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Body mass index levels and changes before and after dementia diagnosis and risk of all-cause mortality: a nationwide cohort study

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Abstract

Background Dementia is a progressive disease associated with various health risks, including increased mortality. Although weight changes have been linked to adverse outcomes in older adults, limited research has examined the impact of body weight status after dementia diagnosis and its changes before and after diagnosis on mortality. This study aimed to address these gaps.

Methods Using nationwide data from the Korean National Health Insurance Service, we included 37,717 individuals newly diagnosed with dementia between 2010 and 2016, comprising 29,982 with Alzheimer's disease (AD) and 3,220 with vascular dementia (VaD). Participants were followed up until the end of 2019. Multivariable Cox proportional hazards regression models were used to estimate adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) for all-cause mortality based on body mass index (BMI) status after diagnosis and BMI changes before and after diagnosis.

Results During a mean follow-up of 4.1 years, 21.7% of individuals with all-cause dementia died. Compared to those with normal BMI (18.5–22.9 kg/m^2), underweight individuals (< 18.5 kg/m^2) after diagnosis had a higher risk of all-cause mortality (HR 1.57, 95% CI 1.46–1.69). Excessive weight loss or gain was associated with the highest mortality risk—for example, weight loss from obesity to underweight yielded the highest risk (HR 2.09, 95% CI 1.26–3.46). Conversely, maintaining obesity (≥ 25 kg/m^2) (HR 0.69, 95% CI 0.65–0.74) or modest weight gain from normal or overweight to obesity (HR 0.84, 95% CI 0.76–0.93) was associated with lower mortality risk. These associations remained consistent across dementia subtypes. Subgroup analyses revealed stronger associations in middle-aged individuals and in women with all-cause dementia.

Conclusions This large-scale cohort study suggests that excessive weight loss is significantly associated with the risk of all-cause mortality in individuals with dementia, while excessive weight gain may also be a marker of increased risk. Maintaining stable weight or modest weight gain is associated with lower mortality risk. These findings highlight the importance of monitoring body weight in dementia patients, especially during the critical period following diagnosis, to reduce mortality risk.

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Introduction

Dementia is a progressive neurodegenerative disease that impairs memory, thinking, and judgement. The disease significantly interferes with daily life, especially in its severe stage. Approximately 36 million people are currently living with dementia worldwide, and this number is projected to triple by 2050 due to the rapid aging of populations [1]. Alzheimer's disease (AD) is the most common type of dementia, followed by vascular dementia (VaD) [2]. Each type has distinct risk factors and pathophysiological mechanisms [3]. Moreover, individuals with dementia have a shorter life expectancy than those without [4, 5]. Although there are several anti-dementia medications, no treatments exist that can cure dementia.

Weight loss, often caused by poor nutritional intake and increased energy expenditure, is associated with declines in cognitive function, physical functioning, and quality of life [6], as well as increased risk of mortality and hospitalization in older adults [7]. Weight loss is particularly common among individuals with AD [8] and has been linked to higher risks of mortality [9] and hospitalization [9, 10]. In contrast, among those with VaD, weight loss has not been consistently associated with increased mortality risk [9]. Conversely, weight gain, often resulting from sedentary lifestyle [11], may lead to obesity [12]. However, some studies have found no significant association between weight gain and the risk of all-cause dementia, including AD and VaD [13].

Previous studies have identified associations between weight change and all-cause mortality in individuals with dementia [9, 13]. However, few studies have directly compared the risks associated with weight gain and weight loss, particularly in relation to mortality. Unlike one previous study [14], there remains a lack of research investigating how weight change, both before and after dementia diagnosis, is related to all-cause mortality in patients with dementia.

Given these inconsistent findings, studies exploring the associations between body mass index (BMI) after dementia diagnosis and changes in BMI and all-cause mortality among individuals with dementia remain limited. Therefore, this study aimed to investigate these associations using nationally representative data of Korean patients with dementia.

Methods

Data source and study population

In this cohort study, we used the data provided by the Korean National Health Insurance Service (NHIS), a mandatory single-payer insurance system covering approximately 97% of the South Korean population. The NHIS offers a biennial national health-screening program for all Korean adults aged 40 years or older and all employees. This database includes extensive health information, health screening results, medical diagnosis, and prescription data based on the International Classification of Diseases, 10th Revision (ICD-10) codes [15].

From this database, we identified 302,906 individuals who were newly diagnosed with dementia between January 1, 2010, and December 31, 2016, representing a 50% random sample of eligible dementia cases provided by NHIS. Dementia was defined as the prescription of anti-dementia medications (donepezil, rivastigmine, memantine, or galantamine) at least twice per year, along with relevant ICD-10 codes (F00 or G30 for AD; F01 for VaD; and F02, F03, G23.1, or G31 for other types of dementia). This information was confirmed based on records from outpatient visits or inpatient admissions. In Korea, anti-dementia medications are reimbursed under the NHIS for patients with a Mini-Mental State Examination score ≤ 26 and either a Clinical Dementia Rating of 1–3 or a Global Deterioration Scale of 3–7, as in previous studies [16, 17]. Among these, we identified 39,190 individuals aged ≥ 40 years who underwent NHIS health screening within 2 years both before and after their dementia diagnosis. Individuals with missing data (n = 276) and those who died within the first year of follow-up (1-year lag period) (n = 1,197) were excluded. Ultimately, 37,717 individuals with dementia (12,985 men and 24,732 women) were included in the analyses (Supplementary Fig. 1).

The study was conducted in accordance with the principles of the Declaration of Helsinki. This study was approved by the Institutional Review Board of the Korea University Guro Hospital, Seoul, Korea (No. 2022GR0325). The requirement for written informed consent was waived due to the use of deidentified and retrospective data.

