations and monitoring of weight changes in the community may have a positive effect on mortality and morbidity.

This study has important limitations. First, as described above the exact reasons for missing data on height or weight are not clear in the present study. Therefore, the exact cause of poor outcomes of participants with lack of weight measurement at baseline during the 2-year observation was not evaluated. As described in the methods, participants included in our present study were limited to those using visiting nurse services, which may have introduced a selection bias into the study. Because of the observational design of the present study, differences in unmeasured factors, including the severity of chronic diseases of patients, may account in part for the findings. In addition, these findings may not be generalizable to other populations, given the differing health practices, a variety of social and economic factors, and ethnic attitudes regarding caring for very old people.

In the present study we showed that there are many older people living in the community with functional disability whose weight or height is not measured for various reasons. Those subjects, especially those for whom weight data are unavailable, are more likely to have high risk for mortality and hospitalization.

Conflict of interest statement

None declared.

Acknowledgments

The authors wish to thank all the patients, caregivers and the many nurses participating in the study, and the Nagoya City Health Care Service Foundation for Older People for their vigorous cooperation.

Financial disclosure(s): This study was supported by a Grant-in Aid for the Comprehensive Research on Aging and Health from the Ministry of Health, Labor, and Welfare of Japan, and a Grant from Mitsui Sumitomo Insurance Welfare Foundation. The authors have no conflicts of interest with the manufacturers of any drug evaluated in this paper.

Sponsor's role: The sponsor had no role in the design, methods, subject recruitment, data collection, analysis, or paper preparation.

References

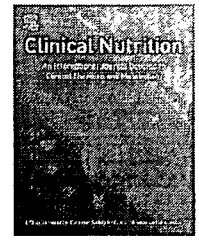
1. Omran ML, Morley JE. Assessment of protein energy malnutrition in older persons, part I: History, examination, body composition, and screening tools. *Nutrition* 2000;16:50–63.
2. Corish CA, Kennedy NP. Anthropometric measurements from a cross-sectional survey of Irish free-living elderly subjects with smoothed centile curves. *Br J Nutr* 2003;89:137–45.
3. Shatenstein B, Kergoat MJ, Nadon S. Anthropometric changes over 5 years in elderly Canadians by age, gender, and cognitive status. *J Gerontol A Biol Sci Med Sci* 2001;56:M483–8.
4. Newman AB, Yanez D, Harris T, Duxbury A, Enright PL, Fried LP. Cardiovascular Study Research Group. Weight change in old age and its association with mortality. *J Am Geriatr Sor* 2001;49:1309–18.
5. Corrada MM, Kawas CH, Mozaffar F, Paganini-Hill A. Association of body mass index and weight change with all-cause mortality in the elderly. *Am J Epidemiol* 2006;163:938–49.
6. Sullivan DH, Johnson LE, Bopp MM, Roberson PK. Prognostic significance of monthly weight fluctuations among older nursing home residents. *J Gerontol A Biol Sci Med Sci* 2004;59:M633–9.
7. Woo J, Ho SC, Sham A. Longitudinal changes in body mass index and body composition over 3 years and relationship to health outcomes in Hong Kong Chinese age 70 and older. *J Am Geriatr Soc* 2001;49:737–46.
8. Launer LJ, Harris T, Rumpel C, Madans J. Body mass index, weight change, and risk of mobility disability in middle-aged and older women. The epidemiologic follow-up study of NHANES I. *J Am Med Assoc* 1994;271:1093–8.
9. Flegal KM, Graubard BI, Williamson DF, Gail MH. Excess deaths associated with underweight, overweight, and obesity. *J Am Med Assoc* 2005;293:1861–77.
10. Breeze E, Clarke R, Shipley MJ, Marmot MG, Fletcher AE. Cause-specific mortality in old age in relation to body mass index in middle age and in old age: follow-up of the Whitehall cohort of male civil servants. *Int J Epidemiol* 2006;35:169–78.
11. Taylor Jr. DH, Ostbye T. The effect of middle-and old-age body mass index on short-term mortality in older people. *J Am Geriatr Soc* 2001;49:1319–26.
12. Kuzuya M, Masuda Y, Hirakawa Y, et al. Underuse of medications for chronic diseases in the oldest of community-dwelling older frail Japanese. *J Am Geriatr Soc* 2006;54:598–605.
13. Kuzuya M, Masuda Y, Hirakawa Y, et al. Day care service use is associated with lower mortality in community-dwelling frail older people. *J Am Geriatr Soc* 2006;9:1364–71.
14. Cambell JC, Ikegami N. Long-term care insurance comes to Japan. *Health Aff* 2000;19:26–39.
15. Ikegami K. Impact of public long-term care insurance in Japan. *Geriatr Gerontol Int* 2004;4:S146–8.
16. Izawa S, Kuzuya M, Okada K, et al. The nutritional status of frail elderly with care needs according to the mini-nutritional assessment. *Clin Nutr* 2006;25:962–7.
17. Geriatric Depression Scale. Psychopharmacol Bull 1988; 24: 709–11.
18. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis* 1987;40:373–83.
19. Alibhai SM, Greenwood C, Payette H. An approach to the management of unintentional weight loss in elderly people. *CMAJ* 2005;172:773–80.
20. Payette H, Coulombe C, Boutier V, Gray-Donald K. Nutrition risk factors for institutionalization in a free-living functionally dependent elderly population. *J Clin Epidemiol* 2000;53: 579–87.

Clinical Nutrition (2007) 26, 84–90

Available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevierhealth.com/journals/clnu



ORIGINAL ARTICLE

Is serum albumin a good marker for malnutrition in the physically impaired elderly?

