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# Impact of Mediterranean diet on mortality in vertebral compression fracture patients

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## Abstract

**Background** Vertebral compression fractures (VCF) is a common fragility fracture with high mortality worldwide. The management and prevention of VCF start with a proper nutrition. The Mediterranean diet (MD) is rich in balanced nutrients and has been shown to be beneficial for several chronic diseases. However, the association of adherence to Mediterranean diet (aMED) and prognosis of VCF patients remains unclear.

**Purposes** To explore the association between aMED and all-cause and cardiovascular disease (CVD)-cause morality in VCF patients.

**Methods** In present study, patients aged ≥ 40 years old and with the VCF patients measurement were extracted from the National Health and Nutrition Examination Survey (NHANES) 2013–2014. The bone mineral density (BMD) dual-energy X-ray absorptiometry (DXA) was used to diagnose VCF. We used the weighted univariable Cox proportional hazards model to screen the covariates related to the prognosis of VCF patients. We utilized the weighted multivariable Cox proportional hazards models to explore the association between aMED and the risk of mortality in VCF patients, and were described as hazard ratios (HRs) and 95% confidence intervals (CIs). Subgroup analyses based on different complications were further assessed the association.

**Results** A total of 2,730 eligible VCF patients were included. Until 12 December 2019, 218 (7.99%) deaths were documented. After adjusting for all VCFs, we found a high risk of all-cause mortality (HR = 1.75, 95%CI: 1.13–2.73, P = 0.041) and CVD-cause mortality (HR = 2.35, 95%CI: 1.12–4.91, P = 0.038); however, we found no significant association between aMED and all-cause mortality or CVD-cause mortality (all P > 0.05). Compared to patients without VCF and with aMED score ≥ 6, patients with VCF and aMED score < 6 has a higher risk of all-cause (HR = 2.27, 95%CI: 1.25–4.13, *P* = 0.025) and CVD-cause mortality (HR = 4.25, 95%CI: 1.64–11.06, *P* = 0.013). Our study also suggested that compared to patients with aMED ≥ 6, those patients with aMED < 6 has high all-cause (HR = 2.26, 95%CI: 1.22–4.17, *P* = 0.002) and CVD-cause mortality (HR = 3.31, 95%CI: 1.28–8.57, *P* = 0.018), this results suggested that aMED may have a moderating effect on the association of VCF and mortality. Subgroups analysis shown this moderating effect remain robust, especially in patients with dyslipidemia (HR: 2.49, 95%CI: 1.29–4.80, *P* = 0.009), CVD (HR: 3.48, 95%CI: 1.56–7.74, *P* < 0.001) and CKD (HR: 3.64, 95%CI: 1.50–8.78, *P* < 0.001).

**Conclusion** We found aMED have a moderating effect on the association between VCF patients and mortality. Our research further supports the importance of the MD as a potentially healthy eating pattern.

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## Background

Vertebral compression fractures (VCF) refers to the reduction of bone mineral density (BMD), bone quality, and bone strength, which occurs under slight or even no obvious external force [1, 2]. The incidence of VCF in the general population increases with age, and the prevalence was more than 20% among people over 50 years old [3]. VCF can lead to more severe physical limitations, including functional disability, back pain, and the possibility of other fractures, ultimately leading to a higher risk of death [4]. Active study on the modifiable factors related to VCF was necessary to improve the outcome of VCF and reduce the public health burden.

Calcium (Ca) was a major player in bone health, and it was able to provide the vital mineralization of bone to confer bone strength and structure [5]. The main way for the human body to obtain Ca is through dietary intake, and the appropriate dietary Ca intake level has a strong impact on bone accumulation, maintenance and loss at all stages of life [5]. The Mediterranean diet (MD), referring the dietary pattern usually consumed among the population bordering the Mediterranean sea, has become known for its unique health benefits and prevention against several diseases such as cardiovascular diseases (CVD) as well as metabolism-related diseases [6]. A previous study reported that the incidence of osteoporosis and fragility fractures is very variable in the countries of the European Union, but it has been observed that it was lower in the Mediterranean area [7]. Adherence to Mediterranean diet (aMED) score was an index to evaluate adherence to the Mediterranean diet pattern. Several studies have shown that greater aMED can improve the mineral status of the bones and reduce the risk of fractures [8–10].

While the Mediterranean diet (MD) has been widely recognized for its health benefits, including its positive impact on bone health and cardiovascular diseases, it is important to consider other dietary patterns in the context of vertebral compression fractures (VCF). For instance, the Western diet, characterized by high intake of red meat, processed foods, and sugars, has been associated with increased inflammation and oxidative stress, which are detrimental to bone health [11]. In contrast, the DASH (Dietary Approaches to Stop Hypertension) diet, which emphasizes fruits, vegetables, and low-fat dairy products, has been shown to have beneficial effects on blood pressure and cardiovascular health, but its specific impact on VCF outcomes remains less explored [12]. Similarly, the vegetarian diet, which is rich in plant-based foods and often high in antioxidants and fiber, may also have potential benefits for bone health, although more research is needed to establish its role in VCF management [13]. By comparing these dietary patterns, we can better understand the unique contributions of the MD to the prevention and management of VCFs.

On the basis of the above studies about the relationship between MD and osteoporosis, we hypothesized that a greater aMED can improve the prognosis of VCF patients. Herein, using the data from the National Health and Nutrition Examination Survey (NHANES) database, this study aimed to conduct mediation analysis and subgroup analysis based on weighted cox proportional hazard models to explore the moderating effect of aMED on the association between VCF and mortality.

## Methods

### Study design and population

Data of this study were extracted from the NAHNES database 2013–2014. The NHANES is conducted by the National Centers for Health Statistics (NCHS), the Centers for Disease Control and Prevention (CDC) to assess the health and nutritional status of adults and children in the United States. NHANES uses complex, multistage, probability sampling methods based on the board population. All participants have provided informed consent during the survey. According to the Ethics Review Board of Dongzhimen Hospital Beijing University of Chinese Medicine, cross-sectional studies have been exempted from the ethical review.

In present study, 3,815 adults aged ≥ 40 years old were initial extracted from the NHANES database.

Inclusion and Exclusion Criteria:

To ensure the robustness and relevance of our study, we employed specific criteria for the inclusion and exclusion of participants.

Inclusion Criteria:

① Patients aged 40 years or older.
② Patients with a diagnosis of vertebral compression fractures (VCF) confirmed by dual-energy X-ray absorptiometry (DXA) using the vertebral fracture assessment (VFA) method [2].
③ Patients with complete dietary intake information, including the 24-h dietary recall interview conducted at the Mobile Examination Center (MEC) [14].

④ Patients with available survival data up to December 12, 2019.

Exclusion Criteria:

① Patients missing VCF measurement data ($n=591$).
②Patients with incomplete dietary intake information ($n=251$).
③ Patients missing survival data ($n=5$).
④ Patients with extreme energy intake, defined as less than 500 kcal/day or more than 5000 kcal/day for females, and less than 500 kcal/day or more than 8000 kcal/day for males ($n=31$).
⑤ Patients missing information on femoral neck bone mineral density (BMD) ($n=173$).
⑥ Patients missing important covariates such as marital status, smoking, sedentary time, insurance, osteoporosis, and body mass index (BMI) ($n=34$).

After applying these criteria, a total of 2,730 eligible VCF patients were included in the final analysis. The flow chart of population screening is shown in Fig. 1.

### Ascertainment of death

Information on vital status of the participants was obtained from clinical follow-up data collection and from municipal records. Cause of death, coded according to the *International Statistical Classification of Diseases and Related Health Problems, Tenth Revision*, were obtained from death certifications with permission of the Ministry of Health, Labour and Welfare. In present study, we assessed all-cause mortality and deaths due to CVD [15].