Changes in BMI

The NHIS health-screening program includes lifestyle surveys and health examinations, including anthropometric and laboratory measurements, which were included at each health screening. Height and weight were measured, and BMI was calculated as weight divided by height squared (kg/m^2). Participants were classified into five BMI categories after their dementia diagnosis according to the Asia-Pacific BMI classification: underweight (< 18.5 kg/m^2), normal weight (18.5–22.9 kg/m^2), overweight (23.0–24.9 kg/m^2), obesity class Ⅰ (25.0–29.9 kg/m^2), and obesity class Ⅱ–III (≥ 30.0 kg/

m^2) [18]. Then, based on BMI measured within two years before and after the dementia diagnosis, participants were categorized into three groups: underweight (< 18.5 kg/m^2, U), normal to overweight (18.5–24.9 kg/m^2, N), and obesity (≥ 25.0 kg/m^2, O). Using these categories, we identified nine patterns of BMI change: (1) underweight → underweight (no changes); (2) underweight → normal or overweight; (3) underweight → obesity; (4) normal or overweight → underweight; (5) normal or overweight → normal or overweight (no changes); (6) normal or overweight → obesity; (7) obesity → underweight; (8) obesity → normal or overweight; and (9) obesity → obesity (no changes). The BMI change rate was calculated by dividing the change in BMI between two health screenings by the interval between screenings (years).

Study outcome and follow-up

The primary outcome was all-cause mortality, which was identified based on death certificate data from the Korea National Statistical Office. Participants were followed up from 1 year after the index date until death or December 31, 2019, whichever came first.

Covariates

Low income was defined as individuals in the lowest 25th percentile of insurance premiums or Medical Aid eligibility. Place of residence was divided into urban (metropolitan and urban) and rural areas. Disability status was recorded, if present. Lifestyle variables were assessed using self-reported questionnaires. Smoking status was classified as never or ever smoked based on smoking history. Alcohol consumption was classified as nondrinker or drinker (>0 g/day). Regular physical activity was defined as vigorous exercise for at least 3 days/week or moderate exercise for at least 5 days/week. Blood pressure was measured after at least 5 min of rest in a sitting position. Blood samples were collected after overnight fasting.

Hypertension was defined as systolic/diastolic blood pressure of ≥ 140/90 mmHg or at least one antihypertensive medication prescription claim per year under ICD-10 codes I10–I13 or I15. Type 2 diabetes was defined as fasting plasma glucose ≥ 126 mg/dL or at least one antidiabetic medication prescription claim per year under ICD-10 codes E11–E14. Dyslipidemia was defined as a total cholesterol concentration ≥ 240 mg/dL or at least one lipid-lowering medication prescription claim per year under ICD-10 code E78. Chronic kidney disease was defined as estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m^2 by the Modification of Diet in Renal Disease equation.

Statistical analyses

Baseline characteristics were presented as the means ± standard deviation [SD] for continuous variables or numbers (percentages) for categorical variables according to mortality status during follow-up. Differences between groups were compared using analysis of variance for continuous variables and the chi-square test for categorical variables. The mortality rate was calculated as the number of deaths per 1,000 person-years. Kaplan-Meier curves were used to illustrate the cumulative incidence probabilities of all-cause mortality in the BMI and BMI change groups, with comparisons via log-rank tests. Cubic spline curves were used to visualize the associations of continuous BMI and BMI change rate with the probability of all-cause mortality. Multivariate Cox proportional hazards models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for all-cause mortality across the BMI categories and BMI change groups. Three models were applied: Model 1 was not adjusted; Model 2 was adjusted for age, sex, place of residence, income, smoking status, alcohol consumption, physical activity, hypertension, type 2 diabetes, dyslipidemia, and chronic kidney disease; Model 3 was additionally adjusted for disability status, the number of anti-dementia medications, and the interval between the two health screenings to the variables in Model 2. For analysis assessing the association between BMI after dementia diagnosis and all-cause mortality, Model 3 did not adjust for the interval between the two health screenings. The proportional hazards assumption was tested using Schoenfeld residuals and visual inspection of log-log survival plots, and no significant violations were observed. Subgroup analyses were conducted according to sex, age, and type 2 diabetes. Potential effect modification by these factors was evaluated by including interaction terms in the models, and corresponding *P* values for interaction were reported. Sensitivity analyses were conducted by excluding all-cause mortality occurring within 2, 3, and 5 years of follow-up and by stratifying the follow-up period into 1–3 years and 3–5 years after dementia diagnosis. All statistical analyses were performed using SAS software (version 9.4; SAS Institute, Cary, NC, USA). A *P*-value < 0.05 was considered statistically significant.

Results

Baseline characteristics

Table 1 shows the baseline characteristics of 37,717 participants (mean age: 74.2 years [SD: 7.5]) according to their mortality status during the follow-up period. The deceased individuals were older and had a higher proportion of men, rural residents, individuals without low income, those with disabilities, and smokers than the survivors. They had lower mean body weight, BMI, total

