

Research Article**Serum Potassium Levels and Mortality in Acute Myocardial Infarction**

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Article Info

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Abstract

Background: Serum potassium is central to myocardial electrophysiology, and both hypokalemia and hyperkalemia have been linked to adverse cardiac events. Data on the relationship between serum potassium status and in-hospital mortality in patients with acute myocardial infarction (AMI) in the Indian population remain limited.

Methods: A hospital-based cross-sectional observational study was conducted over 18 months in the Department of General Medicine and Cardiology of a tertiary care teaching hospital. One hundred patients admitted with AMI were enrolled by random sampling and stratified into hypokalemic (n = 9), normokalemic (n = 66), and hyperkalemic (n = 25) groups based on serial serum potassium estimations (flame photometry) performed on Day 0, 1, 3, and 5 of admission. Baseline characteristics, ECG pattern, duration of hospital stay, and in-hospital mortality were compared across groups using one-way ANOVA and the chi-square test, with p < 0.05 considered significant.

Results: Overall in-hospital mortality was 28% (28/100). Mortality was 77.8% in the hypokalemic group and 76.0% in the hyperkalemic group, compared with 3.0% in the normokalemic group ($\chi^2 = 60.04$, p < 0.001). Duration of hospital stay was longest in the normokalemic group (5.65 ± 0.83 days) compared with the hypokalemic (3.56 ± 1.51 days) and hyperkalemic (3.68 ± 1.77 days) groups (p < 0.001). Age, sex, prevalence of diabetes mellitus and hypertension, and ECG pattern did not differ significantly between groups.

Conclusion: In-hospital mortality in AMI followed a U-shaped relationship with serum potassium status, with both hypokalemia and hyperkalemia associated with substantially higher mortality than normokalemia. Serum potassium may serve as a simple, widely available prognostic marker in AMI, warranting closer monitoring and correction of dyskalemia in this population.

Keywords: Acute myocardial infarction; Serum potassium; Hyperkalemia; Hypokalemia; Electrolyte imbalance; In-hospital mortality

Introduction

Acute myocardial infarction (AMI) remains one of the foremost causes of morbidity and mortality worldwide, accounting for a substantial share of cardiovascular disease-related deaths [1]. Current international guidelines for the management of ST-segment elevation myocardial infarction emphasize timely reperfusion and comprehensive risk-factor control to limit myocardial damage and improve survival [2]. Lifestyle-related risk factors such as hypertension, dyslipidemia, obesity, and sedentary behaviour continue to drive the cardiovascular disease burden globally [3], and in India this burden has assumed disproportionate significance, with cardiovascular disease increasingly affecting younger and urban populations and exposing gaps in healthcare access and quality [4].

Serum potassium is an electrolyte central to cardiac electrophysiology. It contributes to the resting membrane potential of cardiac myocytes and governs the repolarization phase of the action potential through potassium-channel-mediated efflux [5]. Potassium channels, including the slow and rapid delayed rectifiers and the inward rectifier current, coordinate the plateau and terminal repolarization phases of the cardiac action potential and are essential to normal sinoatrial and ventricular function [7,8,9]. Because potassium homeostasis is tightly regulated by renal and hormonal mechanisms, even modest perturbations can have disproportionate effects on myocardial excitability and are linked to oxidative-stress pathways implicated in a range of cardiovascular and metabolic disorders [6].

Hyperkalemia is a recognized complication in hospitalized patients and in those receiving renin-angiotensin-aldosterone system inhibitors, and its pathogenesis and stepwise management have been well characterized [10,11]. Contemporary approaches to chronic hyperkalemia in patients with chronic kidney disease highlight the therapeutic tension between controlling potassium and preserving guideline-directed cardioprotective

therapy [12], a tension similarly reflected in major heart-failure practice guidelines that identify hyperkalemia as a barrier to optimal pharmacotherapy [13]. Despite this substantial literature on potassium physiology and hyperkalemia management in general clinical populations, the specific relationship between serum potassium status and in-hospital mortality in patients presenting with AMI remains incompletely defined, particularly in the Indian setting. This study was therefore undertaken to characterize serum potassium status at admission and during the early hospital course in patients with AMI, and to examine its association with in-hospital mortality.

