Original research

Association of self-reported and accelerometer-based walking pace with incident cardiac arrhythmias: a prospective cohort study using UK Biobank

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ABSTRACT

Objectives Dedicated studies aimed at investigating the relationship between walking pace and arrhythmia are limited. This study assessed associations between self-reported and accelerometer measured walking pace and incident cardiac arrhythmias, overall and by subtype, and explored metabolic and inflammatory markers as possible mediators.

Methods Self-reported average walking pace was available for 420 925 UK Biobank participants, and accelerometer measured time spent walking at different paces was available for 81 956 participants. Outcomes were incident cardiac arrhythmias: all, atrial fibrillation (AF), other (including bradyarrhythmias and ventricular arrhythmias), bradyarrhythmias and ventricular arrhythmias. Cox proportional regression models were used to investigate the associations.

Results Compared with slow walking pace, average and brisk walking pace were associated with significantly lower risks of all cardiac arrhythmias (hazard ratio (HR) 0.65, 95% confidence interval (CI) 0.62 to 0.68; HR 0.57, 95% CI 0.54 to 0.60), AF (HR 0.62, 95% CI 0.58 to 0.65; HR 0.54, 95% CI 0.50 to 0.57) and other arrhythmias (HR 0.69, 95% CI 0.64 to 0.73; HR 0.61, 95% CI 0.57 to 0.65). Overall, 36.0% of the association between walking pace and all arrhythmias was mediated via metabolic and inflammatory markers. The associations were stronger in women, in those aged <60 years, in those with a body mass index <30, in those who had hypertension and in those with ≥2 long term conditions.

Conclusions Average and brisk self-reported walking pace and time spent walking at moderate and brisk pace were associated with a decreased risk of cardiac arrhythmias, in part mediated via metabolic and inflammatory pathways. Our findings suggest brisk walking may be a safe and effective exercise to reduce arrhythmias, especially for higher risk groups.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ There is evidence of a beneficial effect of physical activity (PA) on incident arrhythmias, but dedicated studies aimed at investigating the relationship between walking pace and arrhythmia are limited.
⇒ No study has explored accelerometer measured walking pace and arrhythmia.

WHAT THIS STUDY ADDS

⇒ Faster self-reported walking pace and accelerometer measured amount of time spent walking at a moderate or brisk pace were associated with a lower risk of developing cardiac arrhythmias, including atrial fibrillation and other arrhythmias.
⇒ The associations were independent of other established cardiovascular risk factors and were observed in all population subgroups investigated, but were of larger magnitude in women, non-obese individuals, those aged <60 years and those with established diseases.
⇒ Over a third of the association between walking pace and arrhythmias appeared to be mediated by metabolic factors and inflammation, with central and general obesity making the largest contributions.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ These new findings reinforce the promotion of faster walking pace in PA recommendations that walking at a brisk pace may have a role in secondary, as well as primary, prevention of cardiac arrhythmias, and provide evidence of higher risk groups to target.
⇒ The ability to investigate the possible mediation roles of metabolic risk factors and inflammation provided insights into the aetiology and pathophysiology of cardiac arrhythmias.

INTRODUCTION

Arrhythmias are a group of common cardiac disorders and of growing public concern due largely to a doubling in the prevalence of atrial fibrillation (AF), the most common type of arrhythmia, over the past three decades. AF cases reached almost 60 million globally in 2019.[1] Previous studies have documented arrhythmias as an important contributor to cardiovascular diseases (CVD), sudden cardiac death, disability and mortality.[2] Given that early detection can reduce the morbidity and mortality associated with arrhythmias, there is an urgent need to identify and address modifiable risk factors.

Walking is a simple and largely accessible form of physical activity (PA) for all ages and therefore holds potential as a pragmatic target for intervention. Walking pace, a simple measure of physical fitness and function, has been shown to be related

to CVD morbidity and mortality.[3] There is evidence of a beneficial effect of PA on incident arrhythmias,[4] but dedicated studies aimed at investigating the relationship between walking pace and arrhythmia are limited. Only one cohort study has reported an inverse dose relationship between self-reported walking distance/pace and incident AF,[5] but the study was limited by small sample size and only focused on older adults and one arrhythmia subtype. There remains a large knowledge gap in relation to other types of arrhythmia and underpinning mechanisms. Moreover, measurement of walking pace by questionnaire is limited by poor reliability and validity. Objective measures, such as accelerometers, have the advantages of counteracting recall bias, eliminating subjective interpretation and providing precise quantification. Nevertheless, no study has explored accelerometer-measured walking pace and arrhythmia.