Table 1 Baseline characteristics of study participants by mortality status

	Total	Alive	Deceased	*P*
N	37,717	29,518	8,199	
Sex (men)	12,985 (34.4)	8,805 (29.8)	4,180 (51.0)	< 0.001
Age (years)	74.2 ± 7.5	73.2 ± 7.3	77.7 ± 6.9	< 0.001
Residential area (urban)	11,843 (31.4)	9,381 (31.8)	2,462 (30.0)	0.003
Income (low)	8,001 (21.2)	6,270 (21.2)	1,731 (21.1)	0.801
Disability (yes)	7,780 (20.6)	5,884 (19.9)	1,896 (23.1)	< 0.001
Ever smoker	7,171 (19.0)	5,008 (17.0)	2,163 (26.4)	< 0.001
Alcohol drinker	4,506 (12.0)	3,528 (12.0)	978 (11.9)	0.953
Regular physical activity	4,906 (13.0)	4,121 (14.0)	785 (9.6)	< 0.001
Abdominal obesity	11,504 (30.5)	9,368 (31.7)	2,136 (26.1)	< 0.001
Height (cm)	154.6 ± 8.9	154.3 ± 8.7	155.7 ± 9.6	< 0.001
Weight (kg)	56.1 ± 10.0	56.6 ± 9.8	54.5 ± 10.4	< 0.001
BMI (kg/m^2)	23.4 ± 3.3	23.7 ± 3.2	22.4 ± 3.3	< 0.001
Waist circumference (cm)	81.9 ± 9.0	82.1 ± 8.9	81.2 ± 9.3	< 0.001
Fasting glucose (mg/dL)	105.8 ± 30.1	104.8 ± 28.1	109.0 ± 36.5	< 0.001
Systolic BP (mmHg)	127.6 ± 15.8	127.5 ± 15.6	128.0 ± 16.6	0.004
Diastolic BP (mmHg)	76.2 ± 10.0	76.1 ± 9.8	76.3 ± 10.4	0.182
Total cholesterol (mg/dL)	190.4 ± 40.9	192.0 ± 40.7	184.5 ± 41.2	< 0.001
eGFR (ml/min/1.73 m^2)	76.5 ± 17.9	77.9 ± 17.1	71.5 ± 19.6	< 0.001
Hypertension	25,080 (66.5)	19,337 (65.5)	5,743 (70.1)	< 0.001
Type 2 diabetes	10,159 (26.9)	7,476 (25.3)	2,683 (32.7)	< 0.001
Dyslipidemia	17,173 (45.5)	14,164 (48.0)	3,009 (36.7)	< 0.001
Chronic kidney disease	6,947 (18.4)	4,761 (16.1)	2,186 (26.7)	< 0.001
Type of dementia				< 0.001
Alzheimer's disease	29,982 (79.5)	23,652 (80.1)	6,330 (77.2)	
Vascular dementia	3,220 (8.5)	2,471 (8.4)	749 (9.14)	
Others	4,515 (12.0)	3,395 (11.5)	1,120 (13.7)	
Number of anti-dementia medication				< 0.001
1	33,226 (88.1)	26,246 (88.9)	6,980 (85.1)	
2	4,145 (11.0)	3,020 (10.2)	1,125 (13.7)	
≥ 3	346 (0.9)	252 (0.86)	94 (1.1)	

Values are presented as means ± standard deviations or numbers (percentages)

Abbreviations: *BMI* body mass index, *BP* blood pressure, *eGFR* estimated glomerular filtration rate

cholesterol, and eGFR, but higher fasting glucose levels and systolic blood pressure. In terms of comorbidities, the prevalence of hypertension, type 2 diabetes, and chronic kidney disease was higher among individuals who died. Conversely, the proportion of patients with AD and VaD was higher among those who survived and the number of anti-dementia medications was higher among those who died. The characteristics of the excluded participants were similar to those of the study participants (Supplementary Table 1).

Associations between BMI after dementia diagnosis and risk of all-cause mortality

During a mean follow-up of 4.1 ± 1.8 years, there were 8,199 deaths (21.7%) among individuals with all-cause dementia, 6,330 deaths (21.1%) in those with AD, and 749 deaths (23.3%) in those with VaD. Table 2 shows the association between BMI after dementia diagnosis and all-cause mortality risk. A significant inverse association was observed between post-diagnosis BMI and mortality risk. Compared with individuals in the normal BMI category (18.5–22.9 kg/m^2), the risk was significantly increased among those who were underweight (HR 1.57, 95% CI 1.46–1.69 in all-cause dementia; HR 1.61, 95% CI 1.48–1.75 in AD; HR 1.90, 95% CI 1.47–2.47 in VaD). Conversely, individuals with BMI ≥ 30 kg/m^2 had the lowest mortality risk (HR 0.62, 95% CI 0.51–0.75 in AD; HR 0.58, 95% CI 0.36–0.93 in VaD), while those with BMI 25.0–29.9 kg/m^2 showed the lowest risk in all-cause dementia (HR 0.64, 95% CI 0.61–0.68). As shown in Supplementary Fig. 2, the cumulative incidence probabilities of all-cause mortality were higher in patients with a lower BMI (log-rank $p < 0.001$). Supplementary Fig. 3 demonstrates an inverse J-shaped association between continuous BMI and mortality, with a reference point at a BMI of 30.2 kg/m^2. By contrast, increased mortality risk was observed as BMI decreased below the reference value of 31.9 kg/m^2 in both AD and VaD.

Table 2 Risk of all–cause mortality by BMI status after dementia diagnosis

BMI (kg/m^2)	*N*	Mortality	Person–years	IR[a]	HR (95% CI)		
					Model 1[b]	Model 2[c]	Model 3[d]
All–cause dementia							
< 18.5	2,173	891	7,671	116.2	1.81 (1.69–1.95)	1.57 (1.46–1.69)	1.57 (1.46–1.69)
18.5–22.9	14,928	3,888	59,664	65.2	1 (Ref.)	1 (Ref.)	1 (Ref.)
23.0–24.9	9,159	1,702	38,499	44.2	0.67 (0.64–0.71)	0.74 (0.70–0.78)	0.74 (0.70–0.78)
25.0–29.9	10,344	1,571	43,537	36.1	0.55 (0.52–0.58)	0.64 (0.61–0.68)	0.64 (0.61–0.68)
≥ 30.0	1,113	147	4,649	31.6	0.49 (0.41–0.57)	0.66 (0.56–0.77)	0.65 (0.55–0.76)
Alzheimer's disease							
< 18.5	1,754	704	6,127	114.9	1.83 (1.69–1.99)	1.60 (1.48–1.74)	1.61 (1.48–1.75)
18.5–22.9	11,905	3,002	47,126	63.7	1 (Ref.)	1 (Ref.)	1 (Ref.)
23.0–24.9	7,249	1,302	30,349	42.9	0.67 (0.63–0.71)	0.74 (0.69–0.79)	0.74 (0.69–0.79)
25.0–29.9	8,213	1,216	34,156	35.6	0.56 (0.52–0.59)	0.65 (0.61–0.69)	0.65 (0.61–0.69)
≥ 30.0	861	106	3,580	29.6	0.46 (0.38–0.56)	0.63 (0.52–0.76)	0.62 (0.51–0.75)
Vascular dementia							
< 18.5	143	71	493	144.1	2.10 (1.63–2.72)	1.89 (1.46–2.45)	1.90 (1.47–2.47)
18.5–22.9	1,186	336	4,799	70.0	1 (Ref.)	1 (Ref.)	1 (Ref.)
23.0–24.9	808	166	3,323	50.0	0.71 (0.59–0.86)	0.78 (0.64–0.94)	0.78 (0.64–0.94)
25.0–29.9	953	158	4,115	38.4	0.55 (0.45–0.66)	0.64 (0.53–0.78)	0.64 (0.53–0.78)
≥ 30.0	130	18	556	32.4	0.46 (0.29–0.74)	0.58 (0.36–0.94)	0.58 (0.36–0.93)