Materials and Methods

Study design, setting, and period

This was a hospital-based prospective observational study conducted over 18 months (July 2021 to December 2022) in the Department of General Medicine and Cardiology of a tertiary care teaching hospital in Kolkata, West Bengal, India.

Study population and sampling

Patients admitted with acute myocardial infarction (ST-segment elevation or non-ST-segment elevation) in the departments of General Medicine and Cardiology were eligible. Participants were selected by simple random sampling using a random number table.

Sample size

The minimum sample size was estimated using the formula $N = Z^2 p(1-p)/L^2$, with $Z = 1.96$ (95% confidence interval), an assumed proportion (p) of 0.6, and a margin of error (L) of 0.1, yielding a minimum requirement of 92 patients. A total of 100 patients were enrolled during the study period.

Inclusion criteria: Patients of any age or sex admitted with ST-segment elevation or non-ST-

segment elevation myocardial infarction who provided informed consent were included.

Exclusion criteria: Patients with pre-existing renal or hepatic disease, those on mechanical ventilation, those receiving medications known to cause hyperkalemia (e.g., angiotensin receptor blockers, digoxin, tacrolimus, cyclosporine), and those not consenting to participate were excluded.

Data collection

Following institutional ethics committee approval and written informed consent, a pretested, predesigned proforma was used to record demographic details, clinical history, and examination findings. Investigations included complete hemogram, liver function test, renal function test with serum electrolytes, 12-lead ECG, and serial serum potassium estimation by the flame photometry technique on the day of admission (Day 0) and on Day 1, Day 3, and Day 5. Serum potassium values >5.0 mmol/L were classified as hyperkalemia, values <3.5 mmol/L as hypokalemia, and intervening values as normokalemia. Patients were considered hypertensive if systolic blood pressure exceeded 140 mmHg, diastolic blood pressure exceeded 90 mmHg, or they were receiving antihypertensive medication, and diabetic if fasting blood glucose exceeded 126 mg/dl, post-prandial blood glucose exceeded 200 mg/dl, or they were receiving anti-diabetic medication. Serial 12-lead ECG monitoring was performed to detect arrhythmias and adverse cardiac events.

Statistical analysis

Continuous variables are expressed as mean $\pm$ standard deviation and were compared across the three potassium groups using one-way ANOVA. Categorical variables are expressed as frequencies

and percentages and were compared using the chi-square test. A p value < 0.05 was considered statistically significant.

Ethical considerations

The study protocol was approved by the Institutional Ethics Committee. Written informed consent, translated into the participant's preferred language (Bengali, Hindi, or English), was obtained from all participants. Confidentiality and voluntary participation were maintained throughout, and any participant found to have morbidity during the study was appropriately treated or referred.

Results

A total of 100 patients admitted with acute myocardial infarction (AMI) were included in the analysis and stratified into three groups based on serum potassium status: Group 1 (hypokalemia, $n = 9$), Group 2 (normokalemia, $n = 66$), and Group 3 (hyperkalemia, $n = 25$).

The mean age of the study population was comparable across groups (Group 1: 61.22 ± 9.09 years; Group 2: 60.83 ± 8.49 years; Group 3: 65.76 ± 10.57 years; $F = 2.71$, $p = 0.072$). Sex distribution did not differ significantly between groups ($\chi^2 = 1.13$, $p = 0.568$), nor did the prevalence of diabetes mellitus ($\chi^2 = 4.36$, $p = 0.113$) or hypertension ($\chi^2 = 0.01$, $p = 0.996$). ECG pattern (STEMI vs non-STEMI) was also similar across groups ($\chi^2 = 2.29$, $p = 0.318$). All patients (100%) tested positive for Troponin T, irrespective of group.

Duration of hospital stay differed significantly between groups ($F = 31.57$, $p < 0.001$): Group 2 had the longest mean stay (5.65 ± 0.83 days) compared with Group 1 (3.56 ± 1.51 days) and Group 3 (3.68 ± 1.77 days).

