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Christa Meisinger, Timo Schmitz, Philip Raake, Jakob Linseisen

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Admission plasma levels of fatty acids and kidney function in patients with ST-segment elevation myocardial infarction

Christa Meisinger [a,*], Timo Schmitz [a], Philip Raake [b], Jakob Linseisen [a]

[a] Epidemiology, Medical Faculty, University of Augsburg, 86156 Augsburg, Germany
[b] University Hospital Augsburg, Department of Cardiology, Respiratory Medicine and Intensive Care, 86156 Augsburg, Germany

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ABSTRACT

Background: Prior studies suggested a link between polyunsaturated fatty acids (PUFAs) and the development of chronic kidney disease (glomerular filtration rate [GFR] < 60 ml/min/1.73 m^2), but so far there are no studies on the association between PUFAs and kidney function in patients with acute myocardial infarction (AMI). We aimed to investigate whether and how plasma fatty acids levels at admission were associated with kidney function in patients with acute ST-elevation myocardial infarction (STEMI).

Methods: This study was based on data from 717 patients with STEMI aged 29 to 98 years admitted to the University Hospital Augsburg between May 2009 and July 2013. In arterial blood samples taken from these patients immediately after admission a panel of plasma fatty acids was measured by a high-throughput nuclear magnetic resonance spectroscopy platform (Nightingale Health, Finland). To examine the association between the fatty acid groups (exposure) and kidney function (continuous eGFR; outcome), linear regression models were calculated. P values were FDR-adjusted.

Results: In multivariable analyses the concentrations of total PUFAs, total omega-6 fatty acids, linoleic acid, and docosahexaenoic acid (DHA) were positively associated with serum eGFR values at admission. These findings were supported by a significantly positive association between these fatty acids in relation to total fatty acids (except for the ratio DHA/total fatty acids). While the concentration of monounsaturated fatty acids (MUFAs) was not associated with eGFR, a significantly inverse association was observed between the ratio MUFAs/total fatty acids and kidney function.

Conclusions: PUFAs, particularly omega-6 fatty acids, seem to be associated with kidney function in patients with AMI. Given the prognostic importance of renal function in coronary heart disease/AMI patients, further research on interventions to minimize worsening renal function and protect kidney function in this patient group is warranted.

1. Introduction

Worldwide, chronic kidney disease (CKD), defined by a glomerular filtration rate (GFR) < 60 ml/min/1.73 m^2) and characterized by damage to kidney structure and function, is becoming a major public health problem with the risk of kidney failure requiring dialysis or transplantation [1,2]. As study on the prevention of end-stage renal and vascular disease in Europe has shown, up to 12 % of the adult population suffer from kidney dysfunction [3]. In association with increasing age and risk factors as well as comorbidities that contribute to CKD, such as diabetes mellitus, hypertension and cardiovascular disease, the prevalence of CKD is still on the rise [1]. According to the European Kidney Health Alliance CKD is predicted to be the fifth leading cause of death worldwide by 2040 [4].

It is therefore important to identify risk factors and biomarkers for the development of CKD to implement preventive measures. Prior studies suggested that there may be a link between polyunsaturated fatty acids (PUFAs) and the development of CKD [5]; the associations were investigated in some cross-sectional and longitudinal observational studies [6–9]. Also, dietary components rich in higher PUFAs, mainly including omega-3 and omega-6 fatty acids, were found to be associated with better kidney function [10,11].

Subjects with CKD are at high risk of cardiovascular events and it is estimated that ischemic heart disease occurs in 30 %–40 % of them [12].

* Corresponding author at: Epidemiology, Medical Faculty, University of Augsburg, University Hospital Augsburg, Stenglinstraße 2, 86156 Augsburg, Germany.
E-mail address: christine.meisinger@med.uni-augsburg.de (C. Meisinger).

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Moreover, patients with acute myocardial infarction (AMI) often suffer from CKD associated with poor short- and long-term outcomes [13]. In a recent study the association between fatty acids and kidney function in patients after myocardial infarction was investigated [14] but so far there are no studies on this topic conducted in patients with AMI at the time of the acute event. We therefore aimed to investigate whether and how plasma fatty acids levels at admission were associated with kidney function in patients with acute ST-elevation myocardial infarction (STEMI).