Masafumi Kuzuya*, Sachiko Izawa, Hiromi Enoki, Kiwako Okada, Akihisa Iguchi

Department of Geriatrics, Nagoya University Graduate School of Medicine, 65 Tusruma-cho, Showa-ku, Nagoya 466-8550, Japan

Received 10 May 2006; accepted 31 July 2006

KEYWORDS
Albumin;
Malnutrition;
Elderly;
Physical impairment;
Nutritional assessment;
Anthropometry;
Cholesterol;
Subjective global assessment

Summary
Background and Aims: Although serum albumin is well known as a marker of nutritional status, it has remained unclear whether impaired physical function affects serum albumin concentrations in older people. We examined whether hypoalbuminemia can be used as a marker of malnutrition in elderly subjects with various levels of physical impairment.
Methods: A total of 262 elderly subjects without acute illness were enrolled from various geriatric settings. For the nutritional assessment, serum albumin, total cholesterol, anthropometric measurements, and subjective global assessment (SGA) were determined. Physical function was evaluated by rating score of activity of daily living (ADL).
Results: As a whole, participants' serum albumin levels correlated with various nutritional parameters including anthropometric measurements and levels of serum total cholesterol as well as the SGA evaluation. However, after adjusting for age and gender, serum albumin levels in participants with a low ADL function did not correlate with nutritional parameters. Approximately 80% participants with low ADL function who were evaluated as being well nourished according to SGA evaluation had serum albumin levels lower than 35 g/l.
Conclusions: The utility of serum albumin and the traditional cutoff (35 g/l) in older people with low ADL function is questionable even among those without inflammation.

Introduction

Malnutrition is a common finding in the elderly, not only in institutionalized populations but also in community-dwelling

*Corresponding author. Tel.: +81 52 744 2364; fax: +81 52 744 2371.
E-mail address: kuzuya@med.nagoya-u.ac.jp (M. Kuzuya).

0261-5614/$ - see front matter © 2006 Elsevier Ltd and European Society for Clinical Nutrition and Metabolism. All rights reserved.
doi:10.1016/j.clnu.2006.07.009

elderly, with prevalence rates ranging from 12% to 85%.[1,2] Malnutrition is associated with increased hospitalization, increased susceptibility to infection, decreased wound healing, reduced quality-of-life, and increased mortality in the elderly.[3,4]

Multidimensional screening tools such as subjective global assessment (SGA),[5] and anthropometry measurements such as body mass index (BMI), mid-arm circumference (MAC), calf circumference (CC), and skin-fold thickness are generally considered the most easily obtainable, inexpensive, and noninvasive method by which to assess nutritional state. Biochemical measurements such as serum albumin and total cholesterol are also well known as markers for protein energy malnutrition (PEM).[6,7] Among the biochemical parameters, serum albumin levels have long been considered a major measure of malnutrition. On the other hand, some reports have cautioned against using albumin as a measurement of nutritional status in hospitalized patients.[8–10] The criticism is based on the fact that albumin is inversely correlated with markers of inflammatory activity and can behave as an acute-phase reactant, with markedly reduced levels in the setting of acute illness. In addition, it remains unknown whether impaired physical function affects serum albumin concentrations in older people. Thus, we still do not know whether hypoalbuminemia can be used as a marker of malnutrition for elderly people at various levels of activities of daily living (ADL) impairment, especially in the absence of inflammation or acute illness.

In the present study we examined whether hypoalbuminemia defined by a serum albumin level lower than 35 g/l can be used as a marker of malnutrition in elderly subjects without inflammation or acute illness. In addition we also examined whether physical impairment may affect the serum albumin concentration among well-nourished older people.

Subjects and methods

Subjects

We enrolled 262 consecutive elderly subjects (86 males and 176 females, mean age ± SD: 81.8 ± 7.5; range: 65–95 years) from our geriatric outpatient clinic ($n = 69$), a nursing home ($n = 56$), and geriatric hospitals ($n = 72$). Among 262 participants 55 participants were receiving tube feeding and there were no participants receiving parenteral nutrition. The participants from geriatric hospitals were transfers from the acute care setting or from nursing homes for the care of chronic diseases or for the rehabilitation. The nutritional assessments were conducted at the admission. Informed consent for participation, according to procedures approved by the institutional review board of Nagoya University Graduate School of Medicine, was obtained verbally from the patients, or, for those with substantial cognitive impairment, from a surrogate (usually the closest relative or legal guardian) and from caregivers. Subjects diagnosed with infection, inflammation, liver disorders, kidney disorders, cancer at least within 2 months, or serum C-reactive protein ≧1.0 mg/dl were not included among the 262 participants to avoid the influence of inflammation on serum albumin levels.

Anthropometric measurements and biochemical markers

BMI is defined as weight in kg divided by height in meters squared. Triceps skin-fold (TSF) was measured with Harpenden calipers over the triceps muscle at the midway point between the acromion and the olecranon process. MAC and CC were measured on the left arm and calf with a tape measure. Arm muscle circumference (AMC = MAC(cm) - $\pi \times$ TSF(mm)/10) and arm muscle area (AMA) were calculated using the standard formula shown below: AMA $cm^2 = (AMC(cm^2))^2/4\pi$. Three repeat measurements were taken to the nearest 0.5 mm, with the mean taken as the true value. All anthropometric measurements were taken at least twice by two different investigators; the reported values are the means of the repeated measurements. Blood samples were collected after an overnight fast. Serum albumin and total cholesterol levels were determined using automated analyzers.

Nutritional status using SGA was conducted by trained dieticians who were blinded to the levels of serum albumin, total cholesterol, and hemoglobin. SGA consists of a brief nutritional history (weight loss during the last 6 months; dietary change; and a short physical examination of subcutaneous fat, muscle mass, and fluid balance).[5] SGA classifies patients as having PEM or moderate PEM or being well nourished; it focuses on medical issues and was constructed mostly from experience with surgical patients, but the use of SGA in older populations has also been validated.[11]

Each site's nursing staff assessed each patient's functional status which included a rating for seven basic ADL (feeding, bathing, grooming, dressing, using the toilet, walking, and transferring) using summary scores ranging from 0 (total disability) to 20 (no disability).[12] Information obtained from medical records included physician-diagnosed chronic conditions comprising the Charlson comorbidity index,[13] which represents the sum of a weighted index that takes into account the number and seriousness of preexisting comorbid conditions.