Accumulated evidence has shown that metabolic disorders and inflammation have a critical role in the pathogenesis of arrhythmias.[6] The large population cohort studies showed that metabolic factors and systemic inflammation were associated with an increased risk of arrhythmias.[7 8] Compared with slow walking pace, brisk walking pace was associated with better cardiometabolic health markers and lipid profile, and lower inflammation levels.[9] Therefore, we speculated that metabolic factors and inflammation may partially mediate the association between walking pace and arrhythmias.

To address this evidence gap, we investigated the associations between self-reported walking pace and incident cardiac arrhythmias and arrhythmia subtypes, including atrial fibrillation, other cardiac arrhythmias (non-AF arrhythmias), bradyarrhythmias and ventricular arrhythmias. The associations between accelerometer measured time spent walking at different paces and incident cardiac arrhythmias and arrhythmia subtypes were further explored. Additionally, we investigated whether metabolic factors and inflammation mediated the associations between self-reported walking pace and incident cardiac arrhythmias. The effect modification by age, sex, obesity, smoking status, alcohol drinking and the number of long term conditions (LTCs) on the association between self-reported walking pace and incident cardiac arrhythmias were also explored.

METHODS

Study population

UK Biobank recruited adults from the general population aged 40–69 years between 2006 and 2010, who completed touchscreen questionnaires, had physical measurements taken and provided biological samples in 22 assessment centres across England, Wales and Scotland.[10] More information about the UK Biobank protocol is available online (http://www.ukbiobank.ac.uk).

Exposures

Self-reported walking pace

At baseline, walking pace was measured using a self-completed touchscreen questionnaire with the question: "How would you describe your usual walking pace: slow pace (<3 miles per hour); steady/average pace (3–4 miles per hour); brisk pace (>4 miles per hour); none of the above; prefer not to answer". Thus self-reported walking pace was categorised as slow, average or brisk.

Accelerometer measured walking pace

Between February 2013 and December 2015, a subset (n=103 695) of all the UK Biobank participants recorded their PA data using a wrist worn Axivity AX3 accelerometer on their dominant wrist for 7 days.[11] A total of 96 459 raw accelerometer datasets were received for data analysis. Walking pace was classified based on the Euclidean Norm Minus One (ENMO) acceleration over three axes as slow, moderate and brisk using previously validated thresholds of energy expenditure.[12]

Ascertainment of outcomes

The outcome of interest in this study was all cardiac arrhythmias and subtypes of cardiac arrhythmias, including AF and other (non-AF) arrhythmias. Bradyarrhythmias and ventricular arrhythmias were the two main subtypes of other arrhythmias that we studied. The outcomes were ascertained from hospital admission and day case records using the International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10) codes (online supplemental table 2).

Potential mediators and covariates

Mediators included body mass index (BMI), systolic blood pressure (SBP), serum total cholesterol (TC), haemoglobin A1C (HbA1c) and C reactive protein (CRP). We selected the mediators based on knowledge of potential causal pathways of arrhythmias.[13] For inflammation markers, CRP was selected because it is a commonly used marker of systemic inflammation and has been shown to be significantly and non-linearly associated with arrhythmias.[8] Covariates are provided in the supplemental methods. Details of these measurements can be found in the UK Biobank online protocol (https://www.ukbiobank.ac.uk/).

Equity, diversity and inclusion statement

The author team is gender balanced and comprises junior and senior researchers, and all are in one discipline (public health). Our study population had male and female participants from diverse socioeconomic and racial backgrounds. The discussion considered the limited generalisability.

Statistical analyses

Baseline characteristics of the study population are reported as mean (SD) or median (IQR) for continuous variables, and frequency (%) for categorical variables. Cox proportional hazard regression models were used to estimate hazard ratios (HRs) and their 95% confidence intervals (CIs) for incident arrhythmias associated with self-reported walking pace (low, average and brisk) and quartiles of time spent on different types of walking pace. Per 1−SD increase in time spent at each walking pace was also evaluated because it has more practical interpretations that may be converted to quantitative public health recommendations for time spent on walking pace.