Associations between BMI changes and risk of all-cause mortality

Table 3 shows the associations between changes in BMI status before and after dementia diagnosis and all-cause mortality, with individuals with normal BMI or overweight as the reference group. Among those underweight prior to diagnosis, both maintaining that status (HR 1.73, 95% CI 1.55–1.92) and gaining weight to normal or overweight (HR 1.48, 95% CI 1.29–1.70) were associated with increased mortality. For individuals with normal BMI or overweight, weight loss to underweight increased mortality risk (HR 1.76, 95% CI 1.60–1.93), while weight gain to obesity reduced the risk (HR 0.84, 95% CI 0.76–0.93). Among those with obesity before diagnosis, weight loss to underweight showed the highest mortality risk (HR 2.09, 95% CI 1.26–3.46), whereas maintaining obesity showed the lowest risk (HR 0.69, 95% CI 0.65–0.74). These associations were consistent in both AD and VaD. In particular, among VaD patients, weight gain from underweight to obesity was associated with markedly elevated risk (HR 10.23, 95% CI 1.42–73.61), whereas maintaining obesity was associated with the lowest risk (HR 0.66, 95% CI 0.54–0.81). Similarly, weight loss from obesity to underweight (HR 5.68, 95% CI 1.80–17.89), sustained underweight (HR 2.52, 95% CI 1.76–3.59) and weight loss from normal/overweight to underweight (HR 1.74, 95% CI 1.22–2.49) were also associated with higher mortality in VaD. Supplementary Table 2 shows the associations between 16 BMI change groups before and after dementia diagnosis and all-cause mortality, using individuals with normal BMI as the reference group. Among individuals with all-cause dementia, weight loss from overweight to underweight was associated with markedly elevated risk (HR 2.47, 95% CI 1.82–3.35), whereas maintaining obesity was associated with the lowest risk (HR 0.63, 95% CI 0.59–0.68). Similar patterns were observed in both AD and VaD. As shown in Fig. 1, the cumulative incidence probabilities of all-cause mortality across BMI change statuses before and after dementia diagnosis showed similar trends (log-rank, $p < 0.001$). Supplementary Fig. 4 shows U-shaped associations between BMI change rate and all-cause mortality risk among individuals with all-cause dementia, AD, and VaD.

Subgroup analyses and sensitivity analysis

Table 4 shows the associations between changes in BMI status and all-cause mortality across sex and age subgroups. The associations exhibited similar tendencies in all subgroups. There were significant interactions with age, with stronger associations observed in individuals aged 40–64 years for both all-cause dementia (P for interaction < 0.001) and AD (P for interaction < 0.001). Among individuals with all-cause dementia, the association was more pronounced in women (P for interaction = 0.047). In contrast, no significant interaction was observed with type 2 diabetes.

Supplementary Tables 3 and 4 present sensitivity analyses, excluding all-cause mortality occurring within 2, 3, and 5 years of follow-up among individuals with dementia. The associations showed similar tendencies even when the lag time was extended to 2, 3, and 5 years. In addition, Supplementary Tables 5 and 6 show the risk of all-cause mortality during 1–3 and 3–5 years among

Table 3 Risk of all-cause mortality by changes in BMI status before and after dementia diagnosis

