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ORIGINAL PAPER

The vital prognosis of elderly adults living in a group home in their mid-eighties

Kikue Todoroki[1] · Yoshimori Ikeya[1] · Sayato Fukui[1] · Chiharu Tanaka[1] · Kaori Sekine[1] · Ryoko Imazeki[1] · Toru Shizuma[1] · Naoto Fukuyama[1] · Hidezo Mori[1]

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Abstract The purpose of the present study is to evaluate the vital prognoses of elderly people in their mid-eighties living in a group home (GH) compared to age- and sex-matched outpatient clinic (OPC) in an observational study conducted over 6 years. We investigated the association between mortality and general, cardiac, and nutritional parameters, including eicosapentaenoic acid (EPA) in 54 GH residents (83 ± 8 years old) and 57 OPC attendees (83 ± 5 years old). Kaplan–Meier curves and Cox proportional hazard ratio analyses were used to assess the association between EPA drug administration and mortality in the GH residents and OPC attendees, respectively. The 54 GH residents had higher mortality and poorer nutritional states, as indicated by lower EPA/arachidonic acid values (median 0.20 vs 0.55, $p < 0.001$), and BMI under the condition without EPA drug administration (1800 mg daily) than did the OPC group. The significant factors that differed between survivors and deceased in the GH residents and OPC attendees were nutritional and cardiac factors. Cox proportional hazard ratio analysis confirmed that a possible determinant of the prognosis was a lower incidence of EPA drug administration and lower hemoglobin in GH. Kaplan–Meier curves and Cox proportional hazard ratio analyses revealed that EPA drug administration significantly reduced the relative mortality by 82 % in the GH residents ($p < 0.001$) but not in the OPC attendees. The vital prognosis in individuals from GHs was potentially improved by EPA drug administration, which was not the case in the OPC group; however, further prospective studies are needed.

Keywords Eicosapentaenoic acid · Geriatric medicine · Nutrition · Preventive medicine · Prognosis

Introduction

Japan will soon become a very old society, thus providing an opportunity to develop a model care system for the elderly. We are interested in achieving better vital prognoses in the elderly. Several epidemiological cohort studies have reported useful clinical indicators of survival, including functional abilities such as gait speed [1–3]. Group homes (GH) in Japan are care services where five to nine residents live together as though they were in their own home. In these facilities, elderly individuals with poor activities of daily living (ADL) or impaired cognitive function live together, and medical, nursing and nutritional care is collaboratively provided [4, 5].

However, vital prognoses and clinical aspects in GH residents have not been sufficiently reported. Recently, we reported the cause of eicosapentaenoic acid (EPA) deficiency in GHs and an improvement in the severity of supraventricular arrhythmias and changes in lipids in response to EPA therapy [6].

To elucidate the possible determinants of vital prognosis in GH residents, we investigated the association between mortality and general, nutritional, and cardiac parameters, including the polyunsaturated fatty acid levels, between GH residents and outpatient clinic (OPC) attendees.

✉ Hidezo Mori
coronary@is.icc.u-tokai.ac.jp

[1] Department of Physiology, Tokai University School of Medicine, Isehara, Kanagawa, Japan

Table 1 Characteristics of the general parameters for 57 outpatient clinic patients and 54 group home residents at baseline

	OPC ($n = 57$)	GH ($n = 54$)	p value
Observation period, months	55 (36–71)	41 (23–72)	0.111
Age, year	82 (79–87)	83 (78–90)	0.351
Sex: male/female, *n*	19/38	10/44	0.076
6-year mortality, %	25 %	50 %	0.006
BMI, kg/m^2	22.5 (18.9–25.0)	20.0 (18.1–22.0)	0.008
SBP, mmHg	131 (119–149)	120 (109–130)	0.001
DBP, mmHg	70 (60–80)	70 (60–75)	0.277
Cognitive function: categories 1/2/3	34/17/6	14/20/20	<0.001
ADL score	90 (63–100)	60 (8–85)	<0.001
Medication			
EPA drug administration	21 (37 %)	33 (61 %)	0.011
Other medications			
Antithrombotic agent	25 (44 %)	15 (28 %)	0.049
Antihypertensive agent	44 (77 %)	30 (56 %)	0.022
Diuretic or inotropic drug	11 (19 %)	20 (37 %)	0.046
Antianginal drugs	12 (21 %)	4 (24 %)	0.049
Anti-dementia agent	6 (11 %)	13 (7 %)	0.067
Hypoglycemic agent	7 (12 %)	4 (30 %)	0.247
Dyslipidemia medication	17 (30 %)	16 (2 %)	0.935
Antiarrhythmic agent	7 (12 %)	1 (7 %)	0.018

