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# Impact of migraine on changes in cardiovascular health profile among Hong Kong Chinese women: insights from the MECH-HK cohort study

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## Abstract

**Background** Previous research has shown an association between migraine and cardiovascular diseases (CVDs). However, limited studies have explored the progression of cardiovascular health (CVH) among individuals with migraine. This cohort study aimed to explore the relationship between changes in CVH and migraine among women of Chinese descent in Hong Kong.

**Method** Data from a cohort study titled "Migraine Exposures and Cardiovascular Health in Hong Kong Chinese Women (MECH-HK)" were analysed. A total of 2,603 women, averaging 56.5 ± 8.5 years of age, were selected, all with complete data at baseline and at a follow-up occurring on average 1.27 years later. CVH profile was assessed by an adapted Life's Essential 8, comprising dietary habits, physical activity, nicotine exposure, sleep duration, body mass index (BMI), lipid levels, blood pressure, and stress. Each component was scored from 0 to 100, with overall CVH as the average. CVH levels were categorized as low (0–49), moderate (50–79), and high (80–100), representing poor to excellent health. Changes in CVH were defined as shifts between these categories from baseline to subsequent follow-up. Migraine cases were identified utilizing the International Classification of Headache Disorders 3.

**Result** A total of 275 (10.6%) women were identified as having migraine. By follow-up, both women with and without migraine experienced significant declines in CVH profiles (all p-value < 0.05). In the fully adjusted model, women with migraine had a 1.36 times higher risk (OR 95% CI: 1.33, 1.39) of decline in overall CVH compared to non-migraineurs. They also had a higher likelihood of shifting to worse CVH levels in several individual CVH components, including physical activity (OR: 1.09), nicotine exposure (OR: 4.27), sleep quality (OR: 1.80), blood lipid levels (OR: 1.03), and stress (OR: 1.23) (all p-value < 0.05). Among women with migraine, those experiencing aura had a higher risk of poorer physical activity, greater nicotine exposure, higher BMI, and increased stress than those without aura (all p-value < 0.05).

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**Conclusion** Women with migraine exhibited worse progression in CVH compared to those without migraine. Targeted monitoring and management of CVH-related factors in this population are crucial to reducing their elevated risk of CVDs.

**Keywords** Migraine, Cardiovascular health profile, Adapted Life's Essential 8

## Introduction

Migraine is a debilitating neurological disorder impacting 18–21% of women and 9–10% of men, ranking among the most common causes of disability globally [1, 2]. Epidemiological research has found a significant association between migraine and many cardiovascular diseases (CVDs) [3], such as stroke [4], myocardial infarction [5], venous thromboembolism [6], and atrial fibrillation [7]. While the mechanisms connecting migraine to CVD remain complex and not fully understood, growing evidence indicates that people experiencing migraine often have a poor cardiovascular risk profile [8]. However, limited longitudinal studies have accessed the relationship between migraine and the progression of cardiovascular risk profiles over time. Understanding this relationship may not only reinforce the evidence linking migraine to CVD but also assist in identifying high-risk individuals for timely intervention.

The cardiovascular health (CVH) profile, commonly assessed by the American Heart Association's Life's Essential 8 (LE8) [9], includes a range of health indicators and lifestyle practices critical for assessing cardiovascular wellness. These factors include dietary habits, exercise, nicotine exposure, sleep quality, BMI, lipid levels, glucose levels, and blood pressure. Studies consistently show that higher LE8 scores are correlated with reduced risks of CVD, stroke, heart disease, and overall mortality, while declines in LE8 scores over time are linked to a heightened risk of CVD across diverse populations [10–12]. In addition to LE8, psychological stress is recognized as a significant contributor to the onset and progression of CVDs, with studies demonstrating that stress can trigger cardiac events and heighten the risk of recurrent CVD [13, 14].

Previous studies have explored the associations between CVH profiles and migraine. A UK cohort study discovered that poor CVH was linked to an elevated risk of developing new-onset migraine [15]. Individuals with migraine exhibited more adverse cardiovascular risk indicators, such as unfavourable cholesterol levels [16], raised blood pressure [17], and higher smoking rates [18]. Additionally, studies indicated that migraineurs exhibited a greater susceptibility to physical inactivity, depression, and sleep disturbances [19, 20]. Notably, women with migraine face a greater likelihood of developing CVD than men [21].