Change in BMI status	*N*	Event	Person–years	IR[a]	HR (95% CI)		
					Model 1[b]	Model 2[c]	Model 3[d]
In all–cause dementia							
Underweight → Underweight	979	388	3,503	110.8	2.02 (1.82–2.24)	1.71 (1.54–1.90)	1.73 (1.55–1.92)
Underweight → Normal or overweight	641	214	2,388	89.6	1.63 (1.42–1.87)	1.48 (1.29–1.70)	1.48 (1.29–1.70)
Underweight → Obesity	12	5	44	113.3	2.09 (0.87–5.01)	1.66 (0.69–3.99)	1.67 (0.70–4.03)
Normal or overweight → Underweight	1,166	488	4,076	119.7	2.19 (2.00–2.40)	1.76 (1.61–1.94)	1.76 (1.60–1.93)
Normal or overweight → Normal or overweight	20,508	4,668	83,697	55.8	1 (Ref.)	1 (Ref.)	1 (Ref.)
Normal or overweight → Obesity	2,355	424	9,581	44.3	0.80 (0.72–0.88)	0.84 (0.76–0.93)	0.84 (0.76–0.93)
Obesity → Underweight	28	15	92	163.1	3.02 (1.82–5.01)	2.13 (1.29–3.54)	2.09 (1.26–3.46)
Obesity → Normal or overweight	2,938	708	12,078	58.6	1.05 (0.97–1.14)	1.02 (0.94–1.10)	1.00 (0.93–1.09)
Obesity → Obesity	9,090	1,289	38,561	33.4	0.60 (0.56–0.64)	0.70 (0.65–0.74)	0.69 (0.65–0.74)
In Alzheimer's disease							
Underweight → Underweight	791	291	2,856	101.9	1.90 (1.68–2.14)	1.61 (1.43–1.82)	1.62 (1.44–1.83)
Underweight → Normal or overweight	497	161	1,811	88.9	1.66 (1.42–1.94)	1.50 (1.28–1.76)	1.51 (1.28–1.76)
Underweight → Obesity	10	4	36	111.9	2.12 (0.80–5.66)	1.81 (0.68–4.83)	1.81 (0.68–4.83)
Normal or overweight → Underweight	940	401	3,201	125.3	2.35 (2.12–2.61)	1.93 (1.74–2.14)	1.92 (1.73–2.14)
Normal or overweight → Normal or overweight	16,374	3,614	66,261	54.5	1 (Ref.)	1 (Ref.)	1 (Ref.)
Normal or overweight → Obesity	1,854	317	7,486	42.3	0.78 (0.69–0.87)	0.83 (0.74–0.93)	0.83 (0.74–0.93)
Obesity → Underweight	23	12	71	169.2	3.22 (1.83–5.68)	1.94 (1.10–3.41)	1.89 (1.07–3.33)
Obesity → Normal or overweight	2,283	529	9,403	56.3	1.03 (0.94–1.13)	1.00 (0.91–1.09)	0.99 (0.90–1.09)
Obesity → Obesity	7,210	1,001	30,214	33.1	0.61 (0.56–0.65)	0.70 (0.65–0.75)	0.70 (0.65–0.75)
In vascular dementia							
Underweight → Underweight	63	34	219	155.1	2.61 (1.84–3.70)	2.46 (1.72–3.52)	2.51 (1.76–3.59)
Underweight → Normal or overweight	40	14	148	94.8	1.62 (0.95–2.76)	1.40 (0.82–2.40)	1.38 (0.80–2.36)
Underweight → Obesity	1	1	2	459.1	8.89 (1.25–63.33)	9.54 (1.33–68.55)	10.23 (1.42–73.6)
Normal or overweight → Underweight	76	34	257	132.1	2.27 (1.60–3.22)	1.75 (1.23–2.50)	1.74 (1.22–2.49)
Normal or overweight → Normal or overweight	1,670	412	6,850	60.1	1 (Ref.)	1 (Ref.)	1 (Ref.)
Normal or overweight → Obesity	231	50	937	53.4	0.90 (0.67–1.20)	0.87 (0.65–1.17)	0.87 (0.65–1.17)
Obesity → Underweight	4	3	16	187.2	3.17 (1.02–9.87)	5.60 (1.78–17.64)	5.68 (1.80–17.89)
Obesity → Normal or overweight	284	76	1,125	67.5	1.13 (0.88–1.44)	1.11 (0.86–1.41)	1.09 (0.85–1.39)
Obesity → Obesity	851	125	3,732	33.5	0.55 (0.45–0.68)	0.66 (0.54–0.81)	0.66 (0.54–0.81)

Abbreviations: BMI, body mass index; IR, incidence rate; HR, hazard ratio; CI, confidence interval

[a]Incidence per 1000 person-years

[b]Model 1 was not adjusted for any variables

[c]Model 2 was adjusted for age, sex, place of residence, income, smoking status, alcohol consumption, physical activity, hypertension, type 2 diabetes, dyslipidemia, and chronic kidney disease

[d]Model 3 was adjusted for age, sex, place of residence, income, smoking status, alcohol consumption, physical activity, hypertension, type 2 diabetes, dyslipidemia, chronic kidney disease, disability, number of anti-dementia medications, and interval between two health screening

individuals with dementia, which were similar to the previous associations.

Discussion

Using a large, nationally representative cohort, this study found that lower BMI after dementia diagnosis was associated with higher all-cause mortality, with the highest risk observed in underweight individuals and progressive decrease in risk as BMI increased across all-cause dementia, AD, and VaD. Maintaining obesity or gaining weight from normal or overweight to obese after diagnosis was associated with reduced mortality, suggesting a higher BMI was associated with a reduced risk of all-cause mortality in this population. In contrast, excessive weight loss–particularly from obesity to underweight–was consistently associated with the highest mortality across dementia types. Sustained underweight and weight gain from underweight to a normal or overweight were linked to increased mortality. These findings suggest that underweight status both before and after dementia diagnosis and experiencing excessive BMI reduction are independently associated with a higher mortality risk.

Our findings align with previous research showing that underweight individuals with dementia have increased mortality risk compared with those with normal weight [19]. Underweight status is associated with susceptibility to infections [20] and reduced muscle mass [21], which is an important risk factor for mortality in patients with nutritional deficits, such as those with dementia [22]. Previous studies have also shown that body weight