### The aMED assessment

Dietary intake information was obtained through 24-h interview. The 24-h dietary recall interviews were conducted by face-to-face communication at the Mobile Examination Center (MEC). All participants were asked to recall all food and beverages (other than regular drinking water) consumed in the 24 h prior to the interview. We assessed our participants' adherence to the Mediterranean diet using the aMED score. The aMED score (total score = 18) are derived by an assigned value of "0", "1", or "2" across nine food categories (vegetables, legumes, fruits, nuts, whole grains, red and processed meats, fish, alcohol and olive oil), with higher scores indicating better

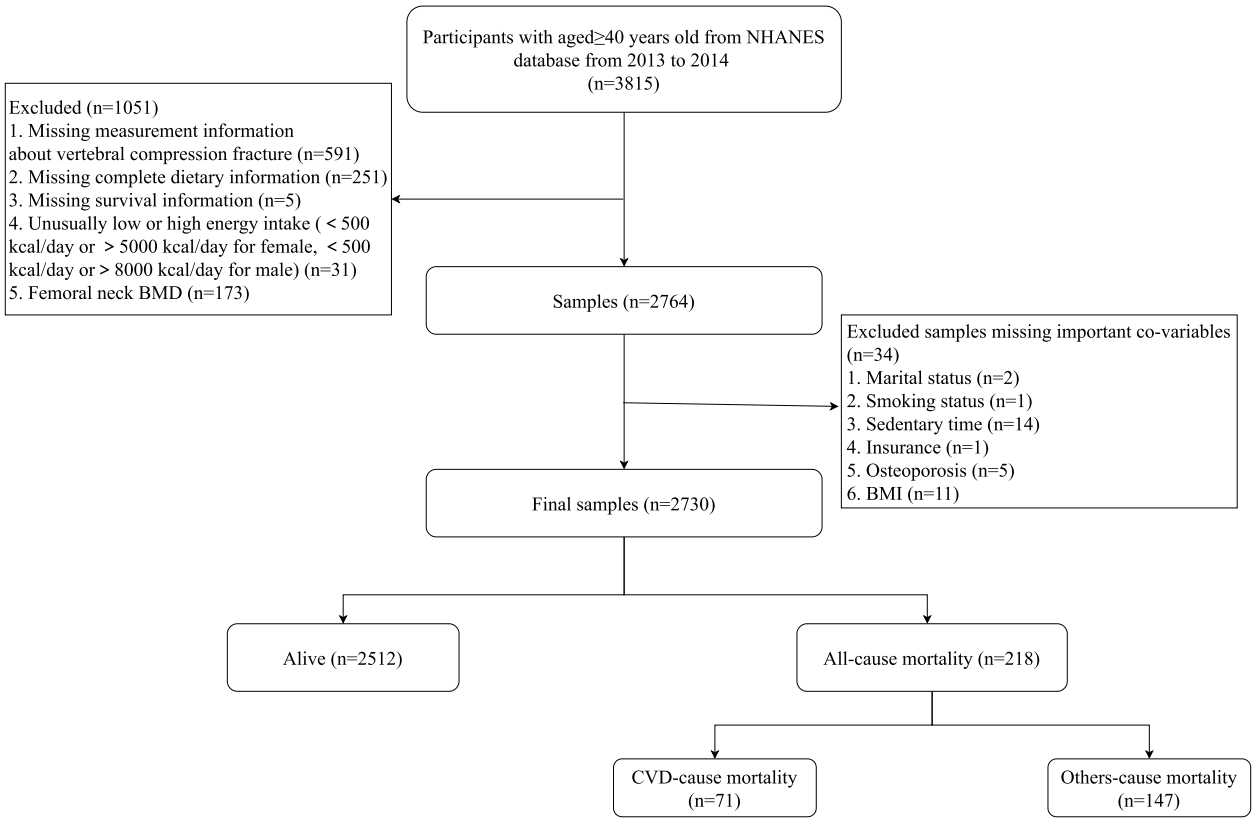


**Fig. 1** The flow chart of population screening

adherence to Mediterranean diet pattern [14]. In present study, aMED scores were divided into two groups according to the weighted median (Q1: aMED < 6; Q2: aMED ≥ 6).

### Definition of VCF

Population-based assessment of VCF can be carried out by common dual energy x-ray absorptiometry (DXA) densitometers. This method is vertebral fracture assessment (VFA) and has been used in many population settings [2].

### Potential covariates

Covariates were considered regarding demographic data, vital signs, disease history, laboratory parameters, disease severity scores, and treatments. Smoking was assessed by the question "Smoked at least 100 cigarettes in life" (yes/no). Physical activity was expressed as the metabolic equivalent (MET) and calculated as follows: physical activity (met·min/week) = recommended MET × exercise time for corresponding activities (min/day) × the number of exercise days per week (day) [16]. Hypertension was defined as systolic blood pressure (SBP) ≥ 130 mmHg, or diastolic blood pressure (DBP) ≥ 80 mmHg, or self-reported hypertension or use of antihypertensive medication [17]. Chronic kidney disease (CKD) was defined as urine albumin-to-creatinine ratio (UACR) > 30 mg/g or estimated glomerular filtration rate (eGFR) < 60 ml/min 1.73 $m^2$ [18]. CVD was assessed by the question "Ever told you had angina or heart failure/ heart attack/coronary heart disease/congestive heart failure?" (yes/no). Drinking was assessed by the question "Had at least 12 alcohol drinks/1 years?" (yes/no). Dyslipidemia was defined as total cholesterol (TC) ≥ 200 mg/dL (5.2 mmol/L), triglyceride (TG) ≥ 150 mg/dL (1.7 mmol/L), low-density lipoprotein cholesterol (LDL-C) ≥ 130 mg/dL (3.4 mmol/L), high-density lipoprotein cholesterol (HDL-C) ≤ 40 mg/dL (1.0 mmol/L), self-reported hypercholesterolemia or receiving lipid-lowering therapy [19]. Diabetes was defined as hemoglobin A1c (HbA1c) ≥ 6.5%, fasting glucose ≥ 126 mg/dL, diagnosed as diabetes by doctor, taking insulin or hypoglycemic agent [20]. Osteopenia was defined as < 0.83 gm/$m^2$ for men and < 0.76 gm/$m^2$ for women [21].

### Statistics analysis

All statistical analyses were performed by the SAS software (version 9.4, SAS Institute). Using the proc surveyfreq in SAS software, the final sample size was weighted with WTDRD1, SDMVPSU and SDMVSTRA. WTDRD1 was dietary day one sample weight. SDMVPSU refers the masked variance unit pseudo-substrate is samvstra, and the masked variance unit pseudo-primary sampling unit (PSU) is sdmvpsu. SDMVSTRA means the CI applied to assess the reliability of an estimate.

Continuous data were expressed as mean and standard error (S.E.), and the weighted T-test or F-test was used for comparison between groups. Categorical variables were described as the number and percentage [n (%)], and comparisons between groups used the $\chi^2$ test. Multivariate imputation by chained equations was used to missing data imputation. Sensitivity analysis was performed before and after missing data imputation (Table S1). The Cox proportional hazards model is a common survival analysis method used to evaluate the predictive ability of variables for survival time. The weighted univariate Cox proportional hazard models were used to screen the covariates related to all-cause and CVD-cause mortality of VCF (Table S2). Then, the mediation analysis and subgroup analysis based on weighted multivariate Cox proportional hazard models were performed to explore the moderating effect of aMED on all-cause and CVD-cause mortality of VCF, with hazard ratios (HRs) and 95% confidence intervals (CIs). In subgroup analyses based on dyslipidemia, CVD and CKD, only the association between aMED score and all-cause mortality of VCF patients was analyzed due to the small sample of CVD death. Model 1 was a crude model without adjusting covariates. Model 2 adjusted age, marital status, physical activity, CKD and CVD. Two-sided $P < 0.05$ was considered as statistically significant.

#### *Handling missing data*

To address missing data, we employed Multivariate Imputation by Chained Equations (MICE) as our primary method for imputation. This approach allows us to create multiple complete datasets by imputing missing values based on the observed data, thereby accounting for the uncertainty associated with missingness.

#### *Validation of imputed data*

To validate the imputed data, we conducted a comparison between the results obtained from the imputed datasets and those derived from a complete-case analysis, where only participants with complete data were included. Specifically, we performed the following steps:

1. Comparison of Key Outcomes: We compared the hazard ratios (HRs) and 95% confidence intervals (CIs) for all-cause and CVD-cause mortality between the imputed dataset and the complete-case analysis. This comparison allowed us to assess whether the imputation significantly altered the estimated associations.
2. Sensitivity Analysis: We also conducted a sensitivity analysis to evaluate the robustness of our findings.

This involved analyzing the imputed datasets under different imputation models and comparing the results to ensure consistency across various assumptions about the missing data.

3. Results Consistency:The results indicated that there were no notable differences in the hazard ratios for all-cause and CVD-cause mortality before and after imputation. This consistency suggests that our imputed data reliably reflect the underlying population characteristics and supports the robustness of our findings.

By explicitly stating our validation process and demonstrating the consistency of results, we aim to reassure readers about the reliability of our findings and the appropriateness of the imputation method used.