Data are presented as the median (lower quartile–upper quartile) or mean ± standard deviation. The degree of cognitive function was derived from the following Mini-Mental State Examination scores: category 1, 24–30; category 2, 10–23; and category 3, 0–9

BMI body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *ADL* activities of daily living

Methods

Subjects

We recruited 54 elderly subjects (83 ± 8, 63–97 years old) in GHs and 57 elderly patients (83 ± 8, 75–95 years old) who regularly visited the Department of Cardiology OPC at the city hospital (Tokyo, Japan) between January 2010 and December 2015. The entry criteria for this study were as follows. For GH residents, the inclusion criteria were all residents who stayed in a GH in the period described above, except for 4 patients who moved from a GH to another care facility. For OPC attendees, the entry criteria were all patients over 75 years of age (83 ± 8, 75–95 years old) who visited the Department of Cardiology at the city hospital and saw the corresponding author for more than 4 months in the period described above; their general parameters are shown in Table 1, and electrocardiograms were performed. The same physician (the corresponding author) treated all 111 enrolled patients from both the OPC and GH groups.

Protocol

In 54 GH residents and 57 OPC patients in their mid-eighties, we examined the general, nutritional, and cardiac parameters listed below (see measurement variables). To determine the possible determinants of clinical characteristics for elderly individuals in their mid-eighties in GHs, we compared the clinical findings of the 54 GH residents to those of 57 control OPC attendees using a non-parametric test. We also compared the clinical variables between 27 survivors and 27 deceased participants of the 54 GH residents and between 43 survivors and 14 deceased participants of the 57 OPC attendees using a non-parametric test. At nearly the midpoint of the observation period (2013), the physician extended EPA drug therapy (1800 mg daily) using a criterion of EPA/AA < 0.465 [7]. The effects of EPA drug administration on vital prognosis were evaluated by Kaplan–Meier curves and Cox proportional hazard ratios between the subjects with and without EPA drug treatment in the GH residents and OPC attendees, respectively.

The Institutional Review Board for Clinical Research of Tokai University (Hospital) approved this study.

Measurement of variables

General parameters, blood chemistry data and cardiac parameters

The general parameters included the body mass index (BMI), systolic blood pressure (SBP), diastolic blood pressure (DBP), and cognitive function, as evaluated using

the Mini-Mental State Examination (MMSE), which was divided into 3 levels (MMSE 24–30:1, MMSE 10–23:2, and MMSE 0–9:3). ADL and medications (including dosing information), such as the EPA drug (EPADEL, MOCHIDA PHARMACEUTICAL CO., LTD. Tokyo, Japan), antithrombotic agents, antihypertensive agents, diuretics or inotropic drugs, antianginal drugs, anti-dementia agents, hypoglycemic agents, dyslipidemia medications and antiarrhythmic agents, were recorded. Blood chemistry data included the serum levels of total protein (TP); albumin (Alb); hemoglobin (Hb); unsaturated fatty acid, EPA, docosahexaenoic acid (DHA), and arachidonic acid (AA); lipid [low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), and triglycerides]; glycosylated hemoglobin (HbA1c) values; and serum and other blood sample data, including renal function parameters [blood urea nitrogen (BUN) and serum creatinine (Cre)], liver function parameters [serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT)], and serum level of N-terminal pro-brain natriuretic peptide (NT-proBNP). Cardiac parameters included the cardiothoracic ratio (CTR) on a chest-X ray, severity of supraventricular and ventricular arrhythmias (SVA and PVC) and 12-lead electrocardiogram (ECG) findings, such as left ventricular hypertrophy, left atrial overload, left axis deviation, right axis deviation, atrial fibrillation, and left and right bundle branch blocks.