The proportional hazard assumption was evaluated and confounders were selected based on established a priori knowledge[14] and a directed acyclic graph (online supplemental methods). Two models were finally used: model 1, adjusted for age, sex, Townsend deprivation index and ethnicity; model 2 additionally adjusted for smoking status, weekly units of alcohol consumed, sleep duration, fruit and vegetable intake, processed meat intake, red meat intake, PA, total sedentary time, grip strength and number of LTCs. We tested interaction terms between self-reported walking pace and the other covariates, and undertook subgroup analyses where these were statistically significant. Subgroup analyses were conducted by age (<60 and ≥60 years), sex (men and women), general obesity (BMI <30 and ≥30), smoking status (never and ever/current smoker), alcohol drinking (yes and no) and number of LTCs (<2 and ≥2).

Table 1 Association of self-reported walking pace and incident all cardiac arrhythmias

		Model 1 (n=4 09 619)	Model 2 (n=3 00 427)
Self-reported walking pace	No of cases/ population	Hazard ratio (95% CI)	Hazard ratio (95% CI)
Slow	4348/27877	Reference	Reference
Average	20518/221664	0.58 (0.56 to 0.60)	0.65 (0.62 to 0.68)
Brisk	11708/171384	0.48 (0.46 to 0.50)	0.57 (0.54 to 0.60)

Model 1: adjusted for age, sex, Townsend deprivation index and ethnicity.
Model 2: additionally adjusted for smoking status, weekly units of alcohol use, sleep duration, fruit and vegetable intake, processed meat intake, red meat intake, physical activity, total sedentary time, grip strength and number of long term conditions.
CI, confidence interval; Ref, reference group.

Table 2 Association of accelerometer measured time spent on different types of walking pace and incident all cardiac arrhythmias

		Model 1 (n=78 184)	Model 2 (n=61 268)
Exposure (min/day)	No of cases	Hazard ratio (95% CI)	Hazard ratio (95% CI)
Slow			
Q1 (<89)	1078	Reference	Reference
Q2 (89-125)	968	0.92 (0.85 to 1.01)	0.97 (0.88 to 1.07)
Q3 (125-186)	1037	0.94 (0.87 to 1.03)	0.95 (0.86 to 1.05)
Q4 (≥186)	1034	0.93 (0.85 to 1.01)	0.95 (0.86 to 1.05)
Per 1–SD (73 min/ day) increase	4117	0.98 (0.95 to 1.01)	1.00 (0.96 to 1.04)
Average			
Q1 (<5)	1430	Reference	Reference
Q2 (5-9)	1042	0.82 (0.76 to 0.89)	0.83 (0.76 to 0.91)
Q3 (9-16)	885	0.75 (0.69 to 0.81)	0.73 (0.66 to 0.81)
Q4 (≥16)	760	0.73 (0.66 to 0.8)	0.73 (0.66 to 0.81)
Per 1–SD (13 min/ day) increase	4117	0.94 (0.90 to 0.97)	0.95 (0.91 to 0.99)
Brisk			
Q1 (<0.4)	1332	Reference	Reference
Q2 (0.36–0.9)	1080	0.91 (0.84 to 0.99)	0.88 (0.8 to 0.96)
Q3 (0.9–1.8)	907	0.83 (0.76 to 0.91)	0.81 (0.74 to 0.90)
Q4 (≥1.8)	798	0.82 (0.75 to 0.90)	0.82 (0.74 to 0.91)
Per 1–SD (3 min/ day) increase	4117	0.92 (0.88 to 0.96)	0.93 (0.88 to 0.97)

Low, average and brisk walking were defined as <3 miles per hour (mph), 3–4 mph and >4 mph, respectively.
Model 1: adjusted for age, sex, Townsend deprivation index and ethnicity.
Model 2: additionally adjusted for smoking status, weekly units of alcohol use, sleep duration, fruit and vegetable intake, processed meat intake, red meat intake, physical activity, total sedentary time, grip strength and number of long term conditions.

Online supplemental figure 1 shows the underlying mediation model. We assessed the mediating role of metabolic status (BMI, SBP, TC and HbA1c) and inflammatory factors (CRP) on the association between self-reported walking pace and incident arrhythmias, in the presence of the mediator outcome confounding variables. Details of the mediation analysis are shown in the supplemental methods. Mediation analyses were conducted using the CMAverse R studio package.