Table 1. Baseline demographic and clinical characteristics of patients by potassium group

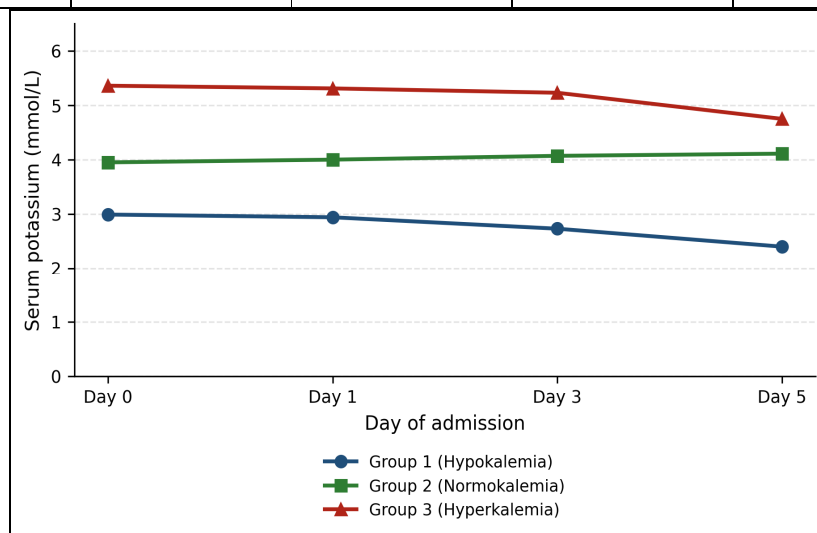
Variable	Group 1 (Hypokalemia) (n = 9)	Group 2 (Normokalemia) (n = 66)	Group 3 (Hyperkalemia) (n = 25)	p value
Age, years (mean $\pm$ SD)	61.22 $\pm$ 9.09	60.83 $\pm$ 8.49	65.76 $\pm$ 10.57	0.072
Sex				0.568
Male, n (%)	4 (44.4)	32 (48.5)	15 (60.0)	
Female, n (%)	5 (55.6)	34 (51.5)	10 (40.0)	
Diabetes mellitus, n (%)	2 (22.2)	39 (59.1)	14 (56.0)	0.113
Hypertension, n (%)	6 (66.7)	45 (68.2)	17 (68.0)	0.996
ECG pattern				0.318
STEMI, n (%)	4 (44.4)	40 (60.6)	18 (72.0)	
Non-STEMI, n (%)	5 (55.6)	26 (39.4)	7 (28.0)	
Duration of hospital stay, days (mean $\pm$ SD)	3.56 $\pm$ 1.51	5.65 $\pm$ 0.83	3.68 $\pm$ 1.77	<0.001

SD, standard deviation; STEMI, ST-elevation myocardial infarction. *p* values by one-way ANOVA (age, duration of stay) or chi-square test (remaining variables).

Serum potassium levels differed significantly between groups at every time point measured (Day 0, 1, 3, and 5), consistent with the group definitions (Table 2, Figure 1). The magnitude of difference between groups was most pronounced at admission (Day 0: $F = 159.25$, $p < 0.001$) and narrowed by Day 5 ($F = 26.79$, $p < 0.001$), with Group 2 and Group 3 levels converging somewhat over the course of admission while Group 1 levels continued to decline.

Table 2. Serial serum potassium levels (mmol/L) across the three groups

Day	Group 1 (mean $\pm$ SD)	Group 2 (mean $\pm$ SD)	Group 3 (mean $\pm$ SD)	p value (One-way ANOVA)
Day 0	2.99 $\pm$ 0.27	3.95 $\pm$ 0.35	5.36 $\pm$ 0.53	<0.001
Day 1	2.94 $\pm$ 0.34	4.00 $\pm$ 0.32	5.31 $\pm$ 0.56	<0.001
Day 3	2.73 $\pm$ 0.15	4.07 $\pm$ 0.33	5.23 $\pm$ 0.62	<0.001
Day 5	2.40 $\pm$ 0.37*	4.11 $\pm$ 0.37	4.75 $\pm$ 0.63	<0.001

**Figure 1.** Trend of mean serum potassium levels (mmol/L) by group across Days 0, 1, 3, and 5 of admission.

Overall in-hospital mortality was 28% (28/100). Mortality differed markedly by potassium group ($\chi^2 = 60.04$, $p < 0.001$): Group 1 (hypokalemia) had a mortality rate of 77.8% (7/9) and Group 3

(hyperkalemia) had a mortality rate of 76.0% (19/25), both substantially higher than Group 2 (normokalemia), which had a mortality rate of 3.0% (2/66) and a discharge rate of 97.0% (64/66).