The blood chemistry data, including the TP, Alb, Hb, fatty acids, lipids, HbA1c, and both renal and liver function, and NT-proBNP, were analyzed using a previously described method [23]. The severity of SVA and PVC was examined on a Holter electrocardiogram. We defined the SVA severity based on the following 6 categories: 1, no arrhythmia; 2, supraventricular extrasystole (a single or couplet); 3, 3–29 supraventricular tachycardia (SVT); 4, $\geq$ 30 SVT; 5, paroxysmal atrial fibrillation (Af); and 6, sustained Af. The PVC severity was classified as follows: 1, no arrhythmia; 2, a single arrhythmia; 3, $\geq$ couplet ventricular premature contraction (PVC); 4, 3–29 PVC; 5, $\geq$ 30 PVC, and 6, ventricular fibrillation.

Statistical analyses

We used the SPSS software (version 23, IBM, Armonk, New York, USA) for all statistical analyses. The chi-squared test was used to compare the categorical values. The Mann–Whitney *U* test was used to compare continuous variables.

To determine the EPA drug therapy for predicting the long-term prognosis in GH residents, the Kaplan–Meier curves and Cox proportional hazard ratio were applied in the GH and OPC groups, respectively.

Results

Comparison between the GH residents and OPC attendees

As shown in Tables 1, 2 and 3, comparing the 54 GH residents and 57 OPC attendees, the elderly in the GH group exhibited increased mortality ($p < 0.01$) as well as poor nutritional states, as indicated by EPA/AA ($p < 0.01$), lower BMI ($p < 0.01$), and HbA1c ($p < 0.01$), under the condition without EPA drug administration. In addition to these parameters, lower SBP ($p < 0.01$), impaired cognitive function ($p < 0.01$), and reduced ADL scores ($p < 0.01$) were noted in GH residents compared with OPC attendees.

Comparison of the clinical findings between those who survived and deceased subjects in the GH residents and OPC attendees

The significant factors between survivors and deceased in the GH residents and OPC attendees were nutritional and cardiac factors (Tables 4, 5, 6). Mann–Whitney analysis showed that NT-proBNP was significantly increased in deceased OPC and GH patients. Lower BMI, left axis deviation and an increased incidence of atrial fibrillation were noted in the deceased OPC patients. Reduced TP, reduced Alb, increased HbA1c, reduced Hb, an increased incidence of left atrial overload and minimal changes in DBP were noted in the deceased GH patients who did not receive EPA drug administration (Tables 4, 5, 6). Cox proportional hazard ratio analysis revealed that in addition to EPA drug treatment, the predictive factors for vital prognosis were Hb in the GH residents and a lower BMI and higher incidence of atrial fibrillation in the OPC attendees (Table 7). The EPA levels under the condition without EPA drug administration were not significantly different between the survivors and deceased in both the GH residents and OPC attendees.

Effects of EPA drug administration on vital prognosis in the GH residents and OPC attendees

As shown in Fig. 1, the physician extended EPA drug administration in the GH group in 2013 (from 17 to 88 % of GH residents as a cumulative administration percentage) using the criterion of EPA/AA < 0.465 reported by Ikeya et al. [7]. As shown in Fig. 2a, Kaplan–Meier curves in the GH residents showed that mortality was significantly lower in the residents given EPA than in those without EPA (18 vs 100 %, $p < 0.001$), indicating a relative reduction of 82 %. In contrast, Kaplan–Meier curves in OPC attendees revealed no significant differences in the attendees with

Table 2 Characteristics of blood chemistry data for 57 outpatient clinic patients and 54 group home residents at baseline

	OPC (n = 22–53)	n	GH (n = 38–46)	n	p value
TP, g/dl	6.8 (6.6–7.1)	52	6.8 (6.3–7.3)	46	0.895
Alb, g/dl	4.1 (3.9–4.2)	22	3.7 (3.3–4.1)	46	0.037
Hb, g/dl	12.8 (11.2–13.7)	53	11.7 (10.4–12.8)	46	0.049
EPA, μg/ml	79.3 (46.9–109.4)	51	34.9 (25.8–56.2)	38	<0.001
DHA, μg/ml	145.8 (116.5–168.7)	51	123.8 (97.3–151.8)	38	0.038
AA, μg/ml	152.1 (131.1–185.1)	51	183.3 (157.8–230.1)	38	0.003
EPA/AA	0.55 (0.32–0.72)	51	0.20 (0.14–0.26)	38	<0.001
LDL-C, mg/dl	107 (94–134)	53	111 (96–133)	41	0.49
HDL-C, mg/dl	54 (47–65)	53	49 (44.5–55.5)	41	0.051
TG, mg/dl	98 (60–136)	53	96 (72.8–119.3)	42	0.805
HbA1c, %	5.8 (5.6–6.2)	53	5.4 (5.1–5.7)	45	<0.001
BUN, mg/dl	17.5 (14.65–23.45)	53	16.8 (13.7–21.1)	46	0.507
CRE, mg/dl	0.74 (0.63–0.92)	53	0.69 (0.55–0.92)	46	0.200
AST, U/l	22 (18–26)	52	19 (17–23)	45	0.032
ALT, U/l	16 (12–19)	52	12 (11–18)	45	0.037
NT-proBNP, pg/ml	252 (116–807)	49	168 (110–446)	46	0.199