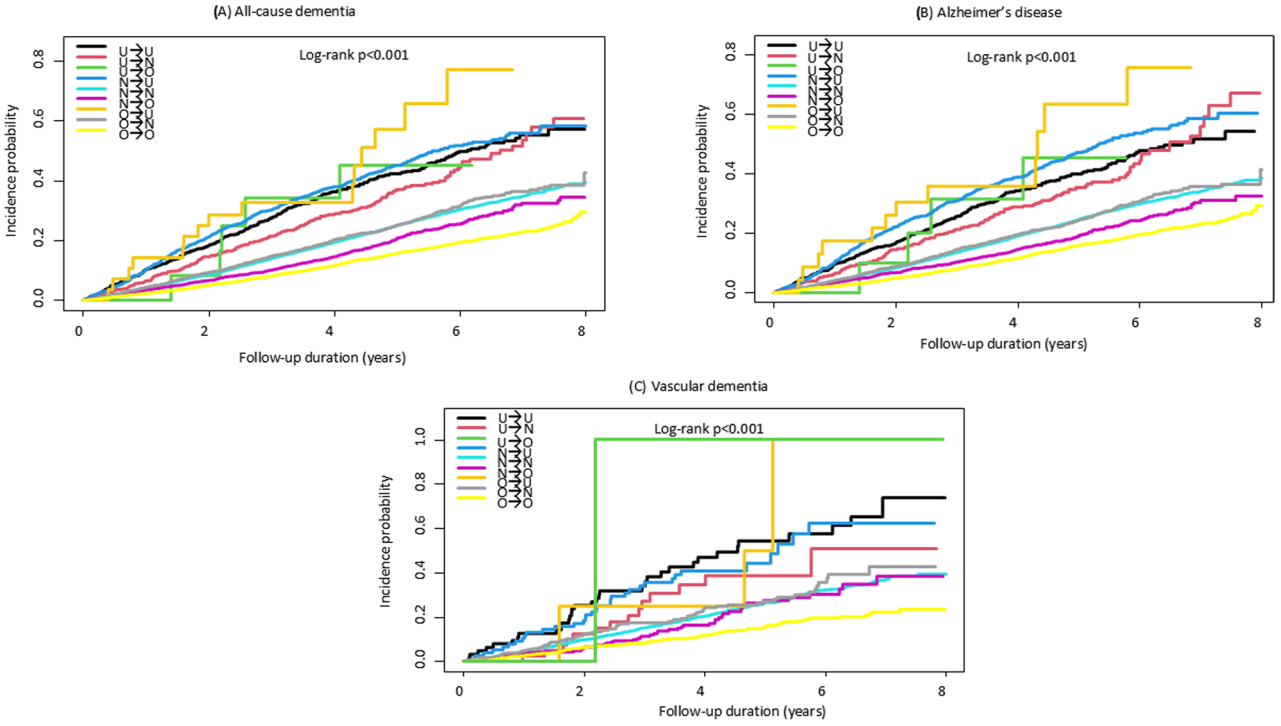

Fig. 1 Kaplan–Meier curves for association between changes in BMI status before and after dementia diagnosis and all-cause mortality (A) All-cause dementia, B) Alzheimer's disease, C) Vascular dementia)

changes are associated with poor health outcomes [23, 24]. One cohort study including 11,607 individuals with AD, VaD, and Lewy body dementia found that weight loss was associated with increased mortality in AD (HR 1.07, 95% CI 1.02–1.15) and with a higher risk of emergency hospitalization in all-cause dementia (HR 1.14, 95% CI 1.08–1.20) [9]. Another study that followed 43,721 Norwegians aged 35–49 years at baseline for an average of 9 years reported that weight loss of ≥ 10% during midlife increased dementia-related mortality risk by 52%, and 5–10% weight loss increased it by 38% [14]. In another prospective cohort study of 79 elderly adults with AD, weight loss > 3 kg over 3 months was associated with a significantly higher all-cause mortality risk (HR 3.82, 95% CI 1.37–10.63) [25]. Weight loss has been reported to occur during the preclinical and prodromal stages of dementia, and may reflect underlying disease progression rather than a consequence of clinically overt dementia. Consistent with these findings, our study revealed that excessive weight loss was associated with the highest risk of mortality among individuals with all-cause dementia, AD, and VaD.

While weight gain is generally regarded as a potential risk factor due to its association with unhealthy lifestyles and metabolic disorders [26], which are known contributors to all-cause mortality [27], our findings indicate a more complex relationship in the context of dementia. Additionally, a recent study of 2,174 Chinese older adults with cognitive impairment showed that higher BMI and waist circumference were linked to a lower risk of mortality [28]. Specifically, individuals who were underweight before their dementia diagnosis and gained weight to reach a normal BMI or who were overweight still showed a significantly increased risk of mortality. This suggests that weight gain from an underweight state does not fully offset underlying vulnerabilities, such as frailty or disease severity. Furthermore, those who changed from underweight to obese did not show a statistically significant association with mortality risk, which may be attributed to the small number of individuals in this subgroup who underwent such significant weight changes, limiting the statistical power. Meanwhile, patients with dementia who maintained obesity or gained weight from normal BMI to obesity after their dementia diagnosis showed a modest reduction in mortality risk. This may reflect the protective effect of higher energy reserves and nutritional buffering in the face of progressive cognitive and functional decline. In addition, weight maintenance or minimal weight change among individuals with dementia may reflect slower disease progression or better physical function.

Several mechanisms can explain these associations. First, excessive BMI change may be a potential indicator when serious comorbidities lead to worse prognoses [24]. In particular, weight loss may be caused by reduced food intake due to swallowing difficulties [29]

