

Nutritional and cognitive relationships and long-term mortality in patients with various dementia disorders

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Abstract

Background: subjects with dementia are at risk for protein-energy malnutrition.

Objective: to study the nutritional status, the short-term effects of adapted nutritional routines and the long-term mortality in subjects admitted for evaluation of cognitive dysfunction.

Design: prospective observational study.

Setting: University Hospital.

Subjects: a total of 231 patients (80 ± 7 years, 65% women).

Methods: Body mass index (BMI, kg/m^2), serum concentrations of albumin, ferritin, vitamin B₁₂, folic acid and haemoglobin as well as Mini-Mental State Examination (MMSE, 0–30 p) results and co-morbidity were recorded at hospital admittance and before discharge. Seven years later, mortality was registered.

Results: mean BMI was in the normal range (23.3 ± 4) as were the biochemical indices, and they did not vary among patients with Alzheimer's disease (AD), vascular dementia (VaD), mild cognitive impairment, mixed dementia and other diagnoses. A BMI of <23 was found in 108 (52%) subjects. Weight and MMSE score correlated weakly ($r=0.18$, $P<0.01$) at inclusion. During a median hospital stay of 3 weeks, an average weight gain of 0.5 ± 1.8 kg ($P<0.001$) and an increase in MMSE score of 0.9 ± 3 ($P<0.001$) was observed. However, these changes did not correlate. A BMI of <23 was associated with an increased risk for 7-year mortality (OR 3, 95% CI=1.3–6.7), which was independent of age, male gender, dementia diagnosis and co-morbidity.

Conclusions: nutritional status did not vary in patients with various dementia diagnoses. A BMI of <23 was related to reduced 7-year survival, but this result was independent of co-morbidity, male gender and age.

Keywords: Alzheimer's disease, cognition, dementia, nutrition, survival, elderly

Introduction

Chronic disease is often associated with protein-energy malnutrition (PEM) [1, 2]. Cross-sectional studies show that demented older people have a lower body mass index (BMI) compared with cognitively intact older people [3, 4]. Up to 50% of institutionalised demented patients are assumed to suffer from malnutrition [5]. Subjects with Alzheimer's disease (AD) have been identified as being at particularly high risk for PEM [6–8]. Within 8 years of onset of AD, 50% of patients need help with feeding or artificial nutrition [9].

Around 8% of older people between 75 and 85 years of age are assumed to suffer from dementia [10]. When seeking means to improve quality of life for older people it is important to understand the relationships between nutrition and cognition [11]. Previous studies on nutrition and dementia have focused on AD. Knowledge of the nutritional status in other dementia conditions—for example, vascular dementia, mixed dementia and mild cognitive impairment (MCI)—is sparse.

The present study was designed to characterize the relationship between nutritional status and cognitive function

in older subjects hospitalised for assessment of cognitive dysfunction. Furthermore, we studied the effects of optimised nutritional routines on weight and cognition during a short hospital stay, and finally we assessed the long-term mortality rate in relation to dementia diagnoses and nutritional status.

Methods

Subjects

A total of 231 subjects (80 ± 7 years, 65% women) were included, all of whom had been consecutively admitted for in-ward investigation of cognitive function between October 1994 and June 1996. The patients were referred from the greater south-west Stockholm area. The majority were referred to the clinic by their general practitioner and considered not capable of being examined as outpatients. The ward had 14 beds and staff that were specially trained to take care of people with dementia. The patients were examined at the time of admission to the ward and again at discharge, i.e. a median of 21 days (17–25 days, 25th–75th percentile) later. Co-morbidity was categorised into nine diagnostic groups and assessed as mild or severe.