Despite these findings, most research has relied on cross-sectional data [19, 22–25] or longitudinal studies where CVH was the exposure and migraine was the outcome [15], leaving a gap in understanding how migraine impacts the long-term trajectory of CVH. A critical question remains: do individuals with migraine experience a more accelerated decline in CVH compared to those without? Investigating this could provide insights into the mechanisms linking migraine and CVD, potentially informing more effective clinical and public health interventions. If migraineurs do experience a faster decline in CVH, personalized management strategies–such as lifestyle modifications and regular cardiovascular health monitoring–could help slow this progression and reduce their future risk of CVD. However, research specifically addressing this issue is currently lacking.

Therefore, this study aims to explore how migraine affects the long-term progression of CVH. We first assessed overall changes in CVH and its individual components among individuals with and without migraine, and then examined the influence of aura on CVH progression within migraineurs. By identifying which CVH components are most susceptible to deterioration in association with migraine or aura, we hope to provide insights that can inform early intervention strategies for those at heightened cardiovascular risk.

## Method

### Study design and population

Data were derived from a prospective cohort study titled "Migraine Exposures and Cardiovascular Health in Hong Kong Chinese Women (MECH-HK)" [26, 27]. Initially, 4,221 women aged 30 years and older were enrolled between October 2019 and December 2020. Participants were eligible if they were free of cardiovascular disease at baseline. Between October 2021 and June 2023, approximately 1.27 years on average after initial enrolment, a follow-up assessment was completed by 3,451 participants. In this study, we included participants with complete CVH metrics and migraine status data at both baseline and follow-up. Participants were excluded if they ① had incomplete CVH metrics at either time point or ② had missing data on migraine status, sociodemographic information, or other relevant variables (Fig. 1).

Sociodemographic, lifestyle, and health-related information were gathered via face-to-face questionnaires conducted through trained research assistants. Additionally, physical examinations and blood tests were performed to obtain biometric data. Informed consent was obtained from all participants, and ethical approval was