2. Materials and methods

2.1. Study population

This study was based on data from the population-based Augsburg Myocardial Infarction Registry, Germany. The registry is running since 1984, when it was established as part of the MONICA-project (Monitoring Trends and Determinants in Cardiovascular disease). Between 1996 and 2020 it has been operated as KORA (Cooperative Health Research in the Augsburg Region) Myocardial Infarction Registry, and since 2021 the registry has been continued as Augsburg Myocardial Infarction Registry at the University Hospital Augsburg. The study area comprises approximately 700,000 inhabitants (city of Augsburg, and the counties Augsburg and Aichach-Friedberg). All AMI patients admitted to one out of seven hospitals in the study area were consecutively registered, given that the patient was between 25 and 74 years (since 2009 between 25 and 84 years) of age and had his/her primary residence in the study region at the time of the infarction. Information on case identification, diagnostic classification of events and quality control of the data can be found in more detail in previous publications [15,16].

For this analysis, patients with STEMI who were admitted to the University Hospital Augsburg between May 2009, and July 2013 were included. Blood samples were taken from these patients immediately after hospital admission and were stored at -80 °C until measurement. For 791 consecutive patients, a panel of fatty acids was measured in 2023. Information on fatty acid measurements or important covariables were missing from 74 patients. Ultimately, 717 STEMI cases were available for the present analysis.

The study (original data collection) was approved by the ethics committee of the Bavarian Medical Association (Bayerische Landesärztekammer), approval number 12057. Furthermore, the collection of blood was approved by the ethics committee of the Bavarian Medical Association, approval number 09016. The study and blood collection were performed in accordance with the Declaration of Helsinki. Written informed consent was obtained from all included patients.

2.2. Data collection

All AMI patients were interviewed by trained study nurses using a standardized questionnaire during their hospital stay. In addition, the patients' medical chart was reviewed to collect a variety of data, such as demographic data, data on cardiovascular risk factors, medical history, comorbidities, medication, laboratory parameters, and electrocardiography.

The blood samples for the 791 patients were obtained within the scope of cardiac catheterization, which was mostly performed immediately after hospital admission. EDTA blood samples (arterial blood) were taken right at the beginning of the catheterization. The samples were then immediately processed in a standardized manner in the catheter laboratory (centrifugation, aliquoting, and freezing at -80 °C).

2.3. Clinical chemistry measurement

All blood parameters including serum creatinine were measured in venous blood samples taken at hospital admission or during hospital stay as part of the regular diagnosis and routine treatment. Estimated glomerular filtration rate (eGFR) was calculated based on creatinine levels at admission using the CKD-EPI formula [17].

Plasma fatty acids were measured by a high-throughput nuclear magnetic resonance (NMR) spectroscopy platform (Nightingale Health, Finland) [18,19]. The fatty acid parameters included long-chain omega-3 docosahexaenoic acid (DHA), omega-6 linoleic acid, total omega-3 PUFA, total omega-6 PUFA, total PUFA, total monounsaturated fatty acid (MUFA), total saturated fatty acid (SFA), and total fatty acids. Both the concentration of each fatty acid and the percentage of different types of fatty acid to total fatty acids were calculated.

2.4. Statistical analysis

Categorical variables were given as absolute frequencies with percentages and compared using Chi-square tests. Continuous variables were presented as mean and standard deviation (SD) in case of normal distribution or median and inter-quartile range (IQR) for non-normally distributed variables. For comparison of normally distributed continuous variables Student's t tests and of non-normally distributed variables Mann–Whitney U tests were used.

2.5. Linear regression analysis

The obtained values for each fatty acid parameter as well as the fatty acid ratios were standardized, that means that the variable was centered and normalized so that the transformed variable had an expectancy value of 0 and a statistical variance of 1. Thus, the comparability between different fatty acid values is possible. To examine the association between fatty acids (exposure) and kidney function (continuous eGFR; outcome), linear regression models were calculated. According to literature research, the models were adjusted for sex (male/female), age (in years), diabetes mellitus (yes/no), smoking status current/former/never) and statin therapy prior to AMI. To control for multiple testing, false discovery rate (FDR) adjustment of the obtained *p*-values was conducted. Observations with Cook's distance values > 0.005 were eliminated from the regression models as this indicates excessively influential data points. The effect estimates (β-coefficient and 95 % CI) of the linear models must be interpreted as the expected change in standardized outcome (eGFR) associated with every increase of one standard deviation in the respective exposure variable.