Data are presented as the median values (lower quartile–upper quartile)

TP total protein, *Alb* albumin, *Hb* hemoglobin, *EPA* eicosapentaenoic acid, *DHA* docosahexaenoic acid, *AA* arachidonic acid, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *TG* triacylglycerols, *HbA1c* glycohemoglobin, *BUN* blood urea nitrogen, *CRE* creatinine, *AST* aspartate aminotransferase, *ALT* alanine transaminase, *NT-proBNP* N-terminal brain natriuretic peptide

Table 3 Characteristics of cardiac parameters for 57 outpatient clinic patients and 54 group home residents at baseline

	OPC	GH	p value
Chest X-ray	n = 55	n = 35	
CTR, %	52.2 (48.2–57.0)	53.0 (47.7–55.5)	0.015
Holter electrocardiogram	n = 40	n = 30	
PVC degree	2.2 ± 0.0853	1.67 ± 1.03	0.904
SVA degree	3.23 ± 1.23	2.93 ± 0.98	0.155
Electrocardiogram	n = 57	n = 50	
Left ventricular hypertrophy	17 (31 %)	6 (14 %)	0.035
Left atrial overload	15 (31 %)	16 (34 %)	0.480
Left axis deviation	11 (19 %)	16 (32 %)	0.058
Right axis deviation	2 (4 %)	1 (2 %)	0.633
Atrial fibrillation	9 (16 %)	3 (6 %)	0.108
Supraventricular extrasystole	6 (11 %)	6 (12 %)	0.820
Ventricular extrasystole	4 (7 %)	2 (4 %)	0.494
Left bundle branch block	1 (2 %)	0 (0 %)	0.345
Right bundle branch block	3 (5 %)	8 (16 %)	0.039

Data are presented as the median values (lower quartile–upper quartile) or mean ± standard deviation

CTR cardiothoracic ratio, *PVC* premature ventricular contraction, *SVA* supraventricular arrhythmia

and without EPA (24 vs 25 %, $p = 0.658$). There was no difference in the plasma EPA levels at baseline between the subjects with and without EPA drug administration in both groups ($p = 0.734$ in GH residents and $p = 0.259$ in OPC attendees, respectively; data not shown). Again, Cox proportional hazard ratio analysis confirmed that EPA drug administration was a significant predictive factor of long-term prognosis in GH residents (Table 7).

Discussion

This study showed that GH residents had higher mortality and poorer nutritional states, as indicated by the lower EPA/AA and BMI compared to the OPC attendees. Cox proportional hazard ratio analysis confirmed that the significant factors between survivors and deceased in GH residents and OPC attendees were essentially nutritional

Table 4 Characteristics of the general parameters for survivors and deceased in 57 outpatient clinics and 54 group home residents at baseline