## Results

### Description of the study VCF patients

Finally, 2,730 eligible VCF patients were included. The basic characteristics and covariates of the population, stratified by survival and all-cause mortality, were presented in Table 1. Among 2,730 patients, 218 (7.99%) were all-cause death and 71 (2.60%) were CVD-cause death. The proportion of VCF patients with higher aMED scores in the all-cause mortality group was lower than in the survival group (46.47% *vs.* 53.36%). Difference was found in age, race, the level of PIR, physical activity and femoral neck BMD, smoking, the history of hypertension, diabetes, CVD, CKD, anti-osteoporosis therapy and VCF between two groups (all $P < 0.05$).

### Relationship between aMED score and all-cause and CVD-cause mortality in VCF patients

We employed two weighted Cox proportional hazard models to explore the association between aMED score and the risk of all-cause and CVD-cause mortality in VCF patients, as presented in Table 2 and Table 3. After adjusted age, marital status, PIR, smoking status, physical activity, hypertension, CVD and CKD, we found patients with VCF had higher risk of all-cause mortality compared with subjects without VCF (HR = 1.75, 95%CI: 1.13–2.73, $P = 0.041$); no significant correlation was found between aMED score and all-cause mortality ($P > 0.05$). After adjusted age, marital status, physical activity, CKD and CVD, patients with VCF were associated with the high risk of CVD-cause mortality compared with participants without VCF (HR = 2.35, 95%CI: 1.12–4.91, $P = 0.038$); no significant correlation was found between aMED score and CVD-cause mortality ($P > 0.05$).

### Joint effect of aMED and VCF on all-cause and CVD-cause mortality

The joint effect of aMED and VCF on all-cause and CVD-cause mortality was depicted in Table 4 and Table 5. After adjusted age, marital status, PIR, smoking, physical activity, hypertension, CKD and CVD in model 2, we observed patients with VCF and low aMED score (< 6) were associated with highest risk of all-cause mortality (HR = 2.27, 95%CI: 1.25–4.13, $P = 0.025$) compared with participants without VCF and high aMED score (≥ 6). After adjusted age, marital status, physical activity, CKD and CVD, the results showed that patients with VCF and low aMED score were associated with the highest risk of mortality compared with participants without VCF and high aMED score (HR = 4.25, 95%CI: 1.64–11.06, $P = 0.013$).

### Moderating effect of aMED score on all-cause and CVD-cause mortality in VCF patients

The moderating effects of aMED score on all-cause and CVD-cause mortality in VCF patients were shown in Table 6 and Table 7. We observed in lower aMED score group (< 6), patients with VCF had a high risk of all-cause mortality (HR = 2.26, 95%CI: 1.22–4.17, $P = 0.002$); while no significant association between aMED and all-cause mortality in patients with high aMED score (≥ 6). Similar results were also observed in CVD-mortality. In lower aMED group, compared to participant without VCF, patients with VCF had a high risk of CVD-cause mortality (HR = 3.31, 95%CI: 1.28–8.57, $P = 0.018$); while no significant association between aMED and CVD-cause mortality were found in patients with high aMED score (≥ 6) ($P > 0.05$). Taken together, high aMED score has a moderating effect on all-cause and CVD-cause mortality in VCF patients.

### Joint effect of aMED score on all-cause and CVD-mortality in VCF patients based on complications

Table 8 shown the joint effect of aMED score and all-cause mortality in VCF patients based on the history of dyslipidemia, CVD and CKD. After adjusted age, marital status, PIR, smoking, physical activity, hypertension, CKD and CVD, compared to the participants without VCF patients and high aMED score, those patients with VCF and low aMED score had a high risk of all-cause mortality, especially among patients with the history of dyslipidemia ($P = 0.040$), CVD ($P < 0.001$) and CKD ($P = 0.007$).

**Table 1** Characteristics of VCF patients

| Variables | Total ($N=2730$) | Survival ($N=2512$) | All-cause death ($N=218$) | Statistics | *P* |
|---|---|---|---|---|---|
| Age, years, n (%) | | | | $\chi^2=53.573$ | < 0.001 |
| < 60 | 1490(60.28) | 1445(62.49) | 45(29.54) | | |
| ≥ 60 | 1240(39.72) | 1067(37.51) | 173(70.46) | | |
| Gender, n (%) | | | | $\chi^2=0.003$ | 0.955 |
| Female | 1377(50.48) | 1279(50.46) | 98(50.71) | | |
| Male | 1353(49.52) | 1233(49.54) | 120(49.29) | | |
| Race, n (%) | | | | $\chi^2=6.576$ | 0.005 |
| Black | 535(10.01) | 489(9.93) | 46(11.26) | | |
| White | 1234(70.91) | 1096(70.26) | 138(79.91) | | |
| Others | 961(19.08) | 927(19.81) | 34(8.83) | | |
| Educational level, n (%) | | | | $\chi^2=0.035$ | 0.854 |
| Less than high school | 601(15.27) | 570(15.23) | 31(16.06) | | |
| High school or above | 2129(84.73) | 2011(84.77) | 118(83.94) | | |
| Marital status, n (%) | | | | $\chi^2=0.851$ | 0.371 |
| No | 601(15.27) | 546(15.06) | 55(18.24) | | |
| Yes | 2129(84.73) | 1966(84.94) | 163(81.76) | | |
| PIR, n (%) | | | | $\chi^2=28.244$ | < 0.001 |
| < 1 | 1650(65.53) | 1564(67.18) | 86(42.58) | | |
| ≥ 1 | 1080(34.47) | 948(32.82) | 132(57.42) | | |
| Insurance, n (%) | | | | $\chi^2=0.240$ | 0.632 |
| No | 445(13.09) | 424(13.23) | 21(11.09) | | |
| Yes | 2285(86.91) | 2088(86.77) | 197(88.91) | | |
| Smoking, n (%) | | | | $\chi^2=15.304$ | 0.001 |
| No | 1478(54.59) | 1391(55.81) | 87(37.65) | | |
| Yes | 1252(45.41) | 1121(44.19) | 131(62.35) | | |
| Drinking, n (%) | | | | $\chi^2=4.215$ | 0.058 |
| No | 754(20.89) | 687(20.46) | 67(26.85) | | |
| Yes | 1976(79.11) | 1825(79.54) | 151(73.15) | | |
| Physical activity, MET·min/week, n (%) | | | | $\chi^2=45.098$ | < 0.001 |
| < 450 | 1032(36.16) | 908(34.67) | 124(56.94) | | |
| ≥ 450 | 1698(63.84) | 1604(65.33) | 94(43.06) | | |
| Sedentary time, hours, n (%) | | | | $\chi^2=2.161$ | 0.162 |
| < 7.5 | 1400(49.05) | 1308(49.53) | 92(42.44) | | |
| ≥ 7.5 | 1330(50.95) | 1204(50.47) | 126(57.56) | | |
| Menopausal status, n (%) | | | | $\chi^2=1.567$ | 0.229 |
| No | 793(29.29) | 741(29.69) | 52(23.78) | | |
| Yes | 584(21.18) | 538(20.77) | 46(26.93) | | |
| Inapplicable (male) | 1353(49.53) | 1233(49.54) | 120(49.29) | | |
| Hypertension, n (%) | | | | $\chi^2=38.217$ | < 0.001 |
| No | 892(35.24) | 871(36.91) | 21(12.02) | | |
| Yes | 1838(64.76) | 1641(63.09) | 197(87.98) | | |
| Diabetes, n (%) | | | | $\chi^2=17.290$ | 0.001 |
| No | 2099(81.89) | 1955(82.78) | 144(69.53) | | |
| Yes | 631(18.11) | 557(17.22) | 74(30.47) | | |
| Dyslipidemia, n (%) | | | | $\chi^2=1.291$ | 0.274 |
| No | 599(22.01) | 557(22.37) | 42(16.99) | | |
| Yes | 2131(77.99) | 1955(77.63) | 176(83.01) | | |
| CVD, n (%) | | | | $\chi^2=42.471$ | < 0.001 |
| No | 2000(76.07) | 1897(77.67) | 103(53.75) | | |