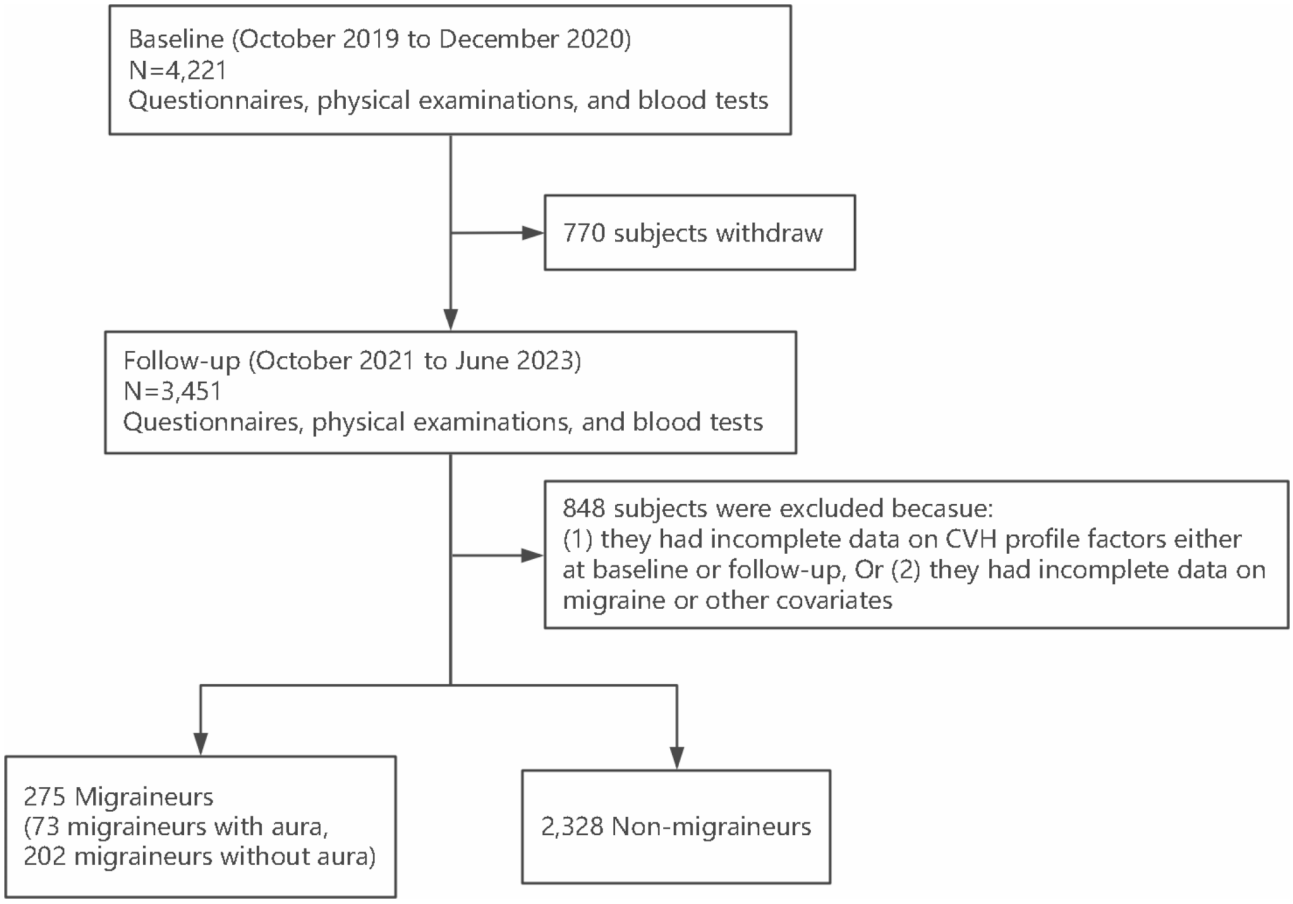


**Fig. 1** Data collection and screening flowchart for analysing the relationship between migraine and CVH profile changes in the MECH-HK cohort study (October 2019 - June 2023)

granted by the Human Research Ethics Review Committee of the Hong Kong Polytechnic University (Reference: HSEARS20171229004) [26, 27].

#### Assessment of the cardiovascular health (CVH) profile

The CVH profile was assessed using a metric consisting of eight components adapted from the LE8 framework [9]. These components included dietary habits, physical activity (PA), nicotine exposure, sleep duration, BMI, lipid levels, blood pressure levels, and stress levels. Fasting blood glucose was excluded due to data unavailability. Stress was incorporated considering its growing acknowledgment as a key factor in CVD risk [13, 14].

Dietary habits were assessed through a questionnaire that assessed participants' consumption frequency across eleven food groups in the previous month [27]. The food groups covered vegetables, fruits, dairy products (including cheese), soy products, eggs, meats, seafood, fish (excluding salted fish), cured meats, cakes, and preserved vegetables. The frequency of intake was classified as 'never,' 'less than once a month,' '1–3 days each month,' '1–3 days each week,' '4–6 days each week,' and 'daily.' Foods such as fish, seafood, bean products, vegetables, and fruits were considered healthy for cardiovascular [28] and were scored from 0 to 5, with higher scores for more frequent intake. Conversely, preserved vegetables, cheese, cured meats, and cakes were considered unhealthy and were scored from 5 to 0 for more frequent consumption. Eggs and meats were excluded from the dietary metric due to the unresolved effect of eggs on cardiovascular risk [29] and the lack of distinction between red and non-red meats in our data, which have conflicting impacts. The total dietary score was derived by adding up all individual component scores, with higher values indicating more healthful dietary habits. These raw scores were then adjusted to a 100-point scale based on quantiles defined by the LE8 scoring criteria [9], with specific scoring details provided in Table 1.

We quantified PA via the International Physical Activity Questionnaire (IPAQ) [30]. To calculate a weekly total, moderate activity minutes were recorded as 1 unit, and vigorous activity minutes were weighted as 2 units. PA scores ranged from 0 to 100 points, with ≥150 min per week corresponding to 100 points, and 0 min corresponding to 0 points. Intermediate ranges aligned with the LE8 framework (Table 1).