	OPC ($n = 57$)			GH ($n = 54$)		
	Survivors ($n = 43$)	Deceased ($n = 14$)	p value	Survivors ($n = 27$)	Deceased ($n = 27$)	p value
Observation period, months	43 (62–72)	8 (27–50)	<0.001	72 (40–72)	28 (13–41)	<0.001
Age, years	78 (82–87)	80 (82–86)	0.773	82 (76–89)	86 (79–92)	0.171
Sex: male/female, *n*	13/30	6/8	0.388	4/23	6/21	0.488
BMI, kg/m^2	19.6 (22.9–25.2)	17.8 (19.1–21.9)	0.020	20 (18.2–23.3)	20 (18.1–21.4)	0.350
SBP, mmHg	120 (135–150)	110 (121–143)	0.145	120 (110–134)	116 (100–130)	0.109
DBP, mmHg	65(73–80)	53(69–75)	0.177	70(66–80)	63(53–74)	0.007
Cognitive function: categories 1/2/3	24/13/6	10/4/0	0.302	5/12/10	9/8/10	0.379
ADL score	55 (90–100)	64 (85–100)	0.856	60 (15–90)	65 (0–85)	0.917
Medication						
EPA drug administration	16 (37 %)	5 (36 %)	0.920	27 (100 %)	6 (22 %)	<0.001
Other medications						
Antithrombotic agent	16 (37 %)	9 (64 %)	0.076	10 (37 %)	5 (19 %)	0.129
Antihypertensive agent	34 (79 %)	10 (71 %)	0.554	16 (59 %)	14 (52 %)	0.584
Diuretic or inotropic drug	6 (14 %)	5 (36 %)	0.073	9 (33 %)	11 (41 %)	0.573
Antianginal drugs	8 (19 %)	4(29 %)	0.427	2 (7 %)	2 (7 %)	1.000
Anti-dementia agent	5 (12 %)	1(7 %)	0.635	9 (33 %)	4 (15 %)	0.111
Hypoglycemic agent	6 (14 %)	1(7 %)	0.500	1 (4 %)	3 (11 %)	0.299
Dyslipidemia medication	13 (30 %)	4 (29 %)	0.906	10 (37 %)	6 (22 %)	0.233
Antiarrhythmic agent	4 (9 %)	3 (21 %)	0.230	1 (4 %)	0 (0 %)	0.313

Data are presented as the median (lower quartile–upper quartile) or mean ± standard deviation

The degree of cognitive function was derived from the Mini-Mental State Examination score, as follows: category 1, 24–30; category 2, 10–23; and category 3, 0–9

OPC outpatient clinic, *GH* group home, *BMI* body mass index, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *ADL* activities of daily living

and cardiac factors. Additionally, EPA drug administration drastically reduced the relative mortality in GH residents but not in OPC attendees.

The 54 GH residents exhibited increased mortality ($p < 0.01$) and poorer nutritional states, as indicated by the EPA/AA, lower BMI and HbA1c, with no EPA drug administration (Tables 1, 2, 3). The Cox proportional hazard ratio analysis followed by the Mann–Whitney *U* test between survivors and deceased subjects in the GH residents and OPC attendees revealed that a lack of EPA drug administration and a lower Hb level were possible determinants of poor vital prognosis in the GH residents. Lower BMI and higher incidence of atrial fibrillation were observed in the OPC attendees (Tables 4, 5, 6, 7). Kaplan–Meier curves showed that EPA drug administration could reduce the mortality in GH residents (Fig. 2a) but not in the OPC attendees (Fig. 2b). All 21 of the patients who did not receive EPA died within the 72-month observation period. Cox proportional hazard ratio analysis, followed by Kaplan–Meier curve analysis, confirmed that EPA drug administration was a significant predictive factor of long-term prognosis in GH residents (Fig. 2a; Table 7).

Fried et al. reported the overall 5-year mortality in the elderly > 65 years of age was 12 % (annual mortality of 2.4 %) [8]. The annual mortalities increased with age as follows: 3.48 % for 75- to 79-year-olds, 5.45 % for 80- to 84-year-olds and 9.66 % for > 85-year-olds. In Japan, the annual mortalities of the elderly groups in 2014 were 2.51 % for those who were 75–79 years of age, 4.55 % for those 80–84 years of age, and 8.18 % for those > 85 years of age [9]. The mortality rates of home-care residents and community residents were 21.5 and 3.9 %, respectively [10].

A lower BMI, Hb, and Alb; hospitalization; and living in a nursing home have been reported as possible risk factors for high mortality in the elderly [7, 11, 12]. Various cardiac abnormalities increased mortality of the elderly [8], who often died from multifactorial diagnoses [13]. We defined acute death as sudden deterioration in life quality within several days in the present study. Fifty-nine percent of the deaths (24 of 41) were attributed to acute deaths. The nine acute deaths in the OPC patients included 2 dissecting aortic aneurysms, 3 strokes, 1 pulmonary embolism and 1 acute myocardial infarction. The remaining 2 patients died

Table 5 Characteristics of a blood test for survivors and deceased in 57 outpatient clinics and 54 group home residents at baseline