Table 3. Clinical outcome (death vs. discharge) by potassium group

Outcome	Group 1 (n = 9)	Group 2 (n = 66)	Group 3 (n = 25)	Total (n = 100)	p value (Chi-square test)
Death, n (%)	7 (77.8)	2 (3.0)	19 (76.0)	28	<0.001
Discharge, n (%)	2 (22.2)	64 (97.0)	6 (24.0)	72	

Discussion

In the present cohort of 100 patients admitted with acute myocardial infarction, in-hospital mortality was strongly associated with serum potassium status, following a distinct U-shaped pattern: both hypokalemia (77.8%) and hyperkalemia (76.0%) were associated with markedly higher mortality than normokalemia (3.0%). This pattern is broadly consistent with earlier work examining potassium and outcomes specifically in AMI. Grodzinsky et al (2016) conducted a study to characterize hyperkalemia in 38,689 patients hospitalized with AMI and reported that mortality rose sharply once potassium exceeded 5.5 mEq/L, with a dose-response relationship between the frequency of hyperkalemic episodes and mortality risk [14]. Colombo et al (2017) conducted a study to evaluate admission serum potassium and long-term mortality in the MONICA/KORA myocardial infarction registry and found that concentrations at or above 5.0 mEq/l carried the highest mortality, while concentrations between 4.5 and 5.0 mEq/l were associated with an intermediate risk [15]. Ma et al conducted a study of 6613 patients with ST-elevation myocardial infarction and identified a potassium range of 4.0 to 4.5 mEq/L as the nadir of risk [16]. Because our cohort predominantly comprised patients at the extremes of the potassium range rather than at intermediate concentrations, our findings extend this literature by demonstrating a comparably steep mortality gradient at the lower end of the range as well [17].

The mechanistic basis for this bidirectional risk is well described. Sica et al proposed that serum

potassium concentrations of 4.0–5.0 mmol/L are optimal for cardiovascular stability, and that both diuretic-induced hypokalemia and untreated hyperkalemia can independently predispose to adverse cardiac events [18]. A recent meta-analysis by Fan et al, pooling 31 cohort studies and over 227,000 participants, confirmed a U-shaped association between potassium and mortality in cardiovascular disease, with the lowest risk observed at approximately 4.2 mmol/L, closely matching the normokalemic range in our own cohort [19]. Toto similarly emphasized that both hypokalemia and hyperkalemia carry independent mortality risk in populations with cardiovascular and renal disease, cautioning that a single baseline measurement may not fully capture dynamic in-hospital potassium fluctuations [20]. Ke et al (2023) conducted a study to examine serum potassium and adverse cardiovascular events in 8263 patients undergoing percutaneous coronary intervention and likewise reported a U-shaped relationship, with the lowest risk between 3.8 and 4.0 mmol/L [21]. Shlomei et al conducted a study restricted to patients with normal-range admission potassium and found that even values at the upper end of the normal range (4.45–5.2 mEq/L) were associated with increased mortality, suggesting that elevated potassium, even without frank hyperkalemia, may indicate greater underlying illness severity [22]. Collins et al, analyzing over 900,000 individuals, confirmed the U-shaped mortality curve across populations with and without heart failure, chronic kidney disease, and diabetes, with the nadir again falling between 4.0 and 5.0 mEq/L [23].

Beyond prognostic associations, the interplay between potassium management and disease-modifying therapy has direct clinical relevance. Pitt et al, in the landmark RALES trial, conducted a study demonstrating that spironolactone reduced all-cause mortality in severe heart failure despite a modest attendant risk of hyperkalemia, underscoring that potassium-related concerns should not by themselves preclude the use of mortality-reducing, renin-angiotensin-aldosterone system-modulating therapy [24]. More recently, Butler et al, in the DIAMOND trial, conducted a study showing that the potassium binder patiromer allowed more consistent use of target-dose renin-angiotensin-aldosterone system inhibitor therapy in patients with heart failure and reduced ejection fraction who had hyperkalemia related to this therapy [25], pointing toward a strategy that could, in principle, be extended to the post-AMI population in whom such inhibition is similarly indicated but potassium derangement may limit its use.