Assessment of nutritional and cognitive status

Body weight was measured with patients wearing light clothing at admittance and at discharge. Height was measured to the nearest 0.5 cm in a standing position, using a horizontal headboard. The care staff of the ward were trained to perform the measurements. BMI (weight (kg)/height (m)²) was calculated. Serum concentrations of albumin, ferritin, vitamin B₁₂, folic acid and haemoglobin (Hb) were analysed as biochemical indicators of nutritional status

using the routine methods and reference ranges (Table 1) of the Laboratory of Clinical Chemistry at the hospital. Albumin was analysed with a bromocresol purple technique (Boehringer Mannheim Corporation, Indianapolis, IN, USA), and BM Tina-quant® Ferritin BM/Hitachi 917 (Boehringer Mannheim GmbH, Germany) was used for ferritin analyses. Haemoglobin was analysed using HB Advia 120® (Bayer, Germany), and vitamin B₁₂ and folic acid with the use of AutoDEL-FIA™ (Dissociation-Enhanced Lanthanide FluoroImmunoAssay; Wallac Oy, Finland).

The patients underwent a complete dementia assessment. In this study, the Mini-Mental State Examination (MMSE) score was used as an indicator of cognitive function [12]. The MMSE assessments were performed by the staff physician at the ward. The diagnoses were confirmed by the responsible ward physician according to DSM-IV criteria (American Psychiatric Association, 1994). The patients were classified as having Alzheimer's disease (AD), vascular dementia (VaD), mild cognitive impairment (MCI), mixed dementia or other diagnoses. Mixed dementia was assigned to individuals with both AD and VaD (Table 1). Age at disease onset was estimated through proxy reports.

Nutritional ward regime

All patients had breakfast between 8 and 10 am, lunch at 12 pm, supper at 5 pm, afternoon coffee at 2 pm and an evening meal at 7 pm in a home-like dining room. The food was served on china and the patients served themselves when possible. The patients were offered whole-fat dairy products such as whole-fat milk and spread, and they received cream-fortified desserts at lunch and supper. Homemade buns and pastries were served in the afternoon. Low-calorie products were not used.

Table 1. Demographic, anthropometric and biochemical variables at the start of the study in 231 older subjects undergoing examination of cognitive function

	All	AD	VaD	MCI	Others	Mixed	Ref values	P ^a
<i>n</i> (%)	231	93 (41)	33 (14)	38 (17)	47 (20)	19 (8)		
Age (years)	80 ± 6.8	80.5 ± 7.1	80.5 ± 6.8	78 ± 7.6	79 ± 6	82 ± 4.4		
Female (<i>n</i>) (%)	151 (65)	67 (72)	20 (61)	24 (63)	26 (55)	14 (74)		
MMSE, 0–30 p	16.6 ± 6	14.8 ± 5.7	16.4 ± 4.7	22.8 ± 4.8	16.4 ± 5.6	13.7 ± 4.2		<0.001 ^b
Days in ward (median, 25th–75th percentile, <i>n</i> = 226)	21 (17–25)	21 (17–25.5)	21 (16–23)	18.5 (15–24)	21 (17–24)	21 (20–30)		
Age at disease onset (years) (<i>n</i> = 208)	77 ± 7.5	76 ± 8.2	77.5 ± 6.8	78.3 ± 7.4	76.5 ± 7.5	80.1 ± 3.6		
Duration of disease (years) (median 25th–75th percentile, <i>n</i> = 208)	2 (1–3)	2 (1–4)	2 (1–3)	1 (1–3)	2 (1–3)	2 (1–5)		
Weight (kg) (<i>n</i> = 228)	61.6 ± 12.3	60.7 ± 12.5	60.6 ± 13.9	62.2 ± 10.6	65.2 ± 12.3	57.1 ± 10.4		
BMI (kg/m ²) (<i>n</i> = 208)	23.3 ± 4.1	23 ± 4.4	23.6 ± 4.3	23.4 ± 3.6	23.8 ± 4.2	22.5 ± 3.7		
BMI <23, <i>n</i> (%) (<i>n</i> = 208)	108 (52)	43 (51)	17 (55)	18 (50)	18 (47)	12 (67)		
Serum albumin (g/l) (<i>n</i> = 216)	35.5 ± 4.6	35.8 ± 4.7	34.5 ± 3.6	36.3 ± 4.6	35.4 ± 5.2	34.6 ± 3.7	35–46	
Serum ferritin (mg/l) (<i>n</i> = 131)	109 ± 112	114 ± 136	101 ± 95	80 ± 55.5	135 ± 115	113 ± 105	10–130	
Serum B ₁₂ (pmol/l) (<i>n</i> = 212)	384 ± 398	356 ± 337	427 ± 495	386 ± 402	412 ± 439	373 ± 375	110–600	
Serum folic acid (nmol/l) (<i>n</i> = 195)	14.7 ± 14.7	15.3 ± 15.9	10.3 ± 5.6	15.7 ± 16.5	14.3 ± 9.2	18.9 ± 24.7	>4	
Serum Hb (g/l) (<i>n</i> = 215)	129 ± 15	130 ± 14.5	129 ± 15	130 ± 14.4	128 ± 19.5	129 ± 10.5	115–145	