Analysis was performed by complete case analysis. Sensitivity analyses were also performed to study the association between self-reported walking pace and arrhythmias by limiting participants with accelerometer data. All analyses were conducted using R, V.4.3.2 (R Foundation for Statistical Computing) statistical packages. Two tailed p values <0.05 were considered to indicate statistical significance.

RESULTS

Self-reported walking pace and arrhythmias

A total of 420 925 participants were included (online supplemental results and figure 2). Mean age was 55.8 (SD 9.30) years, more than half of participants (55.3%) were women and most participants (96.6%) were white (table 1). Overall, 27 877 (6.6%) reported a slow walking pace, 221 664 (52.7%) an average walking pace and 171 384 (40.7%) a brisk walking pace. Over a median follow-up period of 13.7 (IQR 12.8–14.4) years, 36 574 (8.7%) participants developed cardiac arrhythmias (incidence rate 65.9 per 10 000 person years): 23 526 AF (incidence rate 41.9 per 10 000 person years), 19 093 other cardiac arrhythmias (incidence rate 33.9 per 10 000 person-years), 5678 bradyarrhythmias (incidence rate 10.0 per 10 000 person years) and 2168 ventricular arrhythmias (incidence rate 3.7 per 10 000 person years). Participants with a faster walking pace were more likely to be men, live in less deprived areas, have healthier lifestyles, lower waist circumference and BMI, higher grip strength, lower concentrations of total and LDL cholesterol, triglycerides and HbA1c, higher HDL cholesterol, lower levels of inflammatory markers and fewer LTCs (online supplemental table 4).

As shown in table 1 and online supplemental table 5, after adjustment for demographic variables and lifestyle factors, compared with those with low self-reported walking pace, participants with average and brisk walking pace had a significantly lower risk of incident cardiac arrhythmias (HR 0.65, 95% CI 0.62 to 0.68 for average; 0.57, 0.54 to 0.60 for brisk walking pace), AF (0.62, 0.58 to 0.65 for average; 0.54, 0.50 to 0.57 for brisk walking pace), and other cardiac arrhythmias (0.69, 0.64 to 0.73 for average; 0.61, 0.57 to 0.65 for brisk walking pace) (model 2). Following the inclusion of potential mediators in model 3, the inverse association was attenuated but remained significant for all cardiac arrhythmias. Analysing the association in the subgroup of accelerometer data showed that the associations remained significant for all arrhythmia outcomes (online supplemental table 6).

Accelerometer measured walking pace and arrhythmias

A total of 80 773 participants were finally included (online supplemental results and figure 3). Over a median follow-up period of 7.9 (IQR 7.4–8.5) years, 4117 of the participants with accelerometry data developed arrhythmias. Participants who spent more time walking at a brisk pace were generally younger, more likely to be white and male, and were less deprived (online supplemental table 7). They had generally healthier lifestyles; they were less likely to smoke, more likely to have the recommended sleep duration, consumed more fruit and vegetables and less red and processed meat, and they had higher overall levels of PA and less sedentary behaviour. They had lower BMI and higher grip strength, had healthier concentrations of metabolic and inflammatory markers, and were less likely to have hypertension, diabetes and hyperlipidaemia.

After adjustment for potential confounders, the amount of time spent walking at a slow pace was not associated with the risk of any incident arrhythmias (table 2 and online supplemental table 8). In contrast, spending more time walking at an average and brisk pace was associated with a lower risk of cardiac

Table 3 Association of self-reported slow walking pace and incident all cardiac arrhythmias mediated by metabolic profiles and inflammation

Mediators	Total effect	Natural direct effect	Natural indirect effect	Proportion mediated (%) (95% CI)
	Hazard ratio (95% CI)	Hazard ratio (95% CI)	Hazard ratio (95% CI)	
BMI	1.60 (1.53 to 1.68)	1.41 (1.34 to 1.47)	1.14 (1.13 to 1.16)	32.8 (30.1 to 35.9)
TC	1.41 (1.35 to 1.49)	1.40 (1.34 to 1.47)	1.009 (1.007 to 1.011)	2.9 (2.3 to 3.7)
SBP	1.41 (1.35 to 1.47)	1.41 (1.34 to 1.66)	0.997 (0.996 to 0.998)	–1.0 (–0.6 to 1.4)
HbA1c	1.41 (1.34 to 1.48)	1.40 (1.34 to 1.48)	1.003 (1.001 to 1.004)	0.9 (0.4 to 1.4)
CRP (mg/L)	1.41 (1.35 to 1.48)	1.40 (1.33 to 1.47)	1.008 (1.005 to 1.011)	2.8 (1.9 to 3.9)
Total mediation	1.60 (1.54 to 1.68)	1.39 (1.33 to 1.45)	1.157 (1.145 to 1.170)	36.0 (32.7 to 40.1)