Definition of malnutrition

A BMI of less than 20 is widely accepted to indicate that the subject is underweight, particularly in well-developed countries, and 18.5 is recommended as a practical lower limit for most populations.[14] Therefore, a diagnosis of malnutrition was made when BMI was less than 18.5 kg/m^2. Serum albumin and total cholesterol levels were used as the biochemical markers of undernutrition: levels lower than 35 g/l of albumin or 3.88 mmol/l (1.5 g/l) of total cholesterol were taken to indicate malnutrition.[15,16]

Statistical analysis

The ADL score (range 0–20) was categorized into three groups with approximately equal number of participants in each group: high ADL function (ADL score ≥19), mid ADL function (ADL score 2–18), and low ADL function (ADL score <2). Differences between ADL function groups were determined by analysis of variance with a Bonferroni

correction, the χ^2 test, or the Kruskal–Wallis test, as appropriate. Partial rank correlation coefficients adjusted for age and gender were used to measure the relationships between serum albumin levels and anthropometric measurements, biochemical markers, and SGA evaluation. To examine the relationships between ADL scores and serum albumin levels, partial-rank correlation coefficients were used after adjusting for age, gender, and AMC or SGA evaluation. The sensitivity and specificity of 35 g/l of serum albumin as a cutoff point for predicting malnutrition based on the various nutritional markers were also calculated. The significance level was set at 0.05. Data evaluation was carried out using the SPSS software package (SPSS Inc., Chicago, USA).

Results

The age, ADL score, Charlson comorbidity index, anthropometric measurements, serum biochemicals (albumin and total cholesterol), and SGA assessment for total participants and groups categorized by ADL score are shown in Table 1.

The group of low ADL function had the highest comorbidity condition, lowest anthropometric measurements, and lowest levels of serum albumin and total cholesterol compared with the mid or high ADL-function group. Of the low, mid, and high ADL-function groups, 28%, 57.4%, and 87.2% were evaluated as being well nourished according to the SGA classification, respectively.

Among all participants, serum albumin levels were well correlated with various nutritional parameters including anthropometric measurements and the levels of serum total cholesterol as well as SGA classification after adjusting for age and gender (Table 2). Among high and mid ADL-function groups there was also good correlation between serum albumin levels and all nutritional makers tested except for AMA and AMC in the high ADL-function group. However, in the low ADL-function group no correlation was observed between serum albumin level and any nutritional marker tested. Among total participants after adjusting for age, gender and ADL score, serum albumin levels were correlated with BMI ($r = 0.202$, $P = 0.002$), MAC ($r = 0.213$, $P = 0.001$), TSF ($r = 0.265$, $P<0.0001$), CC ($r = 0.190$, $P = 0.003$), serum total cholesterol ($r = 0.275$, $P<0.0001$), and SGA classification ($r = 0.288$, $P<0.0001$) but not with

Table 1 ADL and nutritional characteristics.

	Total, $n = 262$		Low ADL function, ADL score ≦1, $n = 82$		Mid ADL function, ADL score = 2–18, $n = 94$		High ADL function, ADL score ≧19, $n = 86$		P
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Men/women (% of male)	86/176	32.8	29/53	35.4	25/69	26.6	32/54	37.2	0.2666*
Age	81.8	7.5	83.6	8.6	82.5	7.3	79.4	5.7	0.0006
Activities of daily living (ADL, range: 0–20)	10.2	8.7	0.2	0.4	10.3	6.1	19.8	0.4	<0.0001
Charlson index	2.1	1.8	2.6	1.5	2.5	1.9	1.3	1.5	<0.0001
Body mass index (BMI, kg/m^2)	19.7	3.9	17.4	2.8	19.5	3.4	22.2	3.9	<0.0001
Midarm circumference (MAC, cm)	22.2	3.7	20.2	3.3	21.9	3.4	24.6	3.1	<0.0001
Triceps skinfold (TSF, mm)	9.8	5.9	7.2	3.8	8.1	4.1	14.5	6.7	<0.0001
Arm muscle circumference (AMC, cm)	19.1	2.8	17.9	2.7	19.4	2.8	20.0	2.5	<0.0001
Arm muscle area (AMA, cm^2)	29.7	8.6	26.1	7.8	30.6	8.7	32.3	8.1	<0.0001
Calf circumference (CC, cm)	27.0	5.2	22.2	3.3	27.4	3.8	31.7	3.5	<0.0001
Albumin (g/l)	36.0	5.7	31.1	4.0	35.6	4.7	41.0	3.3	<0.0001
Total cholesterol (Tch, mmol/l)	4.8	1.1	4.2	0.9	4.8	1.1	5.3	0.9	<0.0001
Subjective global assessment (n, (% of total))									
Well nourished	152	(58.0)	23	(28.0)	54	(57.4)	75	(87.2)	
Moderatly malnourished	87	(33.2)	42	(51.2)	34	(36.2)	11	(12.8)	<0.0001**
Severely malnourished	23	(8.8)	17	(20.7)	6	(6.4)	0	(0.0)	

Age: high ADL vs. low ADL ($P = 0.0006$) or mid ADL ($P = 0.016$). Charlson index: high ADL vs. low ADL ($P<.0.0001$) or mid ADL ($P<.0.0001$).
BMI, MAC, CC: albumin: high ADL vs. low ADL ($P<0.0001$) or mid ADL ($P<0.0001$); mid ADL vs. low ADL ($P<0.0001$).
TSF: high ADL vs. low ADL ($P<0.0001$) or mid ADL ($P<0.0001$).
AMC; high ADL vs. low ADL ($P<0.0001$), mid ADL vs. low ADL ($P = 0.0012$).
AMA: high ADL vs. low ADL ($P<0.0001$), mid ADL vs. low ADL ($P = 0.0013$).
Tch: high ADL vs. low ADL ($P<0.0001$) or mid ADL ($P = 0.011$), mid ADL vs. low ADL ($P<0.0001$).
SD: Standard deviation.
*χ^2-test.
**Kruskal–Wallis test, others were determined by analysis of variance with a Bonferroni correction.

Table 2 Correlation between serum albumin and neutritional variables.