**Table 1** Distribution of CVH metric scores from baseline to follow-up in migraineurs and non-migraineurs

| CVH metric factor | Points | Migraineurs (*N* = 275) | | | Non-migraineurs (*N* = 2328) | | |
|---|---|---|---|---|---|---|---|
| | | Baseline | Follow up | *p*-value [a] | Baseline | Follow up | *p*-value [a] |
| Diet | | | | 0.354 | | | 0.657 |
| 1st–24th percentile | 0 | 79 (28.7%) | 88 (32%) | | 426 (18.3%) | 391 (16.8%) | |
| 25th–49th percentile | 25 | 57 (20.7%) | 34 (12.4%) | | 439 (18.9%) | 435 (18.7%) | |
| 50th–74th percentile | 50 | 88 (32%) | 88 (32%) | | 814 (35%) | 846 (36.3%) | |
| 75th–94th percentile | 80 | 42 (15.3%) | 49 (17.8%) | | 487 (20.9%) | 496 (21.3%) | |
| ≥ 95th percentile | 100 | 9 (3.3%) | 16 (5.8%) | | 162 (7%) | 160 (6.9%) | |
| Physical activity (min/week) | | | | 0.030 | | | < 0.001 |
| 0 | 0 | 28 (10.2%) | 44 (16%) | | 253 (10.9%) | 342 (14.7%) | |
| 1–29 | 20 | 0 (0%) | 5 (1.8%) | | 13 (0.6%) | 11 (0.5%) | |
| 30–59 | 40 | 7 (2.5%) | 3 (1.1%) | | 42 (1.8%) | 58 (2.5%) | |
| 60–89 | 60 | 8 (2.9%) | 8 (2.9%) | | 46 (2%) | 71 (3%) | |
| 90–119 | 80 | 7 (2.5%) | 11 (4%) | | 50 (2.1%) | 85 (3.7%) | |
| 120–149 | 90 | 30 (10.9%) | 17 (6.2%) | | 294 (12.6%) | 121 (5.2%) | |
| ≥ 150 | 100 | 195 (70.9%) | 187 (68%) | | 1630 (70%) | 1640 (70.4%) | |
| Nicotine exposure | | | | 0.041 | | | < 0.001 |
| Current smoker | 0 | 7 (2.5%) | 8 (2.9%) | | 29 (1.2%) | 24 (1%) | |
| Former smoker (quit < 1y) | 25 | 0 (0%) | 5 (1.8%) | | 0 (0%) | 0 (0%) | |
| Former smoker (quit 1–<5y) and second-hand exposure | 30 | 1 (0.4%) | 5 (1.8%) | | 1 (0%) | 1 (0%) | |
| Former smoker (quit 1–<5y) | 50 | 5 (1.8%) | 11 (4%) | | 1 (0%) | 2 (0.1%) | |
| Former smoker (quit > = 5y) and second-hand exposure | 55 | 3 (1.1%) | 3 (1.1%) | | 14 (0.6%) | 7 (0.3%) | |
| Former smoker (quit > = 5y) | 75 | 4 (1.5%) | 6 (2.2%) | | 21 (0.9%) | 25 (1.1%) | |
| Never smoker but second-hand exposure | 80 | 51 (18.5%) | 35 (12.7%) | | 330 (14.2%) | 207 (8.9%) | |
| Never smoker | 100 | 204 (74.2%) | 202 (73.5%) | | 1932 (83%) | 2062 (88.6%) | |
| Sleep health (average hours/night) | | | | 0.144 | | | 0.009 |
| < 4 | 0 | 3 (1.1%) | 9 (3.3%) | | 31 (1.3%) | 62 (2.7%) | |
| 4–<5 | 20 | 17 (6.2%) | 22 (8%) | | 122 (5.2%) | 144 (6.2%) | |
| 5–<6 or ≥ 10 | 40 | 41 (14.9%) | 52 (18.9%) | | 361 (15.5%) | 369 (15.9%) | |
| 6–<7 | 70 | 108 (39.3%) | 104 (37.8%) | | 767 (32.9%) | 789 (33.9%) | |
| 9–<10 | 90 | 5 (1.8%) | 3 (1.1%) | | 56 (2.4%) | 37 (1.6%) | |
| 7–<9 | 100 | 101 (36.7%) | 85 (30.9%) | | 991 (42.6%) | 927 (39.8%) | |
| Body mass index (kg/m$^2$) | | | | 0.790 | | | 0.566 |
| ≥ 35.0 | 0 | 1 (0.4%) | 3 (1.1%) | | 11 (0.5%) | 10 (0.4%) | |
| 30.0-34.9 | 25 | 16 (5.8%) | 14 (5.1%) | | 63 (2.7%) | 69 (3%) | |
| 25.0-29.9 | 50 | 47 (17.1%) | 49 (17.8%) | | 484 (20.8%) | 451 (19.4%) | |
| 23.0-24.9 | 75 | 52 (18.9%) | 43 (15.6%) | | 470 (20.2%) | 496 (21.3%) | |
| < 23 | 100 | 159 (57.8%) | 166 (60.4%) | | 1300 (55.8%) | 1302 (55.9%) | |
| Blood lipids (mg/dL) | | | | 0.012 | | | < 0.001 |
| Non-HDL ≥ 220 or 190–219 with treated | 0 | 9 (3.3%) | 7 (2.5%) | | 51 (2.2%) | 57 (2.4%) | |
| Non-HDL 190–219 or 160–189 with treated | 20 | 18 (6.5%) | 16 (5.8%) | | 131 (5.6%) | 180 (7.7%) | |
| Non-HDL 160–189 or 130–159 with treated | 40 | 46 (16.7%) | 57 (20.7%) | | 405 (17.4%) | 489 (21%) | |
| Non-HDL 130–159 | 60 | 59 (21.5%) | 83 (30.2%) | | 628 (27%) | 622 (26.7%) | |
| Non-HDL < 130 with treated | 80 | 22 (8%) | 25 (9.1%) | | 192 (8.2%) | 222 (9.5%) | |
| Non-HDL < 130 | 100 | 121 (44%) | 87 (31.6%) | | 921 (39.6%) | 758 (32.6%) | |
| Blood pressure (SBP or DBP), mmHg | | | | 0.247 | | | < 0.001 |
| ≥ 160 or ≥ 100 | 0 | 6 (2.2%) | 3 (1.1%) | | 123 (5.3%) | 70 (3%) | |
| 140–159 or 90–99 treated | 5 | 5 (1.8%) | 3 (1.1%) | | 105 (4.5%) | 81 (3.5%) | |
| 140–159 or 90–99 | 25 | 24 (8.7%) | 20 (7.3%) | | 281 (12.1%) | 241 (10.4%) | |
| 130–139 or 80–89 treated | 30 | 13 (4.7%) | 11 (4%) | | 80 (3.4%) | 101 (4.3%) | |
| 130–139 or 80–89 | 50 | 60 (21.8%) | 49 (17.8%) | | 473 (20.3%) | 428 (18.4%) | |
| 120–129 / < 80 treated | 55 | 5 (1.8%) | 2 (0.7%) | | 39 (1.7%) | 51 (2.2%) | |
| 120–129 / < 80 | 75 | 34 (12.4%) | 31 (11.3%) | | 276 (11.9%) | 288 (12.4%) | |