Table 4 Subgroup analysis

	Changes in BMI status	In all-cause dementia			In Alzheimer's disease			In vascular dementia		
		N	Event	HR (95% CI)	*N*	Event	HR (95% CI)	*N*	Event	HR (95% CI)
Sex										
Men	U → U	375	191	1.63 (1.41–1.89)	302	144	1.50 (1.27–1.78)	21	12	2.21 (1.23–3.98)
	U → N	221	88	1.26 (1.02–1.55)	170	67	1.29 (1.01–1.65)	16	7	1.46 (0.69–3.13)
	U → O	6	2	1.06 (0.26–4.23)	4	1	0.89 (0.13–6.31)	1	1	10.56 (1.47–76.09)
	N → U	406	219	1.56 (1.36–1.79)	310	173	1.69 (1.45–1.98)	36	22	2.09 (1.34–3.26)
	N → N	7,745	2,566	1 (Ref.)	6,006	1,969	1 (Ref.)	706	229	1 (Ref.)
	N → O	787	229	0.89 (0.78–1.02)	594	164	0.87 (0.74–1.02)	93	33	0.97 (0.67–1.40)
	O → U	10	8	2.43 (1.22–4.86)	10	8	2.46 (1.23–4.94)	-	-	-
	O → N	923	316	0.95 (0.85–1.07)	661	218	0.93 (0.81–1.07)	116	48	1.26 (0.92–1.73)
	O → O	2,512	561	0.69 (0.63–0.75)	1,959	441	0.69 (0.62–0.77)	283	56	0.64 (0.47–0.86)
Women	U → U	604	197	1.84 (1.59–2.13)	489	147	1.76 (1.48–2.08)	42	22	2.67 (1.71–4.16)
	U → N	420	126	1.70 (1.42–2.04)	327	94	1.71 (1.39–2.11)	24	7	1.29 (0.60–2.76)
	U → O	6	3	2.71 (0.87–8.43)	6	3	2.78 (0.89–8.63)	-	-	-
	N → U	760	269	1.98 (1.74–2.24)	630	228	2.16 (1.88–2.48)	40	12	1.34 (0.74–2.41)
	N → N	12,763	2,102	1 (Ref.)	10,368	1,645	1 (Ref.)	964	183	1 (Ref.)
	N → O	1,568	195	0.79 (0.68–0.91)	1,260	153	0.78 (0.66–0.92)	138	17	0.74 (0.45–1.21)
	O → U	18	7	1.82 (0.86–3.82)	13	4	1.29 (0.48–3.45)	4	3	5.50 (1.74–17.36)
	O → N	2,015	392	1.06 (0.95–1.18)	1,622	311	1.05 (0.93–1.19)	168	28	0.87 (0.59–1.30)
	O → O	6,578	728	0.70 (0.65–0.77)	5,251	560	0.71 (0.64–0.78)	568	69	0.67 (0.50–0.88)
	P for interaction			0.047			0.093			0.611
Age (years)										
40–64	U → U	70	18	3.24 (2.00–5.25)	63	13	2.79 (1.58–4.93)	3	2	7.37 (1.70–31.99)
	U → N	56	7	1.57 (0.74–3.33)	36	7	3.02 (1.41–6.44)	7	0	-
	U → O	-	-	-	-	-	-	-	-	-
	N → U	70	17	2.60 (1.59–4.28)	57	14	2.91 (1.68–5.05)	3	1	3.25 (0.43–24.69)
	N → N	2,121	193	1 (Ref.)	1,588	141	1 (Ref.)	240	19	1 (Ref.)
	N → O	311	25	1.03 (0.68–1.60)	215	13	0.83 (0.47–1.46)	43	5	1.49 (0.55–3.99)
	O → U	2	2	102.36 (25.43–411.94)	1	1	148.93 (20.77–1067.82)	1	1	107.82 (14.22–817.55)
	O → N	264	24	1.07 (0.70–1.64)	191	16	0.96 (0.57–1.61)	39	3	1.37 (0.41–4.65)
	O → O	1,186	58	0.55 (0.41–0.73)	880	40	0.52 (0.36–0.74)	146	8	0.73 (0.32–1.66)
Age ≥ 65	U → U	909	370	1.67 (1.50–1.86)	728	278	1.57 (1.39–1.78)	60	32	2.39 (1.66–3.46)
	U → N	585	207	1.47 (1.28–1.69)	461	154	1.46 (1.25–1.72)	33	14	1.44 (0.84–2.46)
	U → O	12	5	1.70 (0.71–4.09)	10	4	1.84 (0.69–4.90)	1	1	10.40 (1.44–74.97)
	N → U	1,096	471	1.72 (1.56–1.89)	883	387	1.88 (1.69–2.09)	73	33	1.71 (1.20–2.45)
	N → N	18,387	4,475	1 (Ref.)	14,786	3,473	1 (Ref.)	1,430	393	1 (Ref.)
	N → O	2,044	399	0.83 (0.75–0.92)	1,639	304	0.83 (0.74–0.93)	188	45	0.84 (0.61–1.14)
	O → U	26	13	1.79 (1.04–3.08)	22	11	1.71 (0.94–3.08)	3	2	3.89 (0.96–15.79)
	O → N	2,674	684	1.00 (0.93–1.09)	2,092	513	0.99 (0.90–1.09)	245	73	1.08 (0.84–1.39)
	O → O	7,904	1,231	0.70 (0.66–0.75)	6,330	961	0.71 (0.66–0.76)	705	117	0.66 (0.53–0.81)
	P for interaction			< 0.001			< 0.001			0.213
Type 2 diabetes										
No	U → U	832	318	1.71 (1.52–1.92)	674	236	1.59 (1.39–1.82)	52	29	2.78 (1.88–4.10)
	U → N	530	169	1.45 (1.24–1.69)	414	126	1.44 (1.20–1.72)	29	11	1.98 (1.08–3.63)
	U → O	8	4	2.24 (0.84–5.98)	6	3	2.93 (0.94–9.08)	1	1	10.29 (1.43–74.09)
	N → U	917	356	1.70 (1.52–1.90)	733	289	1.86 (1.65–2.11)	53	20	1.49 (0.94–2.35)
	N → N	15,542	3,233	1 (Ref.)	12,456	2,513	1 (Ref.)	1,218	272	1 (Ref.)
	N → O	1,697	255	0.79 (0.70–0.90)	1,332	191	0.78 (0.68–0.91)	162	28	0.76 (0.51–1.12)
	O → U	21	12	2.56 (1.45–4.51)	16	9	2.30 (1.20–4.43)	4	3	5.60 (1.78–17.65)
	O → N	1,920	410	1.02 (0.92–1.13)	1,491	304	1.00 (0.89–1.13)	184	45	1.08 (0.79–1.49)
	O → O	6,091	759	0.71 (0.66–0.77)	4,833	595	0.73 (0.66–0.80)	547	66	0.62 (0.47–0.82)

Table 4 (continued)

	Changes in BMI status	In all-cause dementia			In Alzheimer's disease			In vascular dementia		
		N	Event	HR (95% CI)	*N*	Event	HR (95% CI)	*N*	Event	HR (95% CI)
Yes	U → U	147	70	1.79 (1.41–2.28)	117	55	1.76 (1.34–2.31)	11	5	1.56 (0.64–3.84)
	U → N	111	45	1.61 (1.20–2.17)	83	35	1.81 (1.29–2.54)	11	3	0.64 (0.20–2.03)
	U → O	4	1	0.83 (0.12–5.92)	4	1	0.85 (0.20–6.02)	-	-	-
	N → U	249	132	1.95 (1.63–2.33)	207	112	2.10 (1.73–2.55)	23	14	2.29 (1.32–4.00)
	N → N	4,966	1,435	1 (Ref.)	3,918	1,101	1 (Ref.)	452	140	1 (Ref.)
	N → O	658	169	0.93 (0.79–1.09)	522	126	0.90 (0.75–1.08)	69	22	1.08 (0.69–1.69)
	O → U	7	3	1.20 (0.39–3.71)	7	3	1.23 (0.39–3.81)	-	-	-
	O → N	1,018	298	0.99 (0.87–1.12)	792	225	0.98 (0.85–1.13)	100	31	1.10 (0.75–1.63)
	O → O	2,999	530	0.67 (0.61–0.75)	2,377	406	0.66 (0.59–0.74)	304	59	0.71 (0.52–0.97)
	P for interaction			0.417			0.329			0.269