	Total, $n = 262$		Low ADL function, ADL score ≦1, $n = 82$		Mid ADL function, ADL score = 2–18, $n = 94$		High ADL function, ADL score ≧19, $n = 86$	
	r	P	r	P	r	P	r	P
Body mass index	0.482	<0.0001	0.135	0.2370	0.367	0.0010	0.2391	0.039
Midarm circumference	0.485	<0.0001	0.176	0.1230	0.395	<0.0001	0.2511	0.030
Triceps skinfold	0.501	<0.0001	−0.022	0.8500	0.417	<0.0001	0.3978	<0.0001
Arm muscle circumference	0.297	<0.0001	0.205	0.0710	0.285	0.0090	−0.0335	0.775
Arm muscle area	0.281	<0.0001	0.195	0.0870	0.265	0.0160	−0.0384	0.744
Calf circumference	0.636	<0.0001	0.096	0.4010	0.457	<0.0001	0.2957	0.010
Total cholesterol	0.469	<0.0001	0.194	0.0890	0.394	<0.0001	0.2525	0.029
Subjective global assessment (SGA)	0.499	<0.0001	0.199	0.0810	0.258	0.0190	0.5488	<0.0001

ADL: activities of daily living. Data were adjusted for age and gender.
SGA rating: 0, well nourished; 1, moderatly malnourished; 2, severely malnourished.

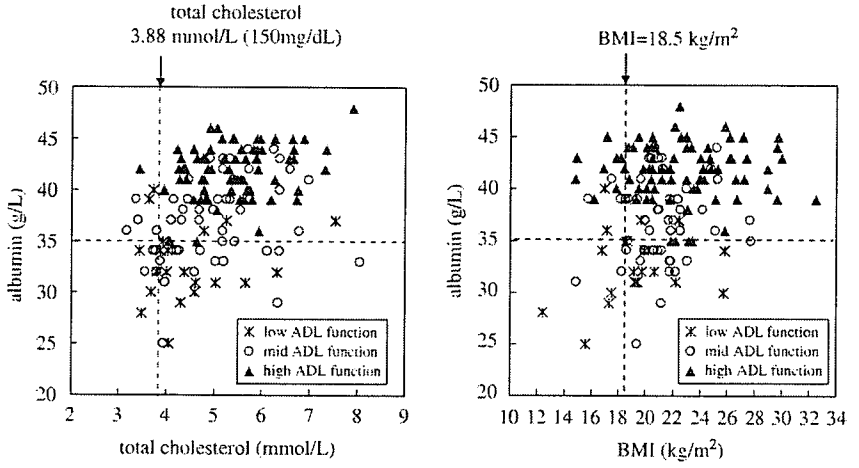


Figure 1 The relationship between levels of serum albumin and total cholesterol or BMI according to the three categories of ADL function among the well-nourished subjects as evaluated by SGA.

AMA ($r = 0.069$, $P = 0.285$) or AMC ($r = 0.086$, $P = 0.183$). Total ADL scores were well correlated with serum albumin concentration after adjusting for gender and age ($r = 0.726$, $P<0.0001$). This correlation persisted after adjusting for SGA classification ($r = 0.650$, $P<0.0001$) or AMC ($r = 0.699$, $P<0.0001$), or both ($r = 0.644$, $P<0.0001$).

Figure 1 shows the relationship between levels of serum albumin and total cholesterol or BMI according to the three categories of ADL function among the subjects evaluated as well nourished by SGA. There were no participants with albumin <35 g/l among the well-nourished high ADL-function group with total cholesterol ≧3.88 mmol/l (150 mg/dl) or BMI ≧18.5 kg/m^2. However, 13 out of 16 participants (81.3%) of the well-nourished low ADL-function group, and 13 out of 44 participants (29.5%) of well-nourished mid ADL-function group had albumin <35 g/l and total cholesterol ≧3.88 mmol/l (150 mg/dl). Furthermore, 12 out of 15 participants (80.0%) of the well-nourished low ADL-function group and 15 out of the 46 participants (32.6%) of the well-nourished mid ADL-function group had albumin <35 g/l and BMI≧18.5 kg/m^2.

In the low ADL-function group, 77.3% of the participants evaluated as being well-nourished according to SGA classification, 78.2% of the participants with serum total cholesterol concentration ≧3.88 mmol/l, and 82.1% of the participants with BMI≧18.5 kg/m^2 had a serum albumin level <35 g/l (Table 3). By contrast, among the high ADL-function group there were no participants with a serum albumin level <35 g/l among those evaluated as being well nourished. Furthermore, only 3.6% of participants with total cholesterol levels ≧3.88 mmol/l and 2.9% of participants with BMI≧18.5 kg/m^2 had serum albumin levels <35 g/l. The sensitivity and specificity of 35 g/l serum albumin as a cutoff point of malnutrition based on the various nutritional markers are presented in Table 3. Among low ADL-function participants with nutritional status based on either SGA evaluation, total cholesterol levels (<3.88 mmol/l), or BMI (<18.5 kg/m^2), the 35 g/l serum albumin cutoff point had

Table 3 Validity of cutoff point of serum albumin (<35 g/l) for malnutritional markers.

Nutritional markers		Serum albumin				P^*	Specificity	Sensitivity
		<35 g/l		≧35 g/l				
		n	%	*n*	%			
Total								
SGA	Well nourished	34	22.8	115	77.2		0.772	
	Moderatly malnourished	57	64.0	32	36.0	<0.0001		
	Severely malnourished	18	78.9	5	21.7			0.783
Tch	≧3.88 mmol/l	73	34.1	141	65.9	<0.0001	0.659	
	< 3.88 mmol/l	35	77.8	10	22.2			0.778
BMI	≧18.5 kg/m^2	45	28.7	112	71.3	<0.0001	0.713	
	< 18.5 kg/m^2	64	62.1	39	37.9			0.621
Low ADL function (*ADL socre*: ≦1)								
SGA	Well nourished	17	77.3	5	22.7	0.421	0.227	
	Moderatly malnourished	34	79.1	9	20.9			
	Severely malnourished	15	88.2	2	11.8			0.882
Tch	≧3.88 mmol/l	43	78.2	12	21.8	0.500	0.218	
	< 3.88 mmol/l	22	84.6	4	15.4			0.880
BMI	≧18.5 kg/m^2	23	82.1	5	17.9	0.787	0.179	
	< 18.5 kg/m^2	43	79.6	11	20.4			0.796
Mid ADL function (*ADL score*: 2–18)								
SGA	Well nourished	17	32.7	35	67.3	0.033	0.673	
	Moderatly malnourished	20	57.1	15	42.9			
	Severely malnourished	3	50.0	3	50.0			0.500
tch	≧3.88 mmol/l	27	36.0	48	64.0	0.003	0.640	
	< 3.88 mmol/l	13	76.5	4	23.5			0.765
BMI	≧18.5 kg/m^2	20	33.9	39	66.1	0.014	0.661	
	< 18.5 kg/m^2	20	60.6	13	39.4			0.606
High ADL function (*ADL socre*: ≧19)								
SGA	Well nourished	0	0.0	75	100.0	<0.0001	1.000	
	Moderatly malnourished	3	27.3	8	72.7			
	Severely malnourished	0		0				
tch	≧3.88 mmol/l	3	3.6	81	96.4	0.947	0.964	
	< 3.88 mmol/l	0	0.0	2	100.0			
BMI	≧18.5 kg/m^2	2	2.9	68	97.1	0.672	0.971	
	< 18.5 kg/m^2	1	6.3	15	93.8			0.063