Model was adjusted for age, sex, Townsend deprivation index, ethnicity, smoking status, weekly units of alcohol use (continuous), sleep duration, fruit and vegetable intake, processed meat intake, red meat intake, PA, total sedentary time, grip strength and number of LTCs.
BMI was additionally adjusted when performing the single mediation analysis of the other four mediators. Firstly, all mediators (BMI, TC, SBP, HbA1c and CRP) were separately explored in the mediation analysis. Secondly, mediators that were significant were together entered into the total mediation model.
BMI, body mass index; CRP, C reactive protein; HbA1c, haemoglobin A1c; LTCs, long term conditions; PA, physical activity; SBP, systolic blood pressure; TC, total cholesterol.

arrhythmias (HR 0.73, 95% CI 0.66 to 0.81; 0.82, 0.74 to 0.91 for the highest quintiles of average and brisk pace, respectively). For the subtypes of cardiac arrhythmias, participants who spent more time walking at an average and brisk pace had a lower risk of AF and other arrhythmias (online supplemental table 8).

Mediation analysis of metabolic factors and inflammation

The association between self-reported slow walking pace and risk of all cardiac arrhythmias was significantly mediated by BMI, TC, SBP, HbA1c and CRP, each mediating between 0.9% and 32.8% of the association. All of these mediators together explained 36.0% (32.7, 40.1) of the association between slow walking pace and cardiac arrhythmias (table 3). The findings were similar across the other arrhythmia outcomes (online supplemental table 9).

Effect modifiers

We found evidence of statistically significant interactions between self-reported walking pace and sex, age, BMI and number of LTCs, whereby some or all of the associations between walking pace and all arrhythmias, AF or other arrhythmias were stronger among people who were female, aged <60 years, had a BMI <30 and had more than two LTCs (online supplemental tables 10 and 11). There were no significant interactions with smoking status or alcohol consumption (data not shown).

DISCUSSION

In this large scale population cohort of 422 654 adults, we found that faster walking pace and amount of time spent walking at a moderate or brisk pace were associated with a lower risk of developing cardiac arrhythmias, including AF and other arrhythmias. The associations were independent of other established cardiovascular risk factors and were observed in all population subgroups investigated, but were of larger magnitude in women, non-obese individuals, those aged <60 years and those with established diseases. Over a third of the association between walking pace and arrhythmias appeared to be mediated by metabolic factors and inflammation, with central and general obesity making the largest contributions.

Comparison with other studies

Previous studies have observed associations between self-reported walking pace and cardiovascular risk factors,[15] CVD[16] and stroke.[17] Protective effects of PA and walking on incident arrhythmias have also been suggested by some individual studies,[4] but a meta-analysis of 23 cohort studies involving about 2 million participants reported the absence of associations between regular PA and AF risk. Our findings have added to the evidence base by demonstrating that any protective effect of walking on arrhythmias, assuming causality, is specific to walking above a slow pace, whether measured in terms of average pace or amount of time spent walking at a moderate or brisk pace. Our finding of an association between walking pace and AF specifically is consistent with the findings of the Cardiovascular Health Study, a smaller study (n=5446) of older adults (≥65 years).[5]

This study is the first to explore the pathways underpinning the association between walking pace and arrhythmias and to provide evidence that metabolic and inflammatory factors may have a role; walking faster decreased the risk of obesity and inflammation which in turn reduced the risk of arrhythmia. This finding is biologically plausible because cumulative epidemiological studies have shown that walking pace is inversely associated with metabolic factors such as obesity,[18] HbA1c,[19] diabetes[20] and hypertension[21] which, in turn, are associated with the risk of arrhythmias. Our results are also supported by interventional research. Brisk walking interventions have been shown to reduce body weight, BMI and waist circumference,[22] blood pressure,[23] fasting blood glucose[23] and inflammatory markers, as well as improving lipid profiles.[24]