**Table 1** (continued)

| Variables | Total (*N* = 2730) | Survival (*N* = 2512) | All-cause death (*N* = 218) | Statistics | *P* |
|---|---|---|---|---|---|
| Yes | 730(23.93) | 615(22.33) | 115(46.25) | | |
| CKD, n (%) | | | | $\chi^2 = 80.939$ | < 0.001 |
| No | 2441(90.46) | 2297(91.88) | 144(70.64) | | |
| Yes | 289(9.54) | 215(8.12) | 74(29.36) | | |
| Nonsteroidal anti-inflammatory agents, n( %) | | | | $\chi^2 = 3.404$ | 0.085 |
| No | 2336(84.74) | 2164(85.35) | 172(76.28) | | |
| Yes | 394(15.26) | 348(14.65) | 46(23.72) | | |
| Anti-osteoporosis therapy, n (%) | | | | $\chi^2 = 9.705$ | 0.007 |
| No | 2679(98.46) | 2469(98.64) | 210(96.00) | | |
| Yes | 51(1.54) | 43(1.36) | 8(4.00) | | |
| Femoral neck BMD, gm/cm$^2$, n (%) | | | | $\chi^2 = 8.844$ | 0.009 |
| Normal | 1420(50.18) | 1344(51.18) | 76(36.30) | | |
| Osteopenia | 1310(49.82) | 1168(48.82) | 142(63.70) | | |
| BMI, kg/m$^2$, n (%) | | | | $\chi^2 = 1.042$ | 0.362 |
| < 25 | 761(27.25) | 689(26.91) | 72(32.07) | | |
| 25–30 | 990(36.65) | 914(36.86) | 76(33.62) | | |
| ≥ 30 | 979(36.10) | 909(36.23) | 70(34.31) | | |
| Total energy, kcal, Mean ± S.E | 2080.07 ± 23.88 | 2089.73 ± 28.08 | 1945.69 ± 80.12 | t = −1.494 | 0.157 |
| aMED score, n (%) | | | | $\chi^2 = 1.760$ | 0.204 |
| < 6 | 1220(47.10) | 1099(46.64) | 121(53.53) | | |
| ≥ 6 | 1510(52.90) | 1413(53.36) | 97(46.47) | | |
| VCF, n (%) | | | | $\chi^2 = 12.511$ | 0.003 |
| No | 2581(94.66) | 2392(95.18) | 189(87.40) | | |
| Yes | 149(5.34) | 120(4.82) | 29(12.60) | | |
| Time, Mean ± S.E | 71.08 ± 1.07 | 73.04 ± 1.12 | 43.79 ± 1.82 | t = −15.926 | < 0.001 |
| Status, n (%) | | | | | |
| Alive | 2512(93.29) | 2512(100.00) | 0(0.00) | | |
| CVD-cause mortality | 71(1.97) | 0(0.00) | 71(29.40) | | |
| Others-cause mortality | 147(4.74) | 0(0.00) | 147(70.60) | | |

*S.E* standard error, *t* weighted t test, $\chi^2$ Rao-Scott Chi-square test, *VCF* vertebral compression fractures, *PIR* poverty-to-income ratio, *CVD* cardiovascular disease, *CKD* chronic kidney disease, *BMD* bone mineral density, *BMI* body mass index, *aMED* adherence to Mediterranean diet

**Table 2** Association between VCF and aMED with all-cause mortality

| | Model 1 | | Model 2 | |
|---|---|---|---|---|
| **Variables** | **HR (95%*CI*)** | ***P*** | **HR (95%CI)** | ***P*** |
| VCF | | | | |
| No | Ref | | Ref | |
| Yes | 2.65 (1.71–4.09) | < 0.001 | 1.75 (1.13–2.73) | 0.041 |
| aMED score | | | | |
| < 6 | Ref | | Ref | |
| ≥ 6 | 0.77 (0.58–1.03) | 0.200 | 0.95 (0.71–1.27) | 0.760 |

Model 1: crude model

Model 2: adjustment for age, marital status, PIR, smoking, physical activity, hypertension, CKD and CVD

*Ref* reference, *VCF* vertebral compression fractures, *aMED* adherence to Mediterranean diet

**Table 3** Association between VCF and aMED with CVD-cause mortality

| | Model 1 | | Model 2 | |
|---|---|---|---|---|
| **Variables** | **HR (95%*CI*)** | ***P*** | **HR (95%CI)** | ***P*** |
| VCF | | | | |
| No | Ref | | Ref | |
| Yes | 3.47 (1.67–7.21) | 0.004 | 2.35 (1.12–4.91) | 0.038 |
| aMED score | | | | |
| < 6 | Ref | | Ref | |
| ≥ 6 | 0.59 (0.34–1.01) | 0.082 | 0.74 (0.42–1.28) | 0.308 |

Model 1: crude model

Model 2: adjustment for age, marital status, physical activity, CKD and CVD

*Ref* reference, *HR* hazard ratio, *CI* confidence interval, *CVD* cardiovascular disease, *VCF* vertebral compression fractures, *aMED* adherence to Mediterranean diet

**Table 4** Joint effect of aMED and VCF to all-cause mortality

| Variables | Model 1 | | Model 2 | |
|---|---|---|---|---|
| | HR (95%*CI*) | *P* | HR (95%CI) | *P* |
| Joint | | | | |
| Non-VCF & aMED score ≥ 6 | Ref | | Ref | |
| Non-VCF & aMED score < 6 | 1.30 (0.95–1.77) | 0.247 | 1.02 (0.74–1.39) | 0.937 |
| VCF & aMED score ≥ 6 | 2.83 (1.48–5.42) | 0.003 | 1.39 (0.72–2.68) | 0.355 |
| VCF & aMED score < 6 | 3.18 (1.75–5.78) | 0.002 | 2.27 (1.25–4.13) | 0.025 |

Model 1: crude model

Model 2: adjustment for age, marital status, PIR, smoking, physical activity, hypertension, CKD and CVD

Non-VCF & aMED score ≥ 6 (Reference Group): This group serves as the baseline for comparison and represents individuals without VCF and with higher adherence to the Mediterranean diet

Non-VCF & aMED score < 6: Compared to the reference group, individuals without VCF but with lower adherence to the Mediterranean diet show a non-significant increase in the risk of all-cause mortality

VCF & aMED score ≥ 6: Interestingly, VCF patients with higher adherence to the Mediterranean diet do not exhibit a significantly increased risk of all-cause mortality compared to those without VCF, suggesting a potential protective effect of the diet

VCF & aMED score < 6: This group experiences the highest risk of all-cause mortality, indicating that the combination of VCF and low adherence to the Mediterranean diet is associated with the poorest outcomes

*Ref* reference, HR hazard ratio, CI confidence interval, VCF vertebral compression fractures, aMED adherence to Mediterranean diet

### Moderating effect of aMED score on all-cause mortality in VCF patients based on complications

Table 9 reports the moderating effect of aMED score on all-cause mortality in VCF patients stratified by the history of dyslipidemia, CVD and CKD. After adjusted age, marital status, PIR, smoking, physical activity, hypertension, CKD and CVD, the moderating effect of aMED score on the association of VCF patients and all-cause mortality still robust, especially in patients with the history of dyslipidemia ($P = 0.009$), CVD ($P < 0.001$) and CKD ($P < 0.001$).

### Summary table: aMED scores and mortality outcomes

To provide a clear and concise overview of the relationship between adherence to the Mediterranean diet (aMED) and mortality outcomes, we have created a summary table (Table 10). This table presents the key findings of our study, correlating aMED scores with all-cause and cardiovascular disease (CVD)-cause mortality in patients with vertebral compression fractures (VCF).

## Discussion

The Mediterranean diet (MD) is renowned for its anti-inflammatory and antioxidant properties, which are crucial in the context of bone and cardiovascular health among VCF patients. Chronic inflammation, a key factor in osteoporosis and fragility fractures, can be mitigated by the MD's rich content of bioactive compounds such as polyphenols and omega-3 fatty acids, which modulate inflammatory pathways and reduce the production of pro-inflammatory cytokines [22] Additionally, the MD's high antioxidant content, including vitamins C and E, carotenoids, and flavonoids, can neutralize reactive oxygen species (ROS), thereby protecting bone and vascular tissues from oxidative damage [23]"."The interplay

**Table 5** Joint effect of aMED and VCF to CVD-cause mortality

| Variables | Model 1 | | Model 2 | |
|---|---|---|---|---|
| | HR (95%*CI*) | *P* | HR (95%CI) | *P* |
| Joint | | | | |
| Non-VCF & aMED score ≥ 6 | Ref | | Ref | |
| Non-VCF & aMED score < 6 | 1.67 (0.92–3.02) | 0.162 | 1.26 (0.69–2.29) | 0.515 |
| VCF & aMED score ≥ 6 | 3.38 (1.00–11.36) | 0.062 | 1.58 (0.47–5.39) | 0.479 |
| VCF & aMED score < 6 | 5.65 (2.18–14.64) | 0.005 | 4.25 (1.64–11.06) | 0.013 |