	OPC ($n = 53$)					GH ($n = 46$)				
	Survivors ($n = 18$–40)	n	Deceased ($n = 4$–13)	n	p value	Survivors ($n = 20$)	n	Deceased ($n = 18$–26)	n	p value
TP, g/dl	6.8 (6.6–7.1)	39	6.9 (6.7–7.3)	13	0.289	7.0 (6.7–7.6)	20	6.5 (6.2–7.1)	26	0.014
Alb, g/dl	4.1 (3.9–4.3)	18	4.0 (3.2–4.1)	4	0.227	4.0 (3.6–4.3)	20	3.6 (3.3–3.9)	26	0.043
Hb, g/dl	13.0 (11.3–13.7)	40	12.3 (10.6–13.6)	13	0.549	12.3 (11.5–13.6)	20	11.4 (10.2–12.3)	26	0.034
EPA, μg/ml	82.5 (46.2–112.5)	38	76.6 (59.2–101.9)	13	0.681	34.6 (27.3–57.8)	20	35.6 (20.9–56.2)	18	0.828
DHA, μg/ml	139.4 (115.0–170.5)	38	152.8 (117.0–170.7)	13	0.812	126.7 (109.7–146.0)	20	112.2 (81.0–164.3)	18	0.478
AA, μg/ml	151.5 (130.9–194.2)	38	163.5 (126.5–175.5)	13	0.795	193.1 (141.0–240.6)	20	183.3 (164.6–204.6)	18	0.762
EPA/AA	0.56(0.31–0.75)	38	0.51 (0.37–0.64)	13	0.673	0.20 (0.17–0.28)	20	0.19 (0.14–0.25)	18	0.426
LDL-C, mg/dl	109(95–133)	40	99 (89–140)	13	0.788	113 (96–134)	20	108 (97–138)	21	0.824
HDL-C, mg/dl	55(46–65)	40	52 (46–64)	13	0.804	52 (45–59)	20	48 (44–54)	21	0.354
TG, mg/dl	102(59–138)	40	90 (69–108)	13	0.379	106 (82–125)	20	88(70–115)	22	0.241
HbA1c, %	5.7 (5.6–6.1)	40	5.9 (5.7–6.9)	13	0.289	5.3 (4.9–5.6)	20	5.6 (5.2–5.8)	25	0.03
BUN, mg/dl	18.0 (14.5–24.1)	40	16.9 (15.6–22.2)	13	0.926	15.1 (12.8–18.7)	20	18.8 (13.9–24.4)	26	0.066
CRE, mg/dl	0.73 (0.62–0.86)	40	0.86 (0.61–1.14)	13	0.420	0.69 (0.56–0.84)	20	0.71 (0.54–1.02)	26	0.649
AST, U/l	22 (18–28)	39	22 (19–24)	13	0.758	20 (18–23)	20	19 (16–23)	25	0.341
ALT, U/l	16 (12–20)	39	16 (13–17)	13	0.656	12 (11–17)	20	13 (10–20)	25	0.872
NT-proBNP, pg/ml	194 (100–471)	38	1064 (579–5809)	11	0.003	121 (74–159)	20	272 (160–865)	26	0.001

Data are presented as the median values (lower quartile–upper quartile)

TP total protein, *Alb* albumin, *Hb* hemoglobin, *EPA* eicosapentaenoic acid, *DHA* docosahexaenoic acid, *AA* arachidonic acid, *LDL-C* low-density lipoprotein cholesterol, *HDL-C* high-density lipoprotein cholesterol, *TG* triacylglycerols, *HbA1c* glycohemoglobin, *BUN* blood urea nitrogen, *CRE* creatinine, *AST* aspartate aminotransferase, *ALT* alanine transaminase, *NT-proBNP* N-terminal brain natriuretic peptide

Table 6 Characteristics of cardiac parameters for survivors and deceased in 57 outpatient clinics and 54 group home residents at baseline