**Table 1** (continued)

| CVH metric factor | Points | Migraineurs (*N* = 275) | | | Non-migraineurs (*N* = 2328) | | |
|---|---|---|---|---|---|---|---|
| | | Baseline | Follow up | *p*-value [a] | Baseline | Follow up | *p*-value [a] |
| < 120 / < 80 treated | 80 | 4 (1.5%) | 12 (4.4%) | | 33 (1.4%) | 52 (2.2%) | |
| < 120 / < 80 | 100 | 124 (45.1%) | 144 (52.4%) | | 918 (39.4%) | 1016 (43.6%) | |
| Stress (PSS-14 score) | | | | 0.805 | | | 0.010 |
| 41–56 | 0 | 1 (0.4%) | 2 (0.7%) | | 10 (0.4%) | 9 (0.4%) | |
| 31–40 | 25 | 39 (14.2%) | 43 (15.6%) | | 197 (8.5%) | 193 (8.3%) | |
| 21–30 | 50 | 191 (69.5%) | 191 (69.5%) | | 1506 (64.7%) | 1408 (60.5%) | |
| 11–20 | 75 | 42 (15.3%) | 36 (13.1%) | | 561 (24.1%) | 659 (28.3%) | |
| 0–10 | 100 | 2 (0.7%) | 3 (1.1%) | | 54 (2.3%) | 59 (2.5%) | |