SGA: subjective global assessment, tch: total cholesterol, BMI: body mass index, ADL: activities of daily living.
*χ^2 test.

high sensitivity (0.882, 0.880, or 0.796, respectively) but low specificity (0.227, 0.218, or 0.179, respectively) as an indicator of malnutrition. Among low ADL-function participants with nutritional status based on SGA evaluation, the 3.88 mmol/l serum total cholesterol as a cutoff point had high specificity (0.727) but low sensitivity (0.500) as an indicator of malnutrition.

Discussion

In the present study we demonstrated that the serum albumin cutoff point of 35 g/l as an indicator malnutrition is not suitable for the elderly with low ADL function. In older people with low ADL function serum albumin levels were not correlated with various nutritional parameters including anthropometric measurements, levels of serum total cholesterol, and SGA evaluation after adjusting for age and gender. Using a serum albumin level <35 g/l as a malnutrition indicator for the ADL-impaired elderly, about 80% of older people without malnutrition would be classified as malnourished (low specificity) while 11–20% of elderly persons with malnutrition would be missed (sensitivity). These results suggest that the use of a serum albumin level <35 g/l as a marker of malnutrition for elderly with low ADL function leads to over-diagnosis of malnutrition. It should be noted that we also observed that the use of a serum total cholesterol level <3.88 mmol/l as a marker of malnutrition would miss the half of the ADL-impaired elderly person with malnutrition.

The observation that serum albumin is a negative acute-phase protein suggests that serum albumin concentration could be a marker of inflammation. In fact, serum levels of albumin decrease in response to acute or chronic inflammation by altering the normal hepatic protein metabolism and inducing capillary leak.[8–10] This concept is responsible for the reports that albumin is not a good marker for the nutritional status of the hospitalized elderly with illness.[17] However, in this study we excluded patients having high C-reactive protein levels or acute illness within the past 2 months. It has been reported that serum albumin levels and SGA, two possible measurements of nutritional status in hospitalized older people, are often discordant.[18] However, this previous interesting report did not address the interaction between serum albumin and the presence of inflammation or ADL status among hospitalized older people.

It has been reported that posture affects serum albumin levels; 1 h in the sitting position after resting in the supine posture during an overnight sleep increases serum albumin by 6.3%.[19] Simply standing upright or sitting increases hydrostatic pressure, and this shift in balance between hydrostatic and oncotic pressures leads to a net movement of fluid from intravascular to interstitial spaces.[20] Most participants with low ADL function in the present study were hospitalized patients, and most of these were bed-ridden elderly. Blood specimens were drawn from low ADL-function participants lying in bed and from high ADL-function ambulatory participants in a sitting position. These postural differences may have affected the serum levels of albumin in both types of participants. However, it has been reported that there is an increase from the lying to the sitting position of about 6.5–7.7% in serum concentrations, not only of proteins but also of lipids including cholesterol.[21,22] Therefore, the posture at the collection of blood samples may not explain our results.

We have demonstrated that ADL function is well correlated with serum albumin levels. One study has demonstrated that severe disability in ADL is strongly associated with anthropometric and biohumoral parameters including serum albumin levels suggesting the presence of malnutrition.[23] However, this is not the case here, since the association between serum albumin and ADL status persists after adjusting for SGA classification, suggesting that this association is not mediated through nutritional status. It is possible that the correlation of serum albumin with ADL function may be mediated by muscle mass, since physical disability is well known to be related with muscle atrophy[24] A cross-sectional study found an association between lower serum albumin concentration and lower muscle mass in the elderly.[25] It is known that several inflammatory cytokines down-regulate serum albumin concentration and increase muscle protein breakdown, which could potentially explain the association of low serum albumin with low muscle mass.[8,26] One study has demonstrated that a low serum albumin concentration in older persons was associated with a greater loss of muscle mass during a 5-year follow-up even after adjusting for the effect of inflammation, although no association was detected between albumin levels and muscle mass at the baseline.[27] In the present study we demonstrated that albumin levels were well correlated with AMC or AMA, markers of muscle mass, among older people without acute illness and inflammation, indicating that inflammation is not involved in the correlation between serum albumin levels and muscle mass, at least in the present study. However, after adjusting for ADL levels there was no correlation between serum albumin and the markers of muscle mass. In addition, the ADL score was well correlated with serum albumin levels after adjusting for muscle mass, suggesting that serum albumin levels might be associated with muscle mass through ADL function rather than with muscle mass directly among older people without acute illness or inflammation. Previous observation has demonstrated that physical exercise increases hepatic synthesis of albumin, resulting in the elevation of plasma albumin content.[28] It is possible that physical activity may be involved in the maintenance of serum albumin concentration through an increase in hepatic synthesis of albumin. Further studies will be required to determine the exact mechanism of the correlation of serum albumin concentration and ADL impairment in well-nourished older people. Since it has been reported that lower serum albumin is independently associated with weaker muscle strength,[29] further research is needed to clarify the exact interactions among serum albumin concentration, ADL status, and not only muscle mass but also muscle strength.

There are limitations in the present study. The distribution of ADL scores of our participants was not the normal distribution. Therefore, no line could be drawn separating the older people with poorer ADL function from those with better ADL function using <35 g/l of serum albumin as the cutoff point of malnutrition. A limitation included the relative small sample size in each categorized ADL subgroup which may affect the correlation between serum albumin and other nutritional parameters. Another potential limitation of this study was the reliance on self-reported past dietary change and past weight change which are included in SGA in subjects with potential for impaired cognition. We used only anthropometric measurements, AMC and AMC, for assessment of muscle mass; upper arm muscle mass might not reflect the full range of muscle mass.