Mean ± SD, median (25th–75th percentile) or *n* (%).

AD = Alzheimer's disease; VaD = vascular dementia; MCI = mild cognitive impairment; Others = other diseases: dementia not otherwise specified (*n* = 29), depression, confusion or psychiatric disease (*n* = 4) Parkinson-dementia (*n* = 2), normal pressure hydrocephalus (NPH) (*n* = 5), frontotemporal dementia (*n* = 4) and examination of cognition (*n* = 1); mixed = dementia with both AD and VaD components; MMSE = Mini-Mental State Examination; BMI = body mass index.

^aDifferences between the groups at start.

^bANOVA.

Mortality follow-up

Mortality data were obtained from Swedish population records 7 years after inclusion.

Statistics and ethics

Data are presented as mean \pm SD or median (25th–75th percentile). To analyse variations between the diagnostic groups, one-way ANOVA, Kruskal–Wallis one-way analysis of variance by ranks and chi-squared tests were used in accordance with the type and distribution of the variables. For multiple comparisons, the Fisher least significant difference (LSD) post hoc test was used. Possible changes at follow-up within groups were evaluated using Student's paired *t* test or the Wilcoxon sign rank test. For correlation analyses, Pearson and Spearman correlation coefficients were calculated depending on type and distribution of the variables. Multiple regression analyses were performed in order to evaluate possible independent relationships between the variables. Logistic regression was used to evaluate the prognostic value of BMI, age, gender, dementia diagnosis, MMSE, co-morbidity, serum levels of albumin, vitamin B₁₂, Hb and folic acid for 7-year mortality. For the logistic regression analyses, co-morbidity was dichotomised, i.e. no/mild and severe co-morbidity. The co-morbidities combined as well as the separate co-morbidities were subsequently entered into various logistic regression models. The biochemical markers were included either as continuous variables or dichotomised according to the lower reference cut-off value. MMSE was included either as a continuous variable or dichotomised according to the median of 17. For survival analysis we used Kaplan–Meier and log rank tests. The Statistica® program package (Statsoft, OK, USA) was used for the statistical calculations.

The study was reviewed and approved by the local ethics committee and conformed to the Helsinki declaration.

Results

Diagnoses and basic characteristics

The distribution of the dementia diagnoses is presented in Table 1. AD was diagnosed in four out of 10 patients. Fifty per cent of the patients had MMSE scores of <17 points and nine out of 10 scored <24 points. Mean MMSE scores for the various diagnostic groups varied from 13.7 to 16.4, except for patients diagnosed as MCI (Table 1). About half of the patients (52%), married or living alone, managed their day-to-day lives without any help from the community.

Co-morbidities were found in 81% of the patients: cardiovascular disorders ($n=102$), hypertension ($n=16$) and diabetes mellitus ($n=7$) without manifest atherosclerotic disease, pulmonary disorders ($n=8$), malignant disease ($n=6$), neurological diseases ($n=9$), more than one main diagnosis ($n=19$) and other diagnoses ($n=20$) such as hyperparathyroidism, renal failure, vitamin B₁₂ deficiency and aortic stenosis. Forty-three patients did not display any ongoing co-morbidity. Co-morbidities varied among the dementia diagnoses groups ($P<0.01$) (data not shown). As expected, cardiovascular disease was common, i.e. present

in 70% of the subjects diagnosed as VaD. Among the AD patients, one-third displayed cardiovascular disease, whereas almost one-third did not have any other ongoing disease.