Data are expressed as hazard ratios (95% confidence intervals) calculated using a multivariable Cox hazards regression model adjusted for age, sex, place of residence, income, smoking status, alcohol consumption, physical activity, hypertension, type 2 diabetes, dyslipidemia, chronic kidney disease, disability, the number of anti-dementia medications, and interval between two health screening

Abbreviations: *BMI* body mass index, *HR* hazard ratio, *CI* confidence interval, *U* underweight, *N* normal weight or overweight, *O* obese

and is an important clinical marker of severe dementia [30]. In addition, weight change may reflect rapid cognitive decline and increased neuropsychiatric symptoms [31], both of which are associated with the progression of dementia. A possible biological pathway involves adipocyte-derived leptin in the central nervous system; the decreased number and function of leptin were associated with cognitive decline in severe AD because leptin leads to reduced amyloid-β production and tau phosphorylation [32]. Finally, body weight fluctuation can affect homeostasis of blood pressure and blood glucose levels [33], which are significantly associated with poor health outcomes [34].

Interestingly, in our subgroup analysis, the association between changes in BMI in all-cause dementia and mortality risk was more pronounced in middle-aged individuals than in older adults. This may reflect a stronger physiological impact of weight change at a younger age, possibly indicating a more aggressive disease course or an atypical presentation of dementia, such as early-onset types [35]. Younger individuals may also have more comorbidities that amplify the negative impact of weight loss [36]. Among patients with AD, the associations were stronger in women, which could be attributed to sex differences in body composition, hormonal changes, and lower baseline muscle mass [37], making them more vulnerable to malnutrition. In patients with VaD, pronounced associations in middle-aged adults may reflect the greater clinical burden of vascular risk factors, such as diabetes and hypertension, which could interact with weight changes to worsen outcomes [38]. However, further studies are required to elucidate the precise mechanisms underlying these associations.

Our study had several limitations. First, owing to the observational nature of the study, reverse causality cannot be excluded. Second, although the mean follow-up duration of 4.1 years may appear relatively short, it is likely sufficient given the typically rapid progression and limited survival of patients with dementia. Third, it was unclear whether observed weight changes were intentional or unintentional, which may have affected mortality outcomes differently, given the claims-based nature of the data. In addition, because the number of participants in certain extreme weight change groups was small (e.g., obese to underweight or underweight to obese), corresponding estimates were imprecise with wide confidence intervals and should be interpreted with caution. Fourth, residual confounding factors, such as dietary data, may exist due to unmeasured variables not included in the NHIS database. Additionally, the NHIS database does not include direct measures of sarcopenia or body composition, such as dual-energy X-ray absorptiometry, therefore, we were unable to distinguish between loss of fat mass and loss of muscle mass, which may be particularly relevant in older adults with dementia. Fifth, lifestyle factors were assessed using self-reported questionnaires and may therefore be subject to recall bias, especially after a dementia diagnosis, however, caregiver assistance may have partially reduced misclassification. Finally, the analysis was limited to a subset of individuals with available health screening data before and after dementia diagnosis, which was required to evaluate longitudinal changes in BMI and may reduce generalizability.

Nevertheless, this study had several notable strengths. Our study utilized a large-scale, population-based Korean database, thereby offering stronger generalizability and more nuanced insights into BMI trajectories and mortality outcomes in patients with dementia. We adjusted for a wide range of potential confounding variables, including sociodemographic factors, comorbidities, health behaviors, and dementia severity, which enhanced the robustness of our findings. Furthermore, we examined

BMI changes both before and after dementia diagnosis for the first time. This longitudinal approach allowed us to capture the dynamic nature of body weight trajectories in patients with dementia and to better reflect real-world clinical scenarios. In addition, we stratified the analyses by dementia subtype and conducted subgroup analyses by age, sex, and type 2 diabetes to provide more detailed and clinically meaningful interpretations. This comprehensive analysis offers new insights into how weight changes during the critical transition period at the time of dementia diagnosis may influence the long-term mortality risk. Collectively, our findings offer new insights into how BMI changes during the critical transition period surrounding dementia diagnosis may influence mortality risk.

In conclusion, this large-scale cohort study suggests that sustained underweight and excessive weight loss before and after dementia diagnosis are associated with increased all-cause mortality, with body weight interpreted as a surrogate marker of underlying health status rather than a causal factor. Conversely, relatively stable or modest weight gain was associated with lower mortality risk. These findings underscore the clinical importance of minimizing unintended weight loss, preventing undernutrition, and maintaining stable or slightly higher weight as part of comprehensive dementia care.

Supplementary Information

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Supplementary Material 1.

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None.

Authors' contributions
Y.H. contributed to the study conception, literature search, collection and assembly of data, and wrote the original draft. D.L., H.J.P., M.Y.Y., S.Y.Y., S.H.K., S.H.K., C.K.K., S.M.K., and H.S.P. discussed the results and commented on the manuscript. K.H., J.J., and B.S.K. contributed data analyses and interpretation. G.E.N. and K.H. contributed to the concept, design, data collection, critical revision of the manuscript, supervised the study, and take responsibility for the study. All authors approved the final version of the manuscript. All listed authors have approved the manuscript and agreed to its submission for publication.

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Data availability
This study was performed using the Korean National Health Insurance System database, and the results do not necessarily represent the opinion of the National Health Insurance Corporation. Restrictions apply to the availability of these data, which were used under license for this study.

Declarations

Ethics approval and consent to participate
The study was conducted in accordance with the principles of the Declaration of Helsinki. This study was approved by the Institutional Review Board of the Korea University Guro Hospital, Seoul, Korea (No. 2022GR0325). The board granted an exemption from informed consent because all data used in the analysis were anonymous and de-identified.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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