Significant associations between walking pace and arrhythmias were found consistently across a series of subgroups in this study, demonstrating the reliability of the findings. A novel finding was the stronger association between walking pace and arrhythmia observed in women than in men. However, the finding is consistent with previous studies showing that women experience greater benefits from PA than men in relation to incident CVD and CVD mortality.[25] The sex specific difference may be due to greater relative improvements in muscle strength from PA in women than men due to sex differences in muscle fibre type and muscle fibre metabolic, contractile and dynamic function.[26] However, the mechanisms of the sex differences need further investigation. The observed effect modifications by age and BMI were also consistent with previous studies on walking pace and CVD.[27] Interestingly, the larger effect sizes in people with hypertension and multiple chronic diseases suggest that walking at a brisk pace may have a role in secondary, as well as primary, prevention. However, further research is needed.

Clinical implications

This study provides new findings about the association between walking pace and arrhythmias, using a large sample size, prospective design, long follow-up and measuring walking pace.

The study not only used the self-reported assessment of walking pace but also the objective assessment by accelerometer. The accelerometer could counteract the limitations of self-reports, and utilisation of both methods enables us to provide more reliable and validated findings. The ability to investigate the possible mediation roles of metabolic risk factors and inflammation provided insights into the aetiology and pathophysiology of cardiac arrhythmias. These findings imply that the promotion of a faster walking pace may be effective in preventing arrhythmias. Furthermore, our research findings have implications that adopting a faster walking pace to reduce arrhythmia risk may be safe in giving PA recommendations for people with chronic conditions in clinical practice.

Limitations

Our study had some limitations. Firstly, we had access to derived accelerometer data but not the raw data. Therefore, while we were able to investigate time spent walking at slow, average and brisk paces, we could not perform more granular analyses using actual speed. When we derived average walking pace using accelerometer data, only 22 participants were classified as having average or brisk walking pace, which may be due to the fact that, when people report their own walking pace, they only include purposeful walking (going for a walk, walking the dog, walking to get somewhere) whereas accelerometry data include all steps taken during the day and therefore have a long tail of slow steps. These limitations mean that we were unable to perform the analysis of different categories of walking pace with arrhythmias to make a direct comparison between self-reported and accelerometer measured walking pace, but the findings on the protective role of faster slowing pace and higher time spent on the faster slowing pace on arrhythmias could lead to the robust findings in a different way.

Secondly, participants in the accelerometer study were selected based on a constrained random sampling approach, and the response rate was about 45%, raising the potential of selection bias. Thirdly, as with most population cohort studies, the UK Biobank was subject to healthy volunteer bias. As a result, frequency measurements should not be generalised but estimated effect sizes should be generalisable, as shown in previous studies.[28] Fourthly, since UK Biobank participants were aged 40–69 years and primarily of white British origin, caution is needed in generalising the results to other ethnic or age groups, or countries. Fifthly, self-report of the exposure and many covariates may have resulted in imprecise measurement, but errors are unlikely to have occurred systematically in relation to the exposures or outcomes of interest. Finally, while we adjusted for a wide range of confounding factors, residual confounding is a potential limitation of any observational study.

CONCLUSIONS

Faster walking pace may be associated with a lower risk of cardiac arrhythmias, which is partially mediated by metabolic factors and inflammation, especially obesity. The association was more pronounced in younger, female and non-obese people and those with pre-existing LTCs, especially hypertension. These novel findings reinforce the promotion of faster walking pace in PA recommendations and provide evidence of higher risk groups to target.

Contributors PQ, FKH, CAC-M and JPP conceptualised the study. FKH, CAC-M and SGT were responsible for the data collection, data curation, and analysis and interpretation. PQ performed the data analysis, visualisation and wrote the initial manuscript draft. FKH, CAC-M, SGT and JPP revised the manuscript. All authors read and approved the final manuscript and JPP guaranted the contributions of all authors.

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Competing interests None declared.

Patient and public involvement Patients and/or the public were not involved in the design, or conduct, or reporting, or dissemination plans of this research.

Patient consent for publication Not applicable.

Ethics approval This study involves human participants. UK Biobank received ethical approval from the North-West Multi-centre Research Ethics Committee (reference 11/NW/03820). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request. Data analysed in this study was from the UK Biobank and is available upon application to UK Biobank https://www.ukbiobank.ac.uk.

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