3. Results

Table 1 presents the characteristics for the study sample stratified by eGFR $</\geq 60$ ml/min/1.73 m^2. STEMI patients with an eGFR < 60 ml/min/1.73 m^2 were significantly older, more frequently females, more frequently suffered from diabetes and hypertension, were treated more frequently with statins, received less often a percutaneous coronary intervention (PCI), and more frequently a coronary artery bypass grafting in comparison to patients with an eGFR ≥ 60 ml/min/1.73 m^2. Furthermore, they had significantly lower levels of total fatty acids, saturated fatty acids, PUFAs, Omega-6 fatty acids, linoleic acid, and PUFA/MUFA ratio than their counterparts. On the other hand, there were significantly higher levels of Omega-3 fatty acids, and DHA but lower levels of MUFA/total fatty acids ratio observed in STEMI patients with an eGFR $>=60$ ml/min/1.73 m^2 than in patients with a lower eGFR.

Fig. 1 shows the results of the linear regression analyses. After adjustment for age, sex, history of diabetes, smoking status, and treatment with statins prior to AMI and FDR adjustment of the p-values, the following fatty acid parameters were significantly positive associated with serum eGFR values at admission: PUFAs, Omega-6 fatty acids, linoleic acid, and DHA. Furthermore, the degree of unsaturation and the ratios PUFAs/total fatty acids, linoleic acid/total fatty acids, Omega-6 fatty acid/total fatty acids, and PUFAs/MUFAs were significantly positive related to eGFR. The only significantly inverse association was

Table 1
Baseline characteristics of the STEMI patients stratified by eGFR </≥ 60 ml/min/1.73 m^2.

	Total sample $N = 717$	GFR ≥ 60 $N = 493$	GFR < 60 $N = 224$	*P*-Value
Age (mean,SD)	63.4 (12.6)	59.9 (11.9)	71.1 (10.4)	<0.001
Female	177 (24.7)	105 (21.3)	72 (32.1)	0.002
Died within 28 days	46 (6.4)	17 (3.4)	29 (12.9)	<0.001
Comorbidities				
Smoking				<0.001
never smoker	194 (27.1)	119 (24.1)	75 (33.5)	
ex smoker	174 (24.3)	114 (23.1)	60 (26.8)	
smoker	284 (39.6)	231 (46.9)	53 (23.7)	
smoking status unknown	65 (9.1)	29 (5.9)	36 (16.1)	
Diabetes (yes)	197 (27.5)	121 (24.5)	76 (33.9)	0.012
Hyperlipidemia (yes)	357 (49.9)	252 (51.1)	105 (47.1)	0.359
Hypertension (yes)	515 (71.9)	330 (66.9)	185 (83.0)	<0.001
Body mass index (kg/m^2)	27.0 (24.7-30.1)	26.8 (24.6 - 29.9)	27.4 (24.9 - 30.6)	0.212
eGFR (ml/min/1.73 m^2)	70.5 (55.9 - 85.4)	80.3 (70.0 - 91.6)	47.0 (36.8 - 55.2)	<0.001
Statins before hospital admission				0.013
yes	54 (7.5)	29 (5.9)	25 (11.2)	
no	305 (42.5)	223 (45.2)	82 (36.6)	
no information on statin use	358 (49.9)	241 (48.9)	117 (52.2)	
Acute treatment				
PCI (yes)	652 (91.1)	457 (92.9)	195 (87.1)	0.017
Bypass therapy (yes)	66 (9.2)	38 (7.7)	28 (12.5)	0.056
Fatty acids				
Degree of unsaturation (degree)	1.31 (1.27 - 1.35)	1.31 (1.27 - 1.35)	1.30 (1.27 - 1.33)	0.004
Total fatty acids (mmol/l)	12.06 (10.69 - 13.67)	12.28 (10.86 - 13.91)	11.75 (10.40 - 13.24)	0.002
Saturated fatty acids (mmol/l)	4.35 (3.83 - 4.97)	4.41 (3.90 - 5.05)	4.19 (3.64 - 4.82)	0.002
Monounsaturated fatty acids (mmol/l)	2.79 (2.41 - 3.39)	2.81 (2.42 - 3.40)	2.73 (2.40 - 3.38)	0.305
Polyunsaturated fatty acids (mmol/l)	4.89 (4.34 - 5.42)	4.95 (4.47 - 5.47)	4.66 (4.17 - 5.34)	<0.001
Omega-6 fatty acids (mmol/l)	4.49 (4.04 - 4.96)	4.55 (4.15 - 5.00)	4.31 (3.88 - 4.85)	<0.001
Linoleic acid (mmol/l)	3.22 (2.82 - 3.69)	3.25 (2.91 - 3.73)	3.05 (2.67 - 3.56)	<0.001
Omega-3 fatty acids (mmol/l)	0.38 (0.28 - 0.49)	0.39 (0.29 - 0.50)	0.36 (0.26 - 0.46)	0.025
Docosahexaenoic acid (mmol/l)	0.20 (0.17 - 0.25)	0.21 (0.17 - 0.25)	0.20 (0.17 - 0.24)	0.044
Ratio of saturated fatty acids to total fatty acids (%)	35.98 (35.27 - 36.89)	36.03 (35.31 - 36.96)	35.86 (34.99 - 36.77)	0.064
Ratio of monounsaturated fatty acids to total fatty acids (%)	23.50 (22.03 - 25.27)	23.20 (21.92 - 25.12)	24.10 (22.66 - 25.76)	0.002
Ratio of polyunsaturated fatty acids to total fatty acids (%)	40.54 (38.23 - 42.35)	40.74 (38.32 - 42.46)	40.22 (38.21 - 42.00)	0.094
Ratio of omega-6 fatty acids to total fatty acids (%)	37.26 (35.33 - 39.18)	37.35 (35.33 - 39.25)	37.11 (35.25 - 39.03)	0.307
Ratio of linoleic acid to total fatty acids (%)	26.87 (24.63 - 28.91)	26.95 (24.70 - 29.11)	26.58 (24.49 - 28.64)	0.24
Ratio of omega-3 fatty acids to total fatty acids (%)	3.12 (2.50 - 3.81)	3.14 (2.54 - 3.77)	3.09 (2.35 - 3.84)	0.276
Ratio of docosahexaenoic acid to total fatty acids (%)	1.72 (1.45 - 2.03)	1.71 (1.45 - 2.02)	1.74 (1.44 - 2.03)	0.705
Ratio of polyunsaturated fatty acids to monounsaturated fatty acids (ratio)	1.74 (1.51 - 1.90)	1.77 (1.53 - 1.93)	1.67 (1.48 - 1.86)	0.009
Ratio of omega-6 fatty acids to omega-3 fatty acids (ratio)	11.88 (9.43 - 14.96)	11.85 (9.43 - 14.93)	12.01 (9.45 - 15.13)	0.391