Nutritional status

Table 1 shows the nutritional status at start in the whole group and in the five diagnostic groups. The BMI did not vary with dementia diagnosis, nor did BMI vary with no/mild (23.3 ± 4.2) or severe (23.3 ± 4.1) co-morbidities. A BMI of <23 was found in half of all subjects (Table 1). Two-thirds of the mixed group patients had a BMI of <23 . The mean vitamin B₁₂, folic acid, Hb and ferritin values were within the normal range in all groups. All but 15 subjects (7%) had vitamin B₁₂ values above the lower reference limit, i.e. 110 pmol/l, whereas 32 subjects (15%) had <150 pmol/l. Twenty-nine individuals (13%) had Hb values below the lower reference range. The mean serum albumin was in the lower reference range and altogether 88 patients (41%) had values <35 g/l. There were no differences between the diagnostic groups according to biochemical nutritional indices (Table 1).

Weight was found to correlate weakly with MMSE ($r=0.18$, $P<0.01$), Hb ($r=0.25$, $P<0.001$), albumin ($r=0.14$, $P<0.05$) and inversely with age ($r=-0.26$, $P<0.001$). MMSE correlated inversely with duration of disease ($rho=-0.16$, $P<0.05$). Multivariate regression analyses showed that the correlation between MMSE and weight was independent of duration of the disease (data not shown).

Follow-up at discharge

During the average hospital stay of 21 days, weight across the whole group of patients increased slightly (Table 2). The weight gain in AD and VaD patients differed from the others. Seventy subjects with a BMI of <22 at admission increased on average by 0.9 ± 2 kg in weight, which differed significantly ($P=0.019$) from the individuals with a BMI of ≥ 22 at the start who were weight stable. The increase in MMSE that was noted in the whole group did not correlate with the change in weight ($r=-0.08$). Vitamin B₁₂ in serum increased across the whole group and particularly in the MCI group. Subjects with serum values of vitamin B₁₂ <110 pmol/l at admission were supplemented.

Mortality follow-up after 7 years

During the 7-year observation period, 191 patients (83%) died with a median survival of 3 years and 7 months. There were no differences in mortality rate between the various dementia groups, except that patients diagnosed as suffering from 'other dementia disorders' tended to have higher mortality (89%, $P=0.15$). Furthermore, mortality did not differ between individuals with MMSE ≥ 17 or MMSE <17 points at the start of the study (data not shown).

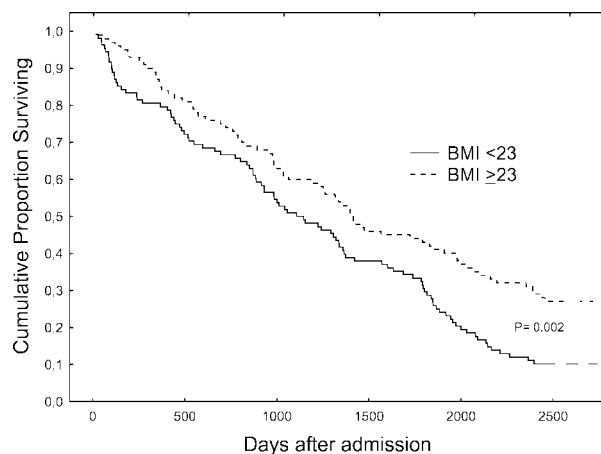
Weight gain during the hospital period tended to predict a better 7-year survival (OR 1.9, 95% CI=0.83–4.5, log rank test $P=0.08$). As expected, those with severe co-morbidities had significantly shorter survival than patients with no or mild co-morbidity ($P<0.0001$). Patients with a BMI of <23 showed higher ($P=0.002$) long-term mortality in survival analysis (Figure 1). After 7 years, a quarter of the individuals

Table 2. Changes in weight, BMI, MMSE, albumin, vitamin B₁₂ and ferritin during the hospital stay (median 21 days) in all patients and in the various diagnostic groups