	OPC ($n = 57$)			GH ($n = 54$)		
	Survivors (n = 30–43)	Deceased ($n = 8$–14)	p value	Survivors ($n = 15$–27)	Deceased ($n = 12$–27)	p value
Chest X-ray	$n = 41$	$n = 14$		$n = 15$	$n = 20$	
CTR, %	51.7 (48.0–55.7)	54.9 (49.4–61.5)	0.065	52.0 (45.6–55.1)	53.5 (51.4–56.7)	0.122
Holter electrocardiogram	$n = 30$	$n = 10$		$n = 18$	$n = 12$	
PVC degree	2.19 ± 0.90	2.17 ± 0.83	0.747	1.56 ± 0.86	1.83 ± 1.27	0.692
SVA degree	3.00 ± 1.05	3.03 ± 1.07	0.259	2.78 ± 0.88	3.17 ± 1.12	0.415
Electrocardiogram	$n = 43$	$n = 14$		$n = 27$	$n = 27$	
Left ventricular hypertrophy	12 (29 %)	5 (38 %)	0.500	4 (17 %)	2 (10 %)	0.485
Left atrial overload	12 (30 %)	3 (38 %)	0.676	4 (17 %)	12 (50 %)	0.018
Left axis deviation	5 (12 %)	6 (43 %)	0.010	9 (36 %)	7 (28 %)	0.544
Right axis deviation	1 (2 %)	1 (7 %)	0.395	0 (0 %)	1 (4 %)	0.312
Atrial fibrillation	3 (7 %)	6 (43 %)	0.001	2 (8 %)	1 (4 %)	0.552
Supraventricular extrasystole	4 (9 %)	2 (14 %)	0.598	2 (8 %)	4 (16 %)	0.384
Ventricular extrasystole	4 (9 %)	0 (0 %)	0.237	1 (4 %)	1 (4 %)	1.000
Left bundle branch block	1 (2 %)	0 (0 %)	0.565	0 (0 %)	0 (0 %)	–
Right bundle branch block	2 (5 %)	1 (7 %)	0.717	3 (12 %)	5 (20 %)	0.44

Data are presented as the median values (lower quartile–upper quartile) or mean ± standard deviation

CTR cardiothoracic ratio, *PVC* premature ventricular contraction, *SVA* supraventricular arrhythmia

Table 7 The possible determinants of vital prognosis in the group home residents and outpatient clinic attendees by Cox proportional hazard analysis

	Hazard ratio (95 % CI)	*p* value
Group home residents		
EPA drug administration	0.058 (0.017–0.193)	<0.001
Hb, g/dl	0.608 (0.425–0.869)	0.006
Left atrial overload	1.906 (0.776–4.683)	0.16
Outpatient clinic attendees		
EPA drug administration	1.29 (0.417–3.988)	0.658
BMI	0.798 (0.677–0.941)	0.007
Atrial fibrillation	7.99 (2.609–24.463)	<0.001

Cox proportional hazard analysis was used to assess the association between EPA drug administration and mortality, adjusting for other factors that seemed to be related to prognosis in group home residents and outpatient clinic attendees

CI confidence interval, *BMI* body mass index, *Hb* hemoglobin

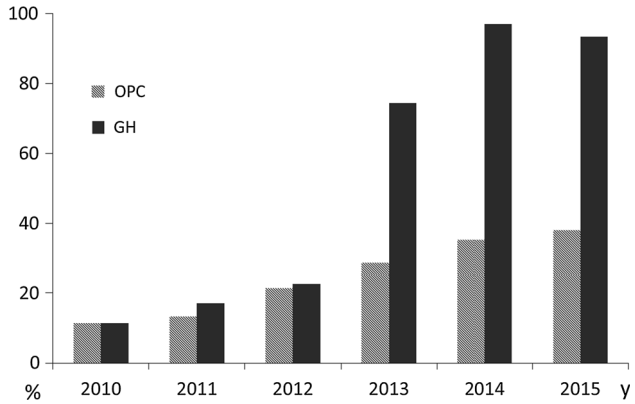

Fig. 1 Accumulated proportions of EPA drug administration. *OPC* outpatient clinic, *GH* group home. The *hatched bars* indicate OPC, and the *black bars* indicate GH