observed between the ratio MUFAs/total fatty acids and eGFR serum levels.

4. Discussion

In the present study analyzing STEMI patients, PUFAs and in particular total omega-6 fatty acids, linoleic acid and the omega-3 fatty acid DHA, were positively associated with admission eGFR values. There were no associations between total omega-3 PUFAs as well as total MUFAs and kidney function. However, an increase of the ratio of monounsaturated fatty acids to total fatty acids was inversely associated with eGFR.

Recent studies on different study populations suggested that PUFAs may have protective effects on kidney function. Higher total dietary intake of PUFAs has been associated with better renal function and lower risk of CKD in participants of the National Health and Nutrition Examination Surveys (NHANES) from 2005 to 2010 [13]. However, while dietary PUFA intake showed promise, genetically determined serum PUFA levels were not significantly associated with renal function measures using a mendelian randomization approach [8]. In the population-based InCHIANTI study, older adults with higher plasma PUFA levels (both n-3 FA and n-6 FA) experienced less decline in creatinine clearance over a period of three years and had a lower risk of developing renal insufficiency [20].

In the present study we observed beneficial effects of higher plasma levels of total omega-6 fatty acids and linoleic acid on kidney function, but previous findings on these associations were conflicting [10]. A cross-sectional study found that higher n-6 PUFA intake was associated with a lower eGFR and an increased risk of CKD [10]. A Mendelian randomization study suggested that higher omega-6 fatty acids and linoleic acid levels may increase eGFR, although the effects were small [21]. In a cross-sectional Korean study, higher n-6 FA intake was positively associated with eGFR levels and potentially beneficial for maintaining kidney function [22]. In contrast, a cohort study of renal transplant recipients found no associations between plasma linoleic acid or arachidonic acid levels and patient or graft survival [23].