	All	AD	VaD	MCI	Others	Mixed	P ^d
Weight (kg) (<i>n</i> = 185)	0.5 ± 1.8 ^c	0.7 ± 2.05 ^b	0.7 ± 1.4 ^a	0.2 ± 1.6	0.1 ± 2	0.4 ± 1.4	0.007
BMI (kg/m ²) (<i>n</i> = 157)	0.2 ± 0.6 ^c	0.2 ± 0.6 ^b	0.3 ± 0.5 ^a	0.1 ± 0.6	0.1 ± 0.7	0.2 ± 0.6	
MMSE (0–30 p) (<i>n</i> = 188)	0.9 ± 3 ^c	0.7 ± 3.2	0.7 ± 2.96	1.3 ± 2.4 ^b	0.7 ± 3.1	1.5 ± 2.7 ^a	<0.001
Serum albumin (g/l) (<i>n</i> = 128)	0.4 ± 1.8	−0.1 ± 3.4	1.4 ± 3.3	−0.07 ± 3.9	0.8 ± 5	0.7 ± 2.6	
Serum vitamin B ₁₂ (pmol/l) median (25th–75th percentile), (<i>n</i> = 117)	1 (−33–50) ^a	−7.5 (−46–49)	0 (−30–41)	37 (0.5–6.5) ^a	−5 (−64–14)	−4 (−28–200)	0.018
Serum ferritin (μg/l) (<i>n</i> = 106)	−6.1 ± 1.9 ^c	−15.5 ± 37.2 ^c	−12.6 ± 32.2	−2.1 ± 25	17.7 ± 79.5	−4.9 ± 26.8	

Medians (vitamin B12) are given due to strongly skewed values.

^a*P* < 0.05; ^b*P* < 0.01; ^c*P* < 0.001, significance levels in changes over time within the various diagnostic groups; ^dsignificance levels in changes over time between the groups.

**Figure 1.** Seven-year survival in individuals with various cognitive disorders and BMI <23 and ≥23, respectively (Kaplan–Meier curves and log rank test).

with a BMI of ≥23 were still alive, while the corresponding figure was 10% in the leaner subjects with a BMI of <23. No other BMI cut-off value was able to identify a group with elevated mortality (data not shown). In logistic regression analyses, a BMI of <23 remained as a predictor of 7-year mortality (OR 3, 95% CI=1.3–6.7) even after adjustment for age, male gender and severe co-morbidity, the latter irrespective of whether it was included in the model as co-morbidity in general or as cardiovascular co-morbidity.

Discussion

In this group of patients admitted to hospital for in-ward evaluation of cognitive dysfunction, a correlation was found between weight and MMSE. There were no differences in weight or BMI among the various diagnostic groups at the start. After an average hospital stay of 3 weeks there were slight increases in weight as well as in MMSE score, improvements that did not correlate. At a 7-year follow-up, a BMI ≥23 was found to be associated with an increased survival rate, independent of age, male gender and co-morbidity. Co-morbidity was found to predict length of survival, but type, severity or duration of dementia disorder did not.

The patients had a mean BMI of around 23 kg/m². Considering that BMI was about 26 in a reference population of Swedish 80-year-old subjects [13], the current patients appeared to be underweight. To identify older patients at risk for being underweight, a cut-off of BMI ≤23 has been suggested [14]. Half of the subjects in the current study had a BMI below this level. Serum levels of albumin, Hb, ferritin, vitamin B₁₂ and folic acid were in the normal range, which indicated that there was no evidence of severe malnutrition or of acute illness in the present group of patients. The correlation between MMSE and weight that was found is in line with a recent report [15]. Possible reasons for weight loss in demented patients may be inadequate dietary intake, increased energy expenditure and dementia-related metabolic disturbances. Some previous studies agree with the finding of no difference in BMI among the dementia diagnosis groups [5, 16]. One study has reported lower weight in patients with AD than in patients with VaD [17]. It is also reported that weight loss increases as the dementia progresses, especially in AD [5, 9, 16].