Model 1: crude model

Model 2: adjustment for age, marital status, physical activity, CKD and CVD

Non-VCF & aMED score ≥ 6 (Reference Group): This group is the reference for comparison and includes individuals without VCF who have higher adherence to the Mediterranean diet

Non-VCF & aMED score < 6: Individuals without VCF but with lower adherence to the Mediterranean diet show a non-significant trend towards increased CVD-cause mortality

VCF & aMED score ≥ 6: VCF patients with higher adherence to the Mediterranean diet do not show a significantly increased risk of CVD-cause mortality, which is consistent with the all-cause mortality findings

VCF & aMED score < 6: This group has the highest risk of CVD-cause mortality, highlighting the synergistic negative effect of VCF and low aMED scores on CVD outcomes

*Ref* reference, *HR* hazard ratio, *CI* confidence interval, *CVD* cardiovascular disease, *VCF* vertebral compression fractures, *aMED* adherence to Mediterranean diet

**Table 6** The moderating effect of aMED score on all-cause mortality in VCF patients

| Variables | Model 1 | | Model 2 | |
|---|---|---|---|---|
| | HR (95%*CI*) | *P* | HR (95%CI) | *P* |
| aMED score < 6 | | | | |
| Non-VCF | Ref | | Ref | |
| VCF | 2.45 (1.34–4.50) | 0.005 | 2.26 (1.22–4.17) | 0.002 |
| aMED score ≥ 6 | | | | |
| Non-VCF | Ref | | Ref | |
| VCF | 2.85 (1.51–5.36) | 0.003 | 1.36 (0.71–2.61) | 0.371 |

Model 1: crude model

Model 2: adjustment for age, marital status, PIR, smoking, physical activity, hypertension, CKD and CVD

*Ref* reference, *HR* hazard ratio, *CI* confidence interval, *VCF* vertebral compression fractures, *aMED* adherence to Mediterranean diet

**Table 7** The moderating effect of aMED score on CVD-cause mortality in VCF patients

| Variables | Model 1 | | Model 2 | |
|---|---|---|---|---|
| | HR (95%*CI*) | *P* | HR (95%CI) | *P* |
| aMED score < 6 | | | | |
| Non-VCF | Refs | | Ref | |
| VCF | 3.36 (1.31–8.60) | 0.022 | 3.31 (1.28–8.57) | 0.018 |
| aMED score ≥ 6 | | | | |
| Non-VCF | Ref | | Ref | |
| VCF | 3.36 (1.02–10.99) | 0.061 | 1.53 (0.46–5.12) | 0.490 |

Model 1: crude model

Model 2: adjustment for age, marital status, physical activity, CKD and CVD

Ref reference, HR hazard ratio, CI confidence interval, CVD cardiovascular disease, VCF vertebral compression fractures, aMED adherence to Mediterranean diet

**Table 8** Joint effect of aMED and VCF to all-cause mortality based on complications

| Subgroup | Interaction | *P* |
|---|---|---|
| Dyslipidemia | | |
| No | Non-VCF & aMED score ≥ 6 | Ref |
| | Non-VCF & aMED score < 6 | 0.765 |
| | VCF & aMED score ≥ 6 | 0.065 |
| | VCF & aMED score < 6 | 0.511 |
| Yes | Non-VCF& aMED score ≥ 6 | Ref |
| | Non-VCF & aMED score < 6 | 0.974 |
| | VCF & aMED score ≥ 6 | 0.765 |
| | VCF & aMED score < 6 | 0.040 |
| CVD | | |
| No | Non-VCF & aMED score ≥ 6 | Ref |
| | Non-VCF & aMED score < 6 | 0.717 |
| | VCF & aMED score ≥ 6 | 0.363 |
| | VCF & aMED score < 6 | 0.376 |
| Yes | Non-VCF & aMED score ≥ 6 | Ref |
| | Non-VCF & aMED score < 6 | 0.818 |
| | VCF & aMED score ≥ 6 | 0.642 |
| | VCF & aMED score < 6 | < 0.001 |
| CKD | | |
| No | Non-VCF & aMED score ≥ 6 | Ref |
| | Non-VCFs & aMED score < 6 | 0.871 |
| | VCF & aMED score ≥ 6 | 0.390 |
| | VCF & aMED score < 6 | 0.554 |
| Yes | Non-VCF & aMED score ≥ 6 | Ref |
| | Non-VCF & aMED score < 6 | 0.965 |
| | VCF & aMED score ≥ 6 | 0.696 |
| | VCF & aMED score < 6 | 0.007 |

*Ref* reference, *VCF* vertebral compression fractures, *aMED* adherence to Mediterranean diet

between bone health, oxidative stress, and metabolic disorders such as dyslipidemia and cardiovascular disease (CVD) is complex and warrants further exploration. Elevated lipid levels in VCF patients can lead to increased adipose tissue, which secretes inflammatory factors that inhibit bone resorption and promote osteoclast differentiation, ultimately affecting bone remodeling. The MD's potential benefits on lipid health through oxidative stress and inflammation, the most common risk factor for metabolic syndrome, suggests a mechanism by which it may improve outcomes in VCF patients with comorbid dyslipidemia and CVD [24]."

In present study, we investigated the association between aMED and mortality in VCF patients. After adjusted all covariates, we observed patients with VCF patients had a high all-cause and CVD-cause mortality; patients with VCF and concomitantly poor adherence to MD had the highest risk of all-cause and CVD-cause mortality. Greater adherence to MD may has a potential moderating effect on mortality risk in patients with VCF, especially among patients with the history of dyslipidemia, CVD and CKD. From the perspective of healthy diet, this study lays a theoretical foundation for improving the prognosis of patients with VCF.

Bone was constantly remodeled, both in order to replace old and damaged bone, and to keep long-term Ca homeostasis. If Ca was cumulatively scarce skeleton over several years, this results in VCF and a higher risk of fracture. Considering the insidious onset of VCF, the number of VCF patients delay optimal treatment because of not detected in time. There was increasing clinical evidence of the mortality entailed by VCF patients [25–27]. In the past few decades, inflammation and oxidative stress associated with aging were fundamental pathogenic mechanism of aged-related bone loss and also possibly loss of muscle masa [28]. Cerullo et al. [29] reported that after the age of 50 years, a progressive decrease in bone and

**Table 9** The moderating effect of aMED score on all-cause mortality related to VCF based on complications

| Subgroup | aMED score (Outcome/Total) | HR (95%CI) | *P* |
|---|---|---|---|
| Dyslipidemia | | | |
| No | < 6 (*n* = 25/255) | 1.34 (0.22–8.17) | 0.765 |
| | ≥ 6 (*n* = 17/344) | 2.57 (0.60–11.08) | 0.158 |
| Yes | < 6 (*n* = 96/965) | 2.49 (1.29–4.80) | 0.009 |
| | ≥ 6 (*n* = 80/1166) | 1.03 (0.48–2.22) | 0.945 |
| CVD | | | |
| No | < 6 (*n* = 57/873) | 1.55 (0.61–3.98) | 0.383 |
| | ≥ 6 (*n* = 46/1127) | 1.36 (0.51–3.64) | 0.451 |
| Yes | < 6 (*n* = 64/347) | 3.48 (1.56–7.74) | < 0.001 |
| | ≥ 6 (*n* = 51/383) | 1.27 (0.54–2.96) | 0.684 |
| CKD | | | |
| No | < 6 (*n* = 78/1067) | 1.45 (0.60–3.50) | 0.476 |
| | ≥ 6 (*n* = 66/1374) | 1.34 (0.61–2.92) | 0.451 |
| Yes | < 6 (*n* = 43/153) | 3.64 (1.50–8.78) | < 0.001 |
| | ≥ 6 (*n* = 31/136) | 1.26 (0.39–4.10) | 0.685 |

*VCF* vertebral compression fractures, *aMED* adherence to Mediterranean diet

**Table 10** Summary of aMED Scores and Mortality Outcomes

| aMED Score | All-Cause Mortality | CVD-Cause Mortality |
|---|---|---|
| < 6 | HR = 2.27 (1.25–4.13), *P* = 0.025 | HR = 4.25 (1.64–11.06), *P* = 0.013 |
| ≥ 6 | HR = 1.75 (1.13–2.73), *P* = 0.041 | HR = 2.35 (1.12–4.91), *P* = 0.038 |

The table shows the hazard ratios and 95% confidence intervals for all-cause and CVD-cause mortality, adjusted for age, marital status, PIR, smoking, physical activity, hypertension, CVD, and CKD