In the present study we demonstrated that impaired physical function reduced serum albumin concentration even in well-nourished older people. The use of <35 g/l serum albumin as a marker of malnutrition for the elderly with low ADL function leads to over-diagnosis of malnutrition. Although the exact mechanism of the association between low albumin concentration and disability of ADL function remains unknown, lower muscle mass or decreased physical activity may be involved in this association. Therefore, when nutritional assessment is conducted for older people with impaired ADL function, special attention should be given to the interpretation of results of anthropometric measurements and serum albumin.

Acknowledgements

We thank the dieticians and nurses for their professional assistance. This work was supported by a Grant-in Aid for the Comprehensive Research on Aging and Health from the Ministry of Health, Labor, and Welfare of Japan.

References

1. Sullivan DH, Sun S, Walls RC. Protein-energy undernutrition among elderly hospitalized patients: a prospective study. *JAMA* 1999;281:2013–9.

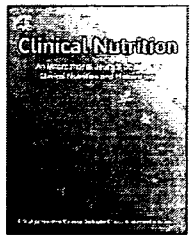
2. Edington J, Boorman J, Durrant ER, et al. Prevalence of malnutrition on admission to four hospitals in England. The Malnutrition Prevalence Group. *Clin Nutr* 2000;**19**:191–5.
3. Sullivan DH, Walls RC. Protein-energy undernutrition and the risk of mortality within six years of hospital discharge. *J Am Coll Nutr* 1998;**17**:571–8.
4. Keller HH, Ostbye T, Goy R. Nutritional risk predicts quality of life in elderly community-living Canadians. *J Gerontol A Biol Sci Med Sci* 2004;**59**:68–74.
5. Detsky AS, Baker JP, Mendelson RA, et al. Evaluating the accuracy of nutritional assessment techniques applied to hospitalized patients: methodology and comparisons. *J Parenter Enteral Nutr* 1984;**8**:153–9.
6. Omran ML, Morley JE. Assessment of protein energy malnutrition in older persons, Part II: laboratory evaluation. *Nutrition* 2000;**16**:131–40.
7. Seiler WO. Clinical pictures of malnutrition in ill elderly subjects. *Nutrition* 2001;**17**:496–8.
8. Johnson AM. Low levels of plasma proteins: malnutrition or inflammation? *Clin Chem Lab Med* 1999;**37**:91–6.
9. Doweiko JP, Nompleggi DJ. Role of albumin in human physiology and pathophysiology. *J Parenter Enteral Nutr* 1991;**15**:207–11.
10. Fuhrman MP, Charney P, Mueller CM. Hepatic proteins and nutrition assessment. *J Am Diet Assoc* 2004;**104**:1258–64.
11. Sacks GS, Dearman K, Replogle WH, et al. Use of subjective global assessment to identify nutrition-associated complications and death in geriatric long-term care facility residents. *J Am Coll Nutr* 2000;**19**:570–7.
12. Mahoney F, Barthel DW. Functional evaluation: the Barthel index. *MD State Med J* 1965;**14**:61–5.
13. Charlson ME, Pompei P, Ales KL, MacKenzie CR. A new method of classifying prognostic comorbidity in longitudinal studies: development and validation. *J Chronic Dis* 1987;**40**:373–83.
14. James WP, Francois PJ. The choice of cut-off point for distinguishing normal body weights from underweight or 'chronic energy deficiency' in adults. *Eur J Clin Nutr* 1994; **48**(Suppl 3):S179.
15. Kuzuya M, Kanda S, Koike T, et al. Evaluation of Mini-Nutritional Assessment for Japanese frail elderly. *Nutrition* 2005;**21**:498–503.
16. Kuzuya M, Kanda S, Koike T, Suzuki Y, Iguchi A. Lack of correlation between total lymphocyte count and nutritional status in the elderly. *Clin Nutr* 2005;**24**:427–32.
17. Rosenthal AJ, Sanders KM, McMurtry CT, et al. Is malnutrition overdiagnosed in older hospitalized patients? Association between the soluble interleukin-2 receptor and serum markers of malnutrition. *J Gerontol A Biol Sci Med Sci* 1998;**53**: M81–6.
18. Covinsky KE, Covinsky MH, Palmer RM, Sehgal AR. Serum albumin concentration and clinical assessments of nutritional status in hospitalized older people: different sides of different coins? *J Am Geriatr Soc* 2002;**50**:631–7.
19. Hyltoft Petersen P, Felding P, Horder M, Tryding N. Effects of posture on concentrations of serum proteins in healthy adults. Dependence on the molecular size of proteins. *Scand J Clin Lab Invest* 1980;**40**:623–8.
20. Youmans JB, Wells HS, Donley D, Miller DG, Frank H. The effect of posture (standing) on the serum protein concentration and colloid osmotic pressure of blood from the foot in relation to the formation of edema. *J Clin Invest* 1934;**13**: 447–59.
21. Miida T, Sasaki H, Sato K, et al. Postural change and within-day variation in total cholesterol and high-density lipoprotein-cholesterol levels. *(Japanese) Rinsho Byori—Jpn J Clin Pathol* 1996;**44**:860–4.
22. Felding P, Tryding N, Hyltoft Petersen P, Horder M. Effects of posture on concentrations of blood constituents in healthy adults: practical application of blood specimen collection procedures recommended by the Scandinavian Committee on Reference Values. *Scand J Clin Lab Invest* 1980;**40**:615–21.
23. Romagnoni F, Zuliani G, Bollini C, et al. Disability is associated with malnutrition in institutionalized elderly people. The I.R.A. Study. Istituto di Riposo per Anziani. *Aging (Milano)* 1999; **11**:194–9.
24. Janssen I, Heymsfield SB, Ross R. Low relative skeletal muscle mass (sarcopenia) in older persons is associated with functional impairment and physical disability. *J Am Geriatr Soc* 2002; **50**:889–96.
25. Baumgartner RN, Koehler KM, Romero L, Garry PJ. Serum albumin is associated with skeletal muscle in elderly men and women. *Am J Clin Nutr* 1996;**64**:552–8.
26. Roubenoff R, Roubenoff RA, Cannon JG, et al. Rheumatoid cachexia: cytokine-driven hypermetabolism accompanying reduced body cell mass in chronic inflammation. *J Clin Invest* 1994;**93**:2379–86.
27. Visser M, Kritchevsky SB, Newman AB, et al. Low serum albumin concentration and change in muscle mass: the health, aging, and body composition study. *Am J Clin Nutr* 2005;**82**: 531–7.
28. Yang RC, Mack GW, Wolfe RR, Nadel ER. Albumin synthesis after intense intermittent exercise in human subjects. *J Appl Physiol* 1998;**84**:584–92.
29. Schalk BW, Deeg DJ, Penninx BW, Bouter LM, Visser M. Serum albumin and muscle strength: a longitudinal study in older men and women. *J Am Geriatr Soc* 2005;**53**:1331–8.