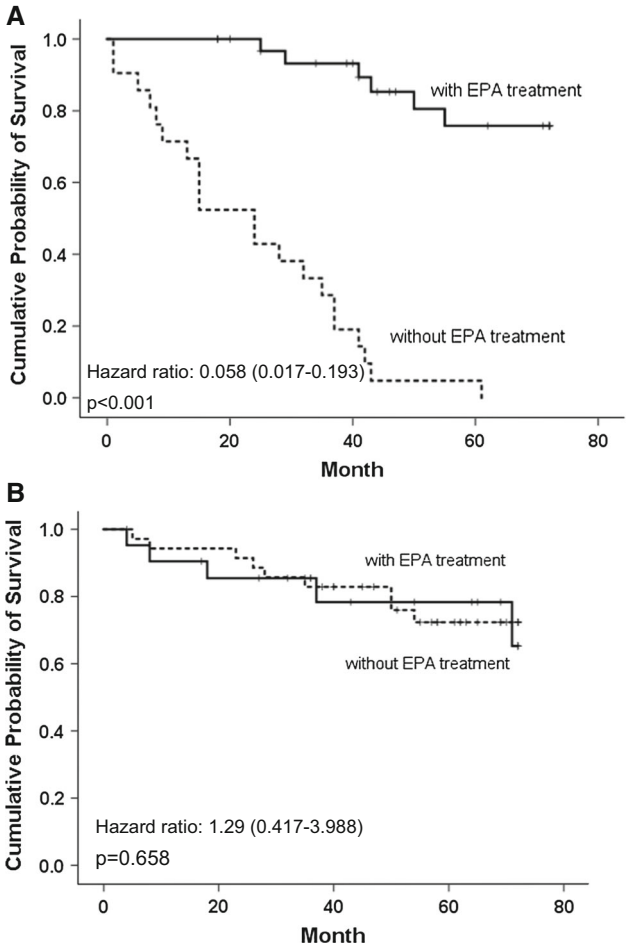

Fig. 2 Survival function by Cox proportional hazard ratio analysis between the subjects with and without EPA drug administration. **a** Fifty-four group home residents. **b** Fifty-seven outpatient clinic attendees

suddenly. Five chronic deaths in the OPC patients included 4 chronic congestive heart failure cases and 1 liver disease case. Fifteen acute deaths in GH residents included 2 pneumonias, 2 acute congestive heart failures, and 11 sudden deaths. Twelve chronic deaths in the GH residents included 4 chronic congestive heart failures, 2 pneumonias, and 2 senile decay cases with 4 unknown causes of death. Four of 41 people who died had had various cancers; however, their cancers were not the primary cause of their death.

EPA drug treatment reduced the major coronary events for patients with a history of coronary artery disease [14] and stroke recurrence [15]. All-cause mortality reduction by EPA [16, 17] or EPA and DHA [18] has been reported in a few prospective studies. N-3 fatty acids exerted a preventive effect on arrhythmia and sudden cardiac death [19, 20]. A randomized study reported that n-3 fatty acids reduced the heart rate variability, which is related to sudden death [21, 22].

One hypothesis generated from our results and the previous reports is that the association between cardiac overload and poor nutritional state, including possibly EPA deficiency, might induce sudden cardiac deterioration in the elderly. EPA deficiency in the vascular endothelium and platelets might enhance thrombogenesis [23, 24]. Furthermore, a lack of unsaturated fatty acids in the lipid bi-layers of the atrial and ventricular endocardia might cause arrhythmogeneicity [23]. Transient tachyarrhythmias in patients with hypertensive heart disease or ischemic heart disease can induce a transient reduction in the cardiac output, which possibly causes acute circulatory deterioration. We recently reported that plasma EPA was low in

other types of elderly facilities, which was probably due to the low incidence of raw fish consumption and EPA supplementation ameliorated SVAs [6]. Low EPA levels in the elderly subjects among the GH residents probably enhanced the risk of acute cardiovascular events, as described above. There was no difference in EPA/AA between survivors and non-survivors in the group home patients at baseline. Therefore, EPA drug administration reduced the risk of such acute cardiovascular events. In contrast, OPC patients with higher EPA/AA levels at baseline had a lower risk of acute cardiovascular events than did the GH residents.

Our study has several limitations. First, it was a cross-sectional study with a small sample size. Second, there was a time lag in the observation period between EPA treatment, which primarily occurred in the later 3 years in the treatment group and in the earlier 3 years in the non-treatment group. Third, we did not perform autopsies to determine the causes of death.

Conclusion

The vital prognoses of elderly GH residents in their mid-eighties was potentially improved by EPA drug administration. Further prospective studies are required to confirm the role of EPA in vital prognosis.

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Compliance with ethical standards

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

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