Overall, our findings of markedly higher mortality among patients with either hypokalemia or hyperkalemia compared with normokalemia corroborate the U-shaped potassium–mortality relationship consistently reported in the AMI and broader cardiovascular literature, while suggesting that in our cohort the magnitude of mortality observed in our cohort was high compared with that reported in some larger cohorts, which may reflect differences in case mix, treatment practices, or the small sample size and single-centre design.

Limitations

This study has several limitations. The sample size was relatively small and the study was conducted at a single tertiary-care centre, which may limit generalizability. The observational design precludes conclusions regarding causality. Potential confounding factors, including infarct severity, left ventricular function, renal function within the inclusion criteria, treatment received,

and reperfusion status, were not assessed in a multivariable model. In addition, the small number of patients in the hypokalemic group may have resulted in imprecise estimates of mortality. Finally, serial potassium measurements may have been influenced by treatment received during hospitalization.

Conclusion

Serum potassium status at admission and during the early hospital course was strongly associated with in-hospital mortality in patients with acute myocardial infarction, with both hypokalemia and hyperkalemia carrying substantially higher mortality risk than normokalemia. These findings support the incorporation of serial serum potassium monitoring into the routine assessment of AMI patients, with a low threshold for correction of dyskalemia, and highlight serum potassium as a simple, inexpensive, and widely available prognostic marker in this setting.

Declarations

Ethics approval and consent to participate:

The study protocol was approved by the Institutional Ethics Committee. Written informed consent was obtained from all participants in their preferred language (Bengali, Hindi, or English). Confidentiality and voluntary participation were maintained throughout the study.

Funding: The authors received no specific funding for this study.

Competing interests: The authors declare that they have no competing interests.

Data availability: The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to institutional and ethical restrictions.

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Use of artificial intelligence: The authors declare that no generative artificial intelligence tools were used to generate the scientific content, data, analysis, or conclusions of this manuscript.

References

1. Salari N, Morddarvanjoghi F, Abdolmaleki A, et al. The global prevalence of myocardial infarction: a systematic review and meta-analysis. *BMC Cardiovasc Disord*. 2023;23:206. doi:10.1186/s12872-023-03231-w
2. Van de Werf F, et al. Management of acute myocardial infarction in patients presenting with persistent ST-segment elevation: the Task Force on the Management of ST-Segment Elevation Acute Myocardial Infarction of the European Society of Cardiology. *Eur Heart J*. 2008;29(23):2909-45. doi:10.1093/eurheartj/ehn416
3. Ghodeshwar GK, Dube A, Khobragade D. Impact of Lifestyle Modifications on Cardiovascular Health: A Narrative Review. *Cureus*. 2023;15(7):e42616. doi:10.7759/cureus.42616
4. Prabhakaran D, Jeemon P, Roy A. Cardiovascular Diseases in India: Current Epidemiology and Future Directions. *Circulation*. 2016;133(16):1605-20. doi:10.1161/CIRCULATIONAHA.114.008729
5. Weiss JN, Qu Z, Shivkumar K. Electrophysiology of Hypokalemia and Hyperkalemia. *Circ Arrhythm Electrophysiol*. 2017;10(3):e004667. doi:10.1161/CIRCEP.116.004667
6. Udensi UK, Tchounwou PB. Potassium Homeostasis, Oxidative Stress, and Human Disease. *Int J Clin Exp Physiol*. 2017;4(3):111-122. doi:10.4103/ijcep.ijcep_43_17
7. Sidorov VY, Woods MC, Wikswo JP. Effects of elevated extracellular potassium on the stimulation mechanism of diastolic cardiac tissue. *Biophys J*. 2003;84(5):3470-9. doi:10.1016/S0006-3495(03)70067-8
8. Aziz Q, Li Y, Tinker A. Potassium channels in the sinoatrial node and their role in heart rate control. *Channels (Austin)*. 2018;12(1):356-366. doi:10.1080/19336950.2018.1532255
9. Schmitt N, Grunnet M, Olesen SP. Cardiac potassium channel subtypes: new roles in repolarization and arrhythmia. *Physiol Rev*. 2014;94(2):609-53. doi:10.1152/physrev.00022.2013
10. Lehnhardt A, Kemper MJ. Pathogenesis, diagnosis and management of hyperkalemia. *Pediatr Nephrol*. 2011;26(3):377-84. doi:10.1007/s00467-010-1699-3
11. Weinstein J, Girard LP, Lepage S, McKelvie RS, Tennankore K. Prevention and management of hyperkalemia in patients treated with renin-angiotensin-aldosterone system inhibitors. *CMAJ*. 2021;193(48):E1836-E1841. doi:10.1503/cmaj.210831
12. Morales E, Cravedi P, Manrique J. Management of Chronic Hyperkalemia in Patients With Chronic Kidney Disease: An Old Problem With New Options. *Front Med (Lausanne)*. 2021;8:653634. doi:10.3389/fmed.2021.653634
13. Heidenreich PA, Bozkurt B, Aguilar D, et al. 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure. *Circulation*. 2022;145(18):e895-e1032. doi:10.1161/CIR.0000000000001063
14. Grodzinsky A, Goyal A, Gosch K, et al. Prevalence and Prognosis of Hyperkalemia in Patients with Acute Myocardial Infarction. *Am J Med*. 2016;129(8):858-65. doi:10.1016/j.amjmed.2016.03.008
15. Colombo MG, Kirchberger I, Amann U, et al. Admission serum potassium concentration