Clinical Nutrition (2007) 26, 597–604

Available at www.sciencedirect.com

ScienceDirect

http://intl.elsevierhealth.com/journals/clnu



ORIGINAL ARTICLE

Anthropometric measurements of mid-upper arm as a mortality predictor for community-dwelling Japanese elderly: The Nagoya Longitudinal Study of Frail Elderly (NLS-FE)

Hiromi Enoki, Masafumi Kuzuya*, Yuichiro Masuda, Yoshihisa Hirakawa, Mitsunaga Iwata, Jun Hasegawa, Sachiko Izawa, Akihisa Iguchi

Department of Geriatrics, Nagoya University Graduate School of Medicine, 65 Tusruma-cho, Showa-ku, Nagoya 466-8550, Japan

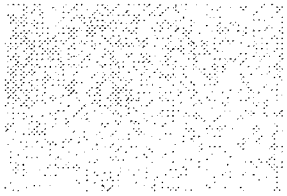
Received 16 January 2007; accepted 14 June 2007

KEYWORDS
Anthropometric measurements;
Elderly;
Malnourishment;
Mortality

Summary
Background & aims: It remains controversial whether mid-arm anthropometric measurements (MAAMs) are reflected with physical impairment or useful predictors of mortality in the frail elderly. We examined the following hypotheses: (1) MAAMs in frail community-dwelling elderly are lower than those of independent elderly, (2) the lower MAAMs are associated with physical function impairment, and (3) are independent predictors of 2-year mortality.
Methods: This study was composed of cross-sectional and prospective cohort analyses of 957 community-dwelling elderly. Data included the clients' demographic characteristics, comorbidity, activities of daily living (ADL), and MAAMs at baseline. The mean scores of MAAMs of participants were compared with Japanese Anthropometric Reference Data. Survival analysis of 2-year mortality was conducted using multivariate Cox proportional hazards models.
Results: Significantly lower arm muscle area (AMA) and higher triceps skinfold (TSF) levels were observed in most of the age groups of the study participants than those of the standard Japanese population. ADL function was correlated with AMA but not with TSF, both of which were independent risk factors for 2-year mortality in the participants (highest tertile versus lowest, AMA, HR:2.03, 95%CI:1.36–3.02; TSF, HR:1.89, 95%CI:1.30–2.75).

*Corresponding author. Tel.: +81 52 744 2364; fax: +81 52 744 2371.
E-mail address: kuzuya@med.nagoya-u.ac.jp (M. Kuzuya).

0261-5614/$ - see front matter
doi:10.1016/j.clnu.2007.06.008



Conclusions: AMA and TSF were independent risk factors for 2-year mortality in the community-dwelling frail elderly.

Introduction

Anthropometric indices of weight, height, body mass index (BMI), skinfold thickness, muscle area, and circumferences are simple, easily obtainable and inexpensive measures of assessing nutritional status. Among them, BMI has been frequently used as an indicator of nutritional status, and is well known as an important predictor of mortality and activities of daily living (ADL) decline among older people.[1,2] However, it is not uncommon that there are frail older people who cannot be weighted or measured for height. In addition, measuring height reliably in older individuals is one of the most problematic areas of anthropometry. In old age there is a decline in sitting and standing height due to vertebral compression, change in the height and shape of vertebral discs, loss of muscle tone and postural changes. When these height measurements are used in the calculation of BMI, BMI will tend to be artificially inflated. In addition, it can be difficult to measure the standing height of older people with ADL impairment.[3,4]

Anthropometric measurements of the mid-upper arm are often performed for measuring body composition because they are a quick, inexpensive, and non-invasive way of measuring nutritional status. Triceps skinfold (TSF) thickness reflects subcutaneous fat, whereas mid-upper-arm circumference (MAC) takes into account the humeral diameter as well as the skeletal muscles and fat covering the limb, therefore reflecting changes in lean body mass and fat. Mid-upper-arm muscular circumference and arm muscle area (AMA), which are derived from MAC and TSF, are also useful indicators of muscle mass.

Although these mid-arm anthropometric measurements (MAAMs) may be useful indicators of undernutrition in older adults, it remains controversial whether these measurements are useful predictors of mortality in the elderly or whether physical impairment reflects these measurements in the older population. In the general population, it has been believed that low muscle mass and high fat mass are associated with mortality.[5] Although, low AMA, an indicator of muscle mass, is associated with high mortality in the elderly,[5–7] the relationship between TSF and mortality in the elderly remains controversial.[8–10] In addition, the relation of the interaction between AMA and TSF to the mortality of the elderly remains unknown. Furthermore, information on the association of these anthropometric measurements of the mid-upper arm with physical impairment or frailty in the elderly remains unknown.

In 2001, Japanese Anthropometric Reference Data (JARD 2001) were newly established as the gold standard for nutritional assessment based on non-invasive methods.[11] It provides the anthropometric norms for healthy men and women without physical function impairment in each 5-year age-bracket, including subjects over age 65, and enables evaluations in relation to body composition.