We observed no association between the plasma level of total n-3 fatty acids and eGFR but found a significant positive association between DHA and eGFR. However, the ratio of DHA to total fatty acids was also not associated with eGFR. Contrary to the present findings, in prior research omega-3 fatty acids featured protective associations with kidney function. Lauretani et al. found that higher plasma n-3 fatty acids were associated with a less steep decline in creatinine clearance (p-value <0.0001) and a lower risk of renal insufficiency [20]. In another study, the highest quartile of long-chain n-3 PUFA intake was associated with reduced CKD prevalence (OR 0.69, 95 % CI 0.49–0.99) and alpha-linolenic acid was positively associated with CKD (OR 1.18, 95 % CI 1.05–1.32) [11]. Beneficial effects of omega-3 PUFAs have also been shown in a recent randomized clinical trial in IgA nephropathy patients with n-3 PUFA supplementation slowing the rate of renal function loss [24]. In renal insufficiency models, omega-3 PUFAs demonstrated

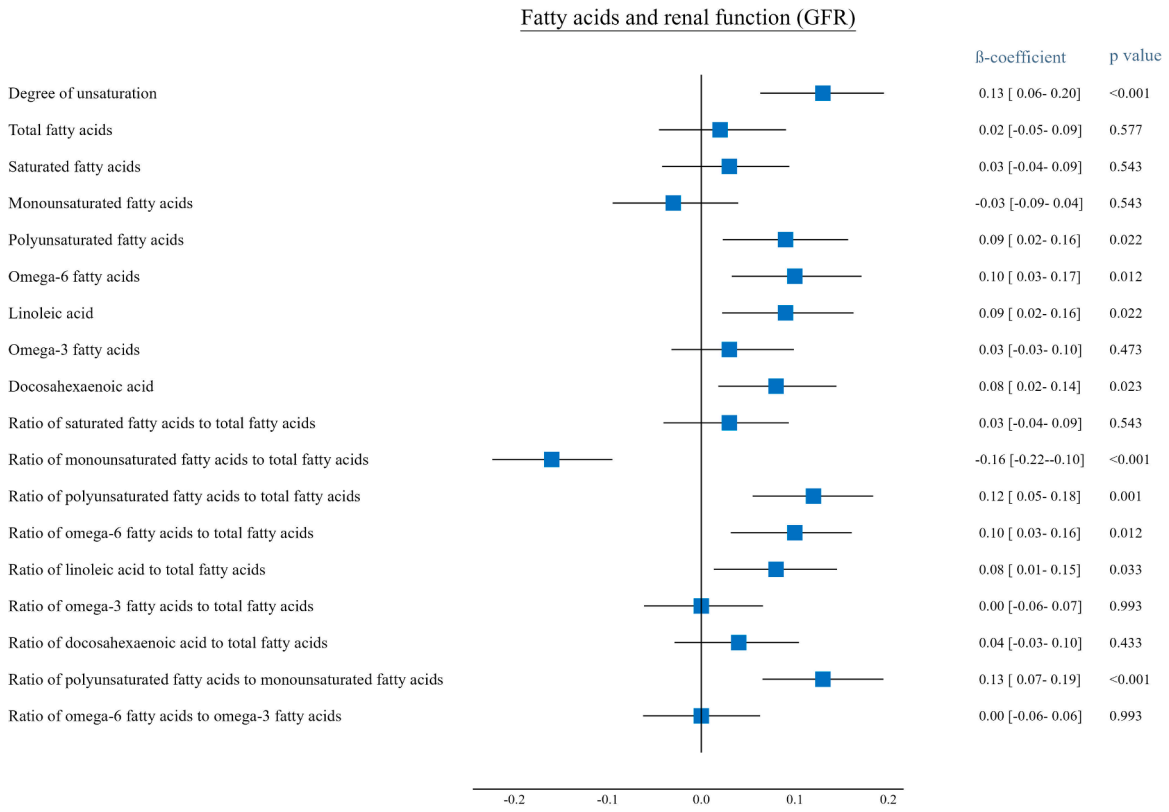

Fig. 1. Results of the associations between the concentration of different fatty acids as well as fatty acid ratios and kidney function (eGFR values). The linear regression models were adjusted for sex (male/female), age (in years), diabetes mellitus (yes/no), and statin therapy prior to AMI. FDR-adjusted p-values are shown.

renoprotective effects, lowering serum cholesterol and urinary eicosanoid excretion, while omega-6 PUFAs increased glomerular pressure and hypertrophy [25]. Acute dietary changes in omega-3 and omega-6 PUFAs significantly impacted survival following ischemic renal injury, with omega-3 diets showing improved outcomes and increased formation of renoprotective DHA-derived protectin D1 [26].