*HR* Hazard Ratio, CI Confidence Interval

muscle by 1–2% every year. Inhibiting inflammation and reducing the generation of free radicals and oxidative stress, the rate of bone loss and muscle wasting among elderly can be reduced [29]. Adequate nutrition was vital in achieving and maintaining optimal bone mass. It was widely accepted that adequate Ca and vitamin D intake are necessary for bone healthy; however, nutritional benefits for bone went beyond these two common nutrients. Several nutrients with antioxidant properties, such as vitamin C, E, and carotenoids, as well as vitamin K, potassium, magnesium, phosphorus, protein, and fat may be required for optimal Ca balance [30]. That is to say, in order to maintain the bone density and strength, there were additional requirements for other nutrients and minerals than just Ca, likely provided by a healthy comprehensive dietary pattern. MD was considered as one of the healthiest dietary patterns owes to its unique way of diet. MD includes daily intake of fresh vegetables and fruits, nuts and seeds, whole grains, dairy products and olive oil, while the consumption of red meat as well as processed high-fat and sugary foods was restricted in this health dietary pattern [31]. This integrated healthy dietary pattern renders the MD rich in anti-inflammatory and antioxidant nutrients. This partly explains the findings of our study, stating that adherence to a healthy dietary pattern improves fracture risk and bone mineral status, as well as reduces the risk of osteoporosis.

The present study observed the moderating effect of aMED on the relationship between VCF patients and all-cause and CVD-cause mortality. Previous studies have confirmed the relationship between the MD and bone health, but few studies have focused on the prognosis of VCF patients. The favorable associations observed in present study were consistent with several previous studies but not all studies. A study of healthy women from Southern Spain suggested that there was a significantly linear trends between MD and BMD in both reproductive age and postmenopausal women [32]. Chen et al. [33] focused on the middle-aged and elderly Chinese reported that higher aMED scores were related positively and dose-dependently associated with BMD after adjustment for all potential covariates. In the EPIC study, per 1-unit increase in aMED score was related to a 7% lower incidence of hip fracture among 48,814 men and 139,981 women after 9 years of follow-up. A case–control study of 726 pairs of urban Chinese in Guangdong suggested that all greater values of the diet-quality scores were significantly associated with a similar decreased risk of hip fractures [34]. A study in adolescents also suggested that a trend of increased BMD at 13 years with greater adherence to the MD pattern was observed in boys and dietary may beyond nutrient adequacy, a limiting determinant of BMD [35]. However, Feart et al. [36] suggested that higher aMED was not associated with a decreased risk of fractures in French older persons. The widely recognized beneficial effects of MD not seem to apply to bone health in these people. Kontogianni et al. [37] aMED was not found to have any significant effect on indices of bone mass in adult women, whereas adherence to a dietary pattern close to the MD was positively associated with bone mass, suggesting potential bone-preserving properties of this pattern in adult stage. The irrelevant relationship of above studies might cause by the smaller sample sizes or discrepancies in the different methods or indexes used to evaluate the adherence to MD.

We also found the moderating effect of aMED on the relationship between VCF patients and all-cause mortality in patients with dyslipidemia, CVD and CKD. Recent studies suggested that obesity-derived metabolic alterations including dyslipidemia may also be risk factors for compromised bone health [11, 38, 39]. Elevated lipid levels in VCF patients can lead to the assimilations

of amounts of adipose in the human body, and as a vital endocrine organ, adipose can secrete inflammatory factors. High levels of inflammatory factors can inhibit bone resorption, thereby promoting osteoclast differentiation and ultimately affecting bone remodeling [39].The MD has potential benefits on lipid health through oxidative stress, inflammation (the most common risk factor for the metabolic syndrome), and modulation of gastrointestinal function [12, 40].Moreover, the existence of a possible relationship between bone and atherogenic pathways has been reported. "Excessive accumulation of osteoclasts in bone leads to bone loss, which may promote calcium deposition in coronary arteries and affect the stability of atherosclerotic plaques. In addition, the activated renin-angiotensin system promotes atherogenesis and promotes osteoclast activity leading to bone loss [41].The association between the MD and cardiovascular health has been well known. Our study suggests that adherence to a Mediterranean diet may be more important for improving outcomes in VCF patients with comorbid dyslipidemia, cardiovascular disease and CKD compared to general VCF patients.

Herein, we provided reference for the improvement of VCF patients outcome based on the relationship between aMED and all-cause and CVD-cause mortality. MD is a dietary habit rather than a structured diet and emphasizes an adequate intake of fruits, vegetables, and whole grains and also contains moderate amounts of legumes, nuts, skim milk, olive oil and some fish, as well as small amounts of red meat, salt and carbohydrates [42].For clinicians and policymakers, as well as VCF patients, it was essential to be aware of the benefits of adherence to the MD for bone health and thus improved the outcomes.Clinicians managing VCF patients should consider assessing their patients' dietary habits and providing counseling on the potential benefits of adhering to a Mediterranean diet. Specific recommendations may include increasing the consumption of fruits and vegetables, incorporating whole grains into the diet, and reducing the intake of processed meats and sugars. Additionally, clinicians may encourage moderate alcohol consumption and regular physical activity, which are also components of the Mediterranean lifestyle that could contribute to better outcomes in VCF patients. However, several limitations of this study can be identified. First, the diet intake information obtained from 24-h dietary recall interview was used to calculate the aMED score, which may not fully reflect the average diet quality of the subjects and also may be some recall bias,The use of 24-h dietary recall interviews to assess dietary intake may introduce some limitations regarding the accuracy of aMED scores. This method is subject to recall bias and may not accurately reflect an individual's long-term dietary habits. The reliance on a single day's dietary intake could lead to misclassification of participants' adherence to the Mediterranean diet, potentially affecting the reliability of our aMED scores. Future studies employing multiple recalls or food diaries over extended periods could provide a more comprehensive assessment of dietary patterns. Second, due to the limitations of the NHANES database, only 2,730 subjects underwent dual-energy X-ray BMD measurements. The sample sizes for outcomes of VCF patients and death were small as of the follow-up date of December 2019, which may cause certain bias to our results, particularly for CVD-cause mortality, the statistical power is limited, especially in subgroup analyses. This constraint affects the robustness of our findings and the generalizability of our results to other populations. We advise caution in interpreting these results and recommend that future studies with larger sample sizes and longer follow-up periods be conducted to validate our findings. Last but not least, NHANES database included representative U.S. population, so the generalizability of moderating effect of aMED on the association between VCF patients and mortality among other populations needs to be explore by further large-scale prospective studies.

The Mediterranean diet (MD) has been widely recognized for its health benefits, extending beyond the specific context of vertebral compression fractures (VCF). The diet's rich content of fruits, vegetables, whole grains, nuts, and olive oil, along with its low intake of red meat and processed foods, contributes to a range of health-promoting effects [6, 43]. These include improved cardiovascular health, reduced risk of type 2 diabetes, and enhanced cognitive function [44].

Cardiovascular Health:The MD's high content of monounsaturated fats, such as those found in olive oil, and its rich supply of antioxidants and polyphenols, have been shown to reduce inflammation and improve lipid profiles [45]. This leads to a lower incidence of cardiovascular diseases, which are a significant cause of morbidity and mortality worldwide [46].

Metabolic Syndrome and Type 2 Diabetes:The diet's emphasis on whole foods and its low glycemic index helps in the management of blood sugar levels, reducing the risk of developing type 2 diabetes [47]. This is particularly important given the increasing prevalence of metabolic syndrome and its associated complications [48].

Cognitive Function:The MD has also been associated with better cognitive function and a reduced risk of neurodegenerative diseases such as Alzheimer's disease [49]. The diet's high content of omega-3 fatty acids and antioxidants plays a crucial role in maintaining brain health and function [50].

Resilience and Overall Health:Beyond these specific health outcomes, the MD promotes overall resilience and well-being. Its anti-inflammatory and antioxidant properties help the body cope with various stressors, enhancing the body's ability to recover from illnesses and maintain optimal health [51]. By adopting the MD, patients can benefit from a holistic approach to health, which not only addresses specific conditions like VCF but also improves their overall quality of life.

In conclusion, the MD offers a comprehensive approach to health that extends beyond the management of VCF. Its benefits are multifaceted, affecting various aspects of health and well-being. This makes the MD a valuable dietary pattern for the prevention and management of a wide range of chronic diseases and conditions.