- and long-term mortality in patients with acute myocardial infarction: results from the MONICA/KORA myocardial infarction registry. *BMC Cardiovasc Disord.* 2017;17(1):198. doi:10.1186/s12872-017-0635-x
16. Ma W, Liang Y, Zhu J, et al. Serum Potassium Levels and Short-Term Outcomes in Patients With ST-Segment Elevation Myocardial Infarction. *Angiology.* 2016;67(8):729-36. doi:10.1177/0003319715617074
 17. Goyal A, Spertus JA, Gosch K, Venkitachalam L, Jones PG, Van den Berghe G, Kosiborod M. Serum potassium levels and mortality in acute myocardial infarction. *JAMA.* 2012;307(2):157-164. doi:10.1001/jama.2011.1967.
 18. Sica DA, Struthers AD, Cushman WC, Wood M, Banas JS Jr, Epstein M. Importance of potassium in cardiovascular disease. *J Clin Hypertens (Greenwich).* 2002;4(3):198-206. doi:10.1111/j.1524-6175.2002.01728.x
 19. Fan Y, Wu M, Li X, et al. Potassium levels and the risk of all-cause and cardiovascular mortality among patients with cardiovascular diseases: a meta-analysis of cohort studies. *Nutr J.* 2024;23:8. doi:10.1186/s12937-023-00888-z
 20. Toto RD. Serum Potassium and Cardiovascular Outcomes: The Highs and the Lows. *Clin J Am Soc Nephrol.* 2017;12(2):220-221. doi:10.2215/CJN.00030117
 21. Ke B, et al. Clinical outcomes of serum potassium in patients with percutaneous coronary intervention: insights from a large single-center registry. *Front Cardiovasc Med.* 2023;10:1216422. doi:10.3389/fcvm.2023.1216422
 22. Shlomai G, Berkovitch A, Pinchevski-Kadir S, et al. The association between normal-range admission potassium levels in Israeli patients with acute coronary syndrome and early and late outcomes. *Medicine (Baltimore).* 2016;95(23):e3778. doi:10.1097/MD.0000000000003778
 23. Collins AJ, Pitt B, Reaven N, et al. Association of Serum Potassium with All-Cause Mortality in Patients with and without Heart Failure, Chronic Kidney Disease, and/or Diabetes. *Am J Nephrol.* 2017;46(3):213-221. doi:10.1159/000479802
 24. Pitt B, Zannad F, Remme WJ, et al. The effect of spironolactone on morbidity and mortality in patients with severe heart failure. *N Engl J Med.* 1999;341(10):709-17. doi:10.1056/NEJM199909023411001
 25. Butler J, Anker SD, Lund LH, et al. Patiromer for the management of hyperkalemia in heart failure with reduced ejection fraction: the DIAMOND trial. *Eur Heart J.* 2022;43(41):4362-4373. doi:10.1093/eurheartj/ehac401