Prior research suggested no clear relationship between MUFAs and kidney function. Dietary MUFA intake showed no significant association with renal function measures in NHANES [8]. Other research observed that elevated serum MUFA levels may contribute to increased cardiovascular risk in CKD patients [27]. However, a long-term dietary intervention study demonstrated that a Mediterranean diet rich in extra-virgin olive oil (high in MUFAs) may preserve kidney function better than a low-fat diet, particularly in coronary heart disease patients with type 2 diabetes and mildly impaired renal function [28].

Impaired renal function is a significant risk factor and adverse prognostic indicator in AMI patients [29]. Worsening renal function occurs in approximately 18.7 % of AMI survivors and is independently associated with higher long-term mortality risk [30]. Van Westing et al. investigated the association of some fatty acids and kidney function in post myocardial infarction patients and found that higher plasma fatty acids, particularly linoleic acid, may potentially delay kidney function decline [14]. This association was stronger in patients with diabetes or chronic kidney disease [14]. Other research suggested that omega-3 fatty acids, particularly eicosapentaenoic acid and DHA, may slow the decline of kidney function in post myocardial infarction patients, with a small beneficial effect observed after long-term supplementation [31]. In the present study on acute STEMI patients, linoleic acid was also significantly associated with an increase of eGFR in patients with STEMI supporting the findings in post myocardial infarction patients. We found no association between plasma levels of MUFAs and eGFR, but the ratio of MUFA to total fatty acids was inversely related to kidney function, a finding, which must be confirmed or refuted in further studies.

These findings and prior literature highlight the complex relationship between fatty acid intake and kidney health, warranting further research to elucidate optimal dietary recommendations for renal function preservation. PUFAs may protect kidney function through various mechanisms [32]. For example, they can mitigate chronic kidney injuries by modulating inflammatory factors, the eicosanoid balance, and cytokine production. Moreover, they also intervene in the processes of kidney fibrosis by regulating the production of pro-inflammatory cytokines, the renin and nitric oxide systems, as well as gene expression [5].

4.1. Strengths and limitations

The study was based on patients from the population-based Augsburg Myocardial Infarction Registry with consecutive enrollment of patients and hence minimizing selection bias. EDTA plasma samples were taken during the PCI intervention and processed in a standardized manner, which guarantees high quality. Furthermore, comprehensive information was available for every AMI case to characterize the study sample and for confounder adjustment in the analyses.

The present study also has some limitations. There was no other cohort from another AMI register available to replicate the present findings. Furthermore, no data on the omega-3 fatty acids eicosapentaenoic acid and alpha-linolenic acid as well as the omega-6 fatty acids gamma-linolenic acid and arachidonic acid was available in the present study. Therefore, no statements can be made about the relationship between these fatty acids and kidney function. As this study is an observational one, no conclusions about causality can be drawn (including the possibility of reverse causality). We also cannot rule out the possibility of residual or unmeasured confounding. Finally, as this study included only STEMI patients between 25 and 84 years, the results may not be generalizable to all age or ethnic groups as well as to non-ST-elevation myocardial infarction events.

5. Conclusions

PUFAs, particularly omega-6 fatty acids, seem to be associated with kidney function in patients with AMI. Given the prognostic importance of renal function in coronary artery diseases and AMI patients, further research on interventions to minimize worsening renal function and protect kidney function in this patient group is warranted [29,30]. Especially, the role of omega-3 fatty acids and PUFAs in this context still needs to be investigated in more detail. As PUFAs are not produced by the body and must be obtained through diet, further studies must identify optimal dietary recommendations for maintaining kidney function in patients with AMI.

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Data availability statement

The datasets generated during and/or analyzed in the current study are not publicly available due to data protection aspects but are available in an anonymized form from the corresponding author on reasonable request. Requests to access these datasets should be directed to Timo Schmitz, timo.schmitz@med.uni-augsburg.de.

CRediT authorship contribution statement

Christa Meisinger: Conceptualization, Investigation, Methodology, Project administration, Writing – original draft. **Timo Schmitz:** Formal analysis, Methodology, Visualization, Writing – review & editing. **Philip Raake:** Investigation, Project administration, Writing – review & editing. **Jakob Linseisen:** Investigation, Project administration, Writing – review & editing.

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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