## Conclusion

Our study suggested aMED may have a moderating effect on the all-cause and CVD-cause mortality in VCF patients, particularly in patients with the history of dyslipidemia, CVD and CKD. Additional well-designed and stratified cohort studies with a range of confounding factors are required to elucidate the association between aMED and mortality among VCF patients.

### Abbreviations

| | |
|---|---|
| VCF | Vertebral compression fractures |
| BMD | Bone mineral density |
| Ca | Calcium |
| MD | Mediterranean diet |
| CVD | Cardiovascular diseases |
| aMED | Adherence to Mediterranean diet |
| NHANES | National Health and Nutrition Examination Survey |
| NCHS | National Centers for Health Statistics |
| CDC | The Centers for Disease Control and Prevention |
| MEC | Mobile Examination Center |
| DXA | Dual energy x-ray absorptiometry |
| VFA | Vertebral fracture assessment |
| MET | Metabolic equivalent |
| SBP | Systolic blood pressure |
| DBP | Diastolic blood pressure |
| CKD | Chronic kidney disease |
| eGFR | Estimated glomerular filtration rate |
| TC | Total cholesterol |
| TG | Triglyceride |
| LDL-C | Low-density lipoprotein cholesterol |
| HDL-C | High-density lipoprotein cholesterol |
| HbA1c | Hemoglobin A1c |
| S.E. | Standard error |
| HRs | Hazard ratios |
| CIs | Confidence intervals |

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s13075-025-03529-4.

Supplementary Material 1.

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### Authors' contributions

LZ designed the study, LZ, YZ wrote the manuscript, JX, SY, QW,JR, ZJ,CZ collected, analyzed and interpreted the data, XM critically reviewed, edited the manuscript, all authors read and approved the manuscript.

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### Data availability

Data of this study were extracted from the NAHNES database 2013-2014. The NHANES is conducted by the National Centers for Health Statistics (NCHS), the Centers for Disease Control and Prevention (CDC) to assess the health and nutritional status of adults and children in the United States. https://wwwn.cdc.gov/Nchs/Nhanes/ and https://wwwn.cdc.gov/nchs/nhanes/tutorials/module2.aspx.

## Declarations

### Ethics approval and consent to participate

The requirement of ethical approval for this was waived by the Institutional Review Board of Dongzhimen Hospital Beijing University of Chinese Medicine for Nationalities, because NHANES is a publicly available dataset and was approved by the NCHS Ethics Review Board. The need for written informed consent was waived by the Institutional Review Board of Dongzhimen Hospital Beijing University of Chinese Medicine for Nationalities due to retrospective nature of the study. All methods were performed in accordance with the relevant guidelines and regulations.

### Consent for publication

Not applicable.

### Competing interests

The authors declare no competing interests.

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### References

1. McCarthy J, Davis A. Diagnosis and Management of Vertebral Compression Fractures. Am Fam Physician. 2016;94(1):44–50.
2. Kutsal FY, Ergin Ergani GO. Vertebral compression fractures: Still an unpredictable aspect of osteoporosis. Turk J Med Sci. 2021;51(2):393–399.
3. Kendler DL, Bauer DC, Davison KS, Dian L, Hanley DA, et al. Vertebral Fractures: Clinical Importance and Management. Am J Med 129(2). https://doi.org/10.1016/j.amjmed.2015.09.020.
4. Arima K, Abe Y, Nishimura T, Okabe T, Tomita Y, Mizukami S, Kanagae M, Aoyagi K. Association of vertebral compression fractures with physical performance measures among community-dwelling Japanese women aged 40 years and older. BMC Musculoskelet Disord. 18(1):176. https://doi.org/10.1186/s12891-017-1531-3.
5. Vannucci L, Fossi C, Quattrini S, Guasti L, Pampaloni B, Gronchi G, Giusti F, Romagnoli C, Cianferotti L, Marcucci G, Brandi ML. Calcium Intake in Bone Health: A Focus on Calcium-Rich Mineral Waters. Nutrients. 2018;10(12):1930. https://doi.org/10.3390/nu10121930.