In the present study targeting frail, community-dwelling elderly persons using the baseline and 2-year follow-up data of the Nagoya Longitudinal Study of Frail Elderly (NLS-FE), the following hypotheses were tested: (1) in frail community-dwelling elderly persons who are disabled or dependent and receiving some assistances using the long-term care insurance program, TSF and AMA levels are lower than those of not-frail, independent elderly persons living in the community; (2) these lower levels of measurements are associated with physical function impairment and comorbidity status; and (3) lower levels of TSF or AMA are independent predictors of relative short-term (2-years) mortality.

Method

Study design and subjects

The present study consisted of baseline data of the participants of the NLS-FE and their mortality during a 2-year follow-up period. Details of the participants and the NLS-FE have been published elsewhere.[12,13]

The study population of NLS-FE consisted of 1875 (men: 632, women: 1243). Community-dwelling elderly (aged 65 years or older) eligible for the LTCI who lived in Nagoya city (Central Japan) and were provided various home care services from the Nagoya City Health Care Service Foundation for Older People, which is comprised of 17 visiting nursing stations accompanying care-managing centers. The LTCI system covers care for both the elderly aged 65 and older. Under the LTCI program, care levels (levels 0–5) are determined according to eligibility criteria. The elderly in the community who are eligible for LTCI are disabled and chronically ill, have physical and mental problems, and easy to admit acute hospital or institute care setting.[14] NLS-FE participants enrolled between 1 December 2003 and 31 January 2004 underwent comprehensive in-home assessments by trained nurses at baseline and 6, 12, and 24 months. At 3-month intervals, data were collected about any events the participants experienced, including admission to the hospital, nursing home admission, and mortality. Death information was obtained from event reports at 3-month intervals. Written informed consent for participation, according to procedures approved by the Institutional Review Board of the Nagoya University Graduate School of Medicine, was obtained from the patients, or, for those with substantial cognitive impairment, from a surrogate (usually the closest relative or legal guardian), and from family member caregivers.

Data collection

The data were collected at the clients' homes from standardized interviews with patients or surrogates, from

caregivers, and from care-managing center records by trained nurses. The data included clients' demographic characteristics and a rating for 10 basic ADL (feeding, mobility on bed, bathing, grooming, dressing, using the toilet, walking inside and outside, transferring, and using stairs) using summary scores ranging from 0 (total disability) to 20 (no disability). Information obtained from care-managing center records included the following physician-diagnosed chronic conditions: ischemic heart disease, congestive heart failure, liver diseases, cerebrovascular disease, diabetes mellitus, dementia, chronic obstructive pulmonary disease, neoplasia, hypertension, and diseases comprising the Charlson comorbidity index,[15] which represents the sum of a weighted index that takes into account the number and seriousness of preexisting comorbid conditions.

Anthropometry

Among 1875 participants, a total 957 (men, 355, women, 602) using visiting nurse services were enrolled for the anthropometric measurements. Anthropometric measurements were conducted by trained nurses at the clients' home. Measurement of TSF (to the nearest 2 mm) was made using caliper and MAC (to the nearest 0.1 cm) using a flexible measuring tape, on the right side of the participant's body unless affected by disability or disease. These measurements were taken at least twice by one trained nurse according to the instruction sheet, and reported values were the means of the repeated measurements. Arm muscle circumference (AMC = MAC (cm)$-\pi \times$ TSF (mm)/10) and AMA were calculated using a standard formula[16] shown below: AMA $cm^2 = (AMC\ (cm))^2/4\pi$. BMI was defined as weight in kg divided by height squared.

The mean scores of anthropometric measurements of patients grouped by age and gender were compared with the JARD 2001. In JARD 2001, a mean value, a central value, standard deviation, maximum value, minimum value, and percentile (5th, 10th, 25th, 75th, 90th, 95th) were determined according to the age division.[11] Therefore, it was possible to compare the obtained measurement values and these reference values.

Statistical analysis

This analysis was conducted using a total of 957 subjects (men: 355; women: 602) extracted from the NLS-FE data set. The difference between male and female were assessed

Table 1 Demographic characteristics of patients.

Variables	Categories	N (%), average $\pm$ SD		p-Value
		Male ($N = 355$)	Female ($N = 602$)	
Age		78.50 $\pm$ 7.49	81.57 $\pm$ 7.97	<0.001
	65–69	47 (13.2)	46 (7.6)	<0.001
	70–74	63 (17.7)	82 (13.6)	
	75–79	100 (28.2)	119 (19.8)	
	80–84	58 (16.3)	124 (20.6)	
	85+	87 (24.5)	231 (38.4)	
Nutrition	Peroral	322 (90.7)	554 (92.0)	0.622
	Enteral feeding	32 (9.0)	45 (7.5)	
	Parenteral nutrition	1 (0.3)	3 (0.5)	
Basic ADL (0–20)		11.0 $\pm$ 6.5	9.9 $\pm$ 7.1	0.013
Charlson comorbidity index (0–35)		2.5 $\pm$ 1.6	2.2 $\pm$ 1.6	0.019
Illness	Ischemic heart disease	31 (8.7)	64 (10.6)	0.496
	Congestive heart failure	32 (9.0)	60 (10.0)	0.818
	Liver disease	13 (3.7)	20 (3.3)	0.714
	Cerebrovascular disease	147 (41.4)	187 (31.1)	<0.001
	Diabetes	38 (10.7)	68 (11.3)	0.914
	Dementia	92 (25.9)	221 (36.7)	0.001
	Chronic pulmonary disease	38 (10.7)	42 (7.0)	0.037
	Neoplasia	37 (10.4)	51 (8.5)	0.242
	Hypertension	70 (19.7)	150 (24.9)	0.068
Anthropometric measurements	Body mass index (kg/m^2)	20.8 $\pm$ 3.4 ($n = 219$)	20.8 $\pm$ 4.4 ($n = 301$)	0.978
	Mid-arm circumference (cm)	24.3 $\pm$ 4.1	23.1 $\pm$ 4.5	<0.001
	Triceps skinfold thickness (mm)	14.3 $\pm$ 9.4	15.5 $\pm$ 9.5	0.200
	Arm muscle area (cm^2)	32.4 $\pm$ 11.6	28.0 $\pm$ 11.5	<0.001

Statistical analysis: Unpaired t-test (age, basic ADL, Charlson comorbidity index and anthrometric measurements), and χ^2-test (age group, nutrition route and illness).