When changes during the hospital stay were recorded, we found that the gain in weight was most evident in the patients diagnosed as suffering from AD and VaD. This finding is in contrast to a general perception that weight loss is inevitably linked to dementia disorders. Furthermore, the subjects with a BMI of <22 at start gained more weight than those with a BMI of ≥22, indicating that the patients who were underweight at admission had the best potential for responding to the adapted nutritional routines. Nutritional supplements together with particular care during meals are reported to improve food intake [18] as well as increase weight in demented patients at nutritional risk [19–22], and to reduce morbidity and mortality [23]. Interestingly, we noticed that weight gain during the short hospital stay identified a group of patients who tended to have better long-term survival.

The majority of nutritional intervention studies focus either on cognition or nutrition as outcome variables. The question of whether cognitive effects may be achieved from nutritional treatment in weight-losing patients with dementia disorders is not resolved. In the present study, with a short observation period of 3 weeks, we found no correlation between change in weight and change in MMSE. The increase in MMSE was likely to be a combined effect of

adapted meal routines with fortified food, a care-giving environment and social interaction. In a study of severely demented residents in units of group-living for the demented, a significant weight gain was noticed after 5 months of oral supplementation. Partly supporting the present data, no positive effects on cognition were found [24].

Finally, long-term mortality in relation to BMI and dementia diagnoses was evaluated. Logistic regression analyses revealed a BMI of <23 to predict increased risk for 7-year mortality independent of age, gender and co-morbidity. The Kaplan–Meier curves indicate that there was already an increased mortality in the lean subjects in the first few months after admission. Thereafter, a slow gradual increase in mortality among the lean subjects was observed. In line with our findings, a 3-year follow-up study of non-hospitalized AD and VaD patients showed that a low BMI predicted death [25]. Our finding is also in agreement with data from groups of geriatric patients with other types of chronic disease [26, 27].

Median survival was 3.6 years, which is in line with other reports [28, 29]. In contrast to a previous study [30], the degree of cognitive impairment, evaluated by MMSE, was not associated with long-term mortality in the present study. Moreover, except for a tendency in the patients who suffered from ‘other dementia diseases’, the type of dementia diagnosis did not have a significant impact on mortality. Cardiovascular co-morbidities were abundant among the patients, and co-morbidities in general as well as cardiovascular co-morbidity in particular predicted mortality. High age and the presence of severe co-morbidities most probably were stronger determinants of mortality than the dementia disease in itself.

In conclusion, a BMI of <23 was found in half of patients with cognitive dysfunction undergoing a diagnostic examination. A low BMI was an independent risk factor for increased mortality. Whether nutritional support in weight-losing patients with dementia disorders is beneficial needs to be investigated further.

Key points

- At the time of diagnostic examination, there were no differences in nutritional status in patient groups with various dementia diagnoses.
- Weight gain and improved cognitive status during a short hospital stay did not correlate.
- A BMI of <23 was related to reduced long-term survival in patients with various dementia diagnoses, whereas the diagnoses were not.

Funding sources

This study was supported by the Swedish Research Council, and funds from the Karolinska Institute and Stockholm City Council.

Acknowledgements

We thank the staff at ward B 79, Karolinska University Hospital, Huddinge, as well as Majken Hansson and Katharina

Wahlund for their assistance and committed involvement in the study. This study was supported by the Swedish Research Council, and funds from the Karolinska Institute and Stockholm City Council. None of the authors have any conflict of interest.

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Received 8 April 2004; accepted in revised form 10 November 2004

Age and Ageing 2005; **34**: 141–147
doi:10.1093/ageing/afi050

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Muscle function and functional ability improves more in community-dwelling older women with a mixed-strength training programme

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Abstract

Background: supervised training can reach a limited number of elderly people.

Objective: to determine the impact of a 1-year mixed-strength training programme on muscle function (MF), functional ability (FA) and physical activity (PA).

Setting: twice-a-week hospital-based exercise classes and a once-a-week home session.

Participants: twenty-eight healthy community-dwelling men and women on the training programme and 20 controls aged over 75 years.

Methods: training with two multi-gym machines for the lower limbs at 60% of the repetition maximum (1RM). At-home subjects used elastic bands.