6. Martínez-González MA, Salas-Salvadó J, Estruch R, Corella D, Fitó M, Ros E, PREDIMED INVESTIGATORS. Benefits of the Mediterranean Diet: Insights From the PREDIMED Study. Prog Cardiovasc Dis. 58(1):50–60. https://doi.org/10.1016/j.pcad.2015.04.003.1.
7. Benetou V, Orfanos P, Pettersson-Kymmer U, Bergström U, Svensson O, Johansson I, Berrino F, Tumino R, Borch KB, Lund E, et al. Mediterranean diet and incidence of hip fractures in a European cohort. Osteoporos Int. 24(5):1587–1598. https://doi.org/10.1007/s00198-012-2187-3.
8. Quattrini S, Pampaloni B, Gronchi G, Giusti F, Brandi ML. The Mediterranean Diet in Osteoporosis Prevention: An Insight in a Peri- and Post-Menopausal Population. Nutrients. 13(2):531. https://doi.org/10.3390/nu13020531.
9. Malmir H, Saneei P, Larijani B, Esmaillzadeh A. Adherence to Mediterranean diet in relation to bone mineral density and risk of fracture: a systematic review and meta-analysis of observational studies. Eur J Nut. 57(6):2147–2160. https://doi.org/10.1007/s00394-017-1490-3.
10. Movassagh EZ, Vatanparast H. Current Evidence on the Association of Dietary Patterns and Bone Health: A Scoping Review. Advances in nutrition (Bethesda, Md.). 8(1):1–16. https://doi.org/10.3945/an.116.013326.
11. Wang Y, Wen G, Zhou R, Zhong W, Lu S, Hu C, Chai Y. Association of Nonalcoholic Fatty Liver Disease With Osteoporotic Fractures: A Cross-Sectional Retrospective Study of Chinese Individuals. Front Endocrinol. 2018;9:408. https://doi.org/10.3389/fendo.2018.00408.
12. Tosti V, Bertozzi B, Fontana L. Health Benefits of the Mediterranean Diet: Metabolic and Molecular Mechanisms. J Gerontol A Biol Sci Med Sci. 2018;73(3):318–326. https://doi.org/10.1093/gerona/glx227.
13. Rizzo NS, Jaceldo-Siegl K, Sabate J, Fraser GE. Nutrient profiles of vegetarian and nonvegetarian dietary patterns. J Acad Nutr Diet. 2013;113(12):1610–9. https://doi.org/10.1016/j.jand.2013.06.349.
14. Fan Y, Zhao L, Deng Z, Li M, Huang Z, Zhu M, Xu W. Non-linear association between Mediterranean diet and depressive symptom in U.S. adults: A cross-sectional study. Front Psychiatry. 13;936283. https://doi.org/10.3389/fpsyt.2022.936283.
15. Han H, Cao Y, Feng C, Zheng Y, Dhana K, Zhu S, Shang C, Yuan C, Zong G. Association of a Healthy Lifestyle With All-Cause and Cause-Specific Mortality Among Individuals With Type 2 Diabetes: A Prospective Study in UK Biobank. Diabetes Care. 45(2):319–329. https://doi.org/10.2337/dc21-1512.
16. Haskell WL, Lee IM, Pate RR, Powell KE, Blair SN, Franklin BA, Macera CA, Heath GW, Thompson PD, Bauman A. Physical activity and public health: updated recommendation for adults from the American College of Sports Medicine and the American Heart Association. Med Sci Sports Exerc. 2007;39(8):1423–34. https://doi.org/10.1249/mss.0b013e3180616b27.
17. Flack JM, Adekola B. Blood pressure and the new ACC/AHA hypertension guidelines. Trends Cardiovasc Med. 30(3):160–164. https://doi.org/10.1016/j.tcm.2019.05.003.
18. Levey AS, Stevens LA, Schmid CH, Zhang YL, Castro AF, 3rd, Feldman HI, Kusek JW, Eggers P, Van Lente F, Greene T, et al. A new equation to estimate glomerular filtration rate. Ann Intern Med. 150(9):604–612. https://doi.org/10.7326/0003-4819-150-9-200905050-00006.
19. Jellinger PS, Smith DA, Mehta AE, Ganda O, Handelsman Y, Rodbard HW, Shepherd MD, Seibel JA. AACE Task Force for Management of Dyslipidemia and Prevention of Atherosclerosis. American Association of Clinical Endocrinologists' Guidelines for Management of Dyslipidemia and Prevention of Atherosclerosis. Endocr Pract. 18 Suppl 1:1–78. https://doi.org/10.4158/ep.18.s1.1.
20. McClure ST, Schlechter H, Oh S, White K, Wu B, Pilla SJ, Maruthur NM, Yeh HC, Miller ER, Appel LJ. Dietary intake of adults with and without diabetes: results from NHANES 2013–2016. BMJ Open Diabetes Res Care. 8(1):e001681. https://doi.org/10.1136/bmjdrc-2020-001681.
21. Chen J, Zhu L, Yao X, Zhu Z. The association between abdominal obesity and femoral neck bone mineral density in older adults. J Orthop Surg Res. 2023;18(1):171. https://doi.org/10.1186/s13018-023-03654-3.
22. Manolagas SC, Parfitt AM. What old means to bone. Trends Endocrinol Metab. 2010;21:369–74. https://doi.org/10.1016/j.tem.2010.01.010.
23. Rizzo NS, Jaceldo-Siegl K, Sabate J, Fraser GE. Nutrient profiles of vegetarian and nonvegetarian dietary patterns. J Acad Nutr Diet. 2013;113:1610–9. https://doi.org/10.1016/j.jand.2013.06.349.
24. Esposito K, Marfella R, Ciotola M, Di Palo C, Giugliano F, Giugliano G, D'Armiento M, D'Andrea F, Giugliano D. Effect of a mediterranean-style diet on endothelial dysfunction and markers of vascular inflammation in the metabolic syndrome: a randomized trial. JAMA. 292:1440–1446 (2004).https://doi.org/10.1001/jama.292.12.1440.
25. Gutiérrez-González R, Royuela A, Zamarron A. Survival following vertebral compression fractures in population over 65 years old. Aging Clin Exp Res. 35(8):1609–1617. https://doi.org/10.1007/s40520-023-02445-4.
26. Lau E, Ong K, Kurtz S, Schmier J, Edidin A. Mortality following the diagnosis of a vertebral compression fracture in the Medicare population. J Bone Joint Surg Am. 90(7):1479–1486. https://doi.org/10.2106/JBJS.G.00675.
27. Choi HG, Lee JK, Sim S, Kim M. Mortality and Cause of Death in Patients With Vertebral Fractures: A Longitudinal Follow-Up Study Using a National Sample Cohort. Spine. 45(5):E280–E287. https://doi.org/10.1097/BRS.0000000000003264.
28. Manolagas SC, Parfitt AM. What old means to bone. Trends in endocrinology and metabolism: TEM. 21(6):369–374. https://doi.org/10.1016/j.tem.2010.01.010.
29. Cerullo F, Gambassi G, Cesari M. Rationale for antioxidant supplementation in sarcopenia. J Aging Res. 2012;316943. https://doi.org/10.1155/2012/316943.
30. Sahni S, Mangano KM, McLean RR, Hannan MT, Kiel DP. Dietary Approaches for Bone Health: Lessons from the Framingham Osteoporosis Study. Curr Osteoporos Rep. 13(4):245–255. https://doi.org/10.1007/s11914-015-0272-1.
31. Bach-Faig A, Berry EM, Lairon D, Reguant J, Trichopoulou A, Dernini S, Medina FX, Battino M, Belahsen R, Miranda G, et al. Mediterranean diet pyramid today. Science and cultural updates. Public Health Nutrition. 14(12A):2274–2284. https://doi.org/10.1017/S1368980011002515.
32. Rivas A, Romero A, Mariscal-Arcas M, Monteagudo C, Feriche B, Lorenzo ML, Olea F. Mediterranean diet and bone mineral density in two age groups of women. Int J Food Sci Nutr. 64(2):155–161. https://doi.org/10.3109/09637486.2012.718743.
33. Chen GD, Dong XW, Zhu YY, Tian HY, He J, Chen YM. Adherence to the Mediterranean diet is associated with a higher BMD in middle-aged and elderly Chinese. Sci Rep. 6.25662. https://doi.org/10.1038/srep25662.
34. Zeng FF, Xue WQ, Cao WT, Wu BH, Xie HL, Fan F, Zhu HL, Chen YM. Diet-quality scores and risk of hip fractures in elderly urban Chinese in Guangdong, China: a case-control study. Osteoporos Int. 25(8):2131–2141. https://doi.org/10.1007/s00198-014-2741-2.
35. Monjardino T, Lucas R, Ramos E, Barros H. Associations between a priori-defined dietary patterns and longitudinal changes in bone mineral density in adolescents. Public Health Nutr. 17(1):195–205. https://doi.org/10.1017/S1368980012004879.
36. Feart C, Lorrain S, Ginder Coupez V, Samieri C, Letenneur L, Paineau D, Barberger-Gateau P. Adherence to a Mediterranean diet and risk of fractures in French older persons. Osteoporos Int. 24(12):3031–3041. https://doi.org/10.1007/s00198-013-2421-7c.
37. Kontogianni MD, Melistas L, Yannakoulia M, Malagaris I, Panagiotakos DB, Yiannakouris N. Association between dietary patterns and indices of bone mass in a sample of Mediterranean women. Nutrition (Burbank, Los Angeles County, Calif.). 25(2):165–171. https://doi.org/10.1016/j.nut.2008.07.019.
38. Wong SK, Chin KY, Suhaimi FH, Ahmad F, Ima-Nirwana S. Effects of metabolic syndrome on bone mineral density, histomorphometry and remodelling markers in male rats. PloS one. 13(2):e0192416. https://doi.org/10.1371/journal.pone.0192416.
39. Wong SK, Chin KY, Suhaimi FH, Ahmad F, Ima-Nirwana S. The Relationship between Metabolic Syndrome and Osteoporosis: A Review. Nutrients. 8(6):347. https://doi.org/10.3390/nu8060347.
40. Muscogiuri G, Verde L, Sulu C, Katsiki N, Hassapidou M, Frias-Toral E, Cucalón G, Pazderska A, Yumuk VD, Colao A, Barrea L. Mediterranean Diet and Obesity-related Disorders: What is the Evidence?. Current Obesity Rep. 11(4):287–304. https://doi.org/10.1007/s13679-022-00481-1.
41. Makovey J, Macara M, Chen JS, Hayward CS, March L, Sambrook PN. High osteoporotic fracture risk and CVD risk co-exist in postmenopausal women. Bone. 52(1):120–125. https://doi.org/10.1016/j.bone.2012.09.025.
42. AlAufi NS, Chan YM, Waly MI, Chin YS, Mohd Yusof BN, Ahmad N. Application of Mediterranean Diet in Cardiovascular Diseases and Type 2 Diabetes Mellitus: Motivations and Challenges. Nutrients. 14(13):2777. https://doi.org/10.3390/nu14132777.

43. Sofi F, Cesari F, Abbate R, Gensini GF, Casini A. Adherence to Mediterranean diet and health status: meta-analysis. BMJ. 2008;337:a1344. https://doi.org/10.1136/bmj.a1344.
44. Lourida I, Soni M, Thompson-Coon J, et al. Mediterranean diet, cognitive function, and dementia: a systematic review. Epidemiology. 2013;24(4):479–89. https://doi.org/10.1097/EDE.0b013e3182944415.
45. Buckland G, González CA, Agudo A, et al. Adherence to the Mediterranean diet and risk of coronary heart disease in the Spanish EPIC cohort study. Am J Epidemiol. 2008;167(10):1159–66. https://doi.org/10.1093/aje.kwm085.
46. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation. 2002;105(9):1135–43. https://doi.org/10.1161/circulationaha.101.129715.
47. Esposito K, Chiodini P, Colao A, et al. Mediterranean diet and type 2 diabetes: a meta-analysis of randomized trials. Endocrine. 2014;45(3):391–8. https://doi.org/10.1007/s12319-014-0001-5.
48. Alberti KGMM, Zimmet P, Shaw J. The metabolic syndrome—a new worldwide definition. Lancet. 2005;366(9491):1059–62. https://doi.org/10.1016/S0140-6736(05)67402-1.
49. Scarmeas N, Luchsinger JA, Schupf N, et al. Mediterranean diet and Alzheimer disease mortality. Neurology. 2009;73(13):1081–8. https://doi.org/10.1212/WNL.0b013e3181b9cc65.
50. Morris MC, Tangney CC, Wang Y, et al. MIND diet associated with reduced incidence of Alzheimer's disease. Alzheimers Dement. 2015;11(9):1007–14. https://doi.org/10.1016/j.jalz.2014.12.010.
51. Kesse-Guyot E, Fezeu L, Andreeva VA, et al. Mediterranean diet and health status: an updated meta-analysis and a systematic review of prospective cohort studies and randomized trials. Am J Clin Nutr. 2013;98(3):662–75. https://doi.org/10.3945/ajcn.112.051794.


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