Abbreviations: HDL, high-density lipoprotein; DBP, diastolic blood pressure; SBP, systolic blood pressure; PSS-14, Chinese version of the 14-item Perceived Stress Scale

[a] Pearson's Chi-squared test/Fisher's exact test by grouping the scores into four categories: <30, 30–50, 51–80, and >80; p-value < 0.05 indicates a significant shift in the distribution of scores from baseline to follow-up

Nicotine exposure was measured through self-reported cigarette use or second-hand exposure. Current smokers scored 0 points, non-smokers scored 100 points, and former smokers were assigned intermediate scores according to the LE8 criteria. Sleep health was measured via participants' reported average sleep duration per night. As defined by the LE8 framework [9], a sleep duration of less than 4 h scored 0 points, while a duration of 7 to less than 9 h scored 100 points, with other durations assigned intermediate scores.

BMI was obtained as weight (kg)/height$^2$ (m$^2$). Individual' height was assessed barefoot utilizing a stadiometer, whereas weight was assessed by the Inbody 270 body composition analyser, ensuring the removal of bulk clothing and accessories. The Chinese BMI scoring standards were applied, with a BMI of ≥ 35.0 scoring 0 points, a BMI of < 23 scoring 100 points, and other values receiving intermediate scores.

Blood lipid levels were evaluated using non-HDL cholesterol and scored based on the LE8 criteria. For individuals managing cholesterol levels with medication, an additional 20 points were deducted from their score. Blood pressure was assessed using both systolic and diastolic readings. Optimal levels (< 120/<80 mmHg) scored 100 points, while readings of ≥ 160 or ≥ 100 mmHg scored 0 points, with intermediate values assigned according to the LE8 framework. For participants with treated hypertension, an additional 20 points were deducted from their score.

Stress was assessed using the widely validated Perceived Stress Scale (PSS) [31], where participants rated 14 items on a scale from 0 ("not at all") to 4 ("very frequently"). The overall PSS score was calculated by summing these item scores, resulting in a total score range of 0 to 56, with higher scores indicating greater perceived stress. For integration into the CVH metric and alignment with the LE8 framework, raw PSS scores were converted to a 0–100 scale: scores of 0–10 were allocated 100 points, 11–20 were allocated 75 points, 21–30 were allocated 50 points, 31–40 were allocated 25 points, and scores above 40 received 0 points, as detailed in Table 1.

In summary, each component was assigned a score on a 0-100 scale, and the overall CVH metric score was assessed by averaging the eight individual component scores. The overall CVH score, as well as the individual component scores, were classified into three levels: Low (0–49), Moderate (50–79), and High (80–100) [15]. Changes of the CVH profile were defined as shifts in these categories from baseline to the follow-up and were classified into three outcomes: "decrease", "stable", or "increase". A decrease indicated that the CVH or individual component category at follow-up was lower than at baseline, while an increase represented an improvement. A stable outcome meant the category remained unchanged between baseline and follow-up.

#### Ascertainment of migraine and aura

Migraine exposure was determined through a multi-step process, with detailed procedures provided in our previously published cohort profile [26]. Initially, participants were surveyed regarding their headache occurrences over the previous year via a structured questionnaire. Participants who responded with 'Yes' were then instructed to finish the ID Migraine™, a screening tool for migraine identification [32]. Participants who reported no headache occurrences were queried about any previous migraine diagnosis made by a physician. Individuals who either screened positive on the ID Migraine™ or reported a pre-existing diagnosis of migraine were classified as potential migraineurs. Participants who screened negative in the ID Migraine™ or lacked any prior diagnosis underwent additional evaluation to determine whether they had experienced migraine episodes at any point in their lifetime. This evaluation focused on identifying key symptoms, including phonophobia, photophobia, nausea, vomiting, or neurologic disturbances (e.g., visual flashes

or tingling sensations) occurring within an hour before a headache. Participants who were confirmed to have experienced migraine attacks based on these criteria were also included as potential migraineurs [26]. A neurologist on our team carried out follow-up telephone interviews with these potential migraineurs to confirm the diagnosis. Based on the International Classification of Headache Disorders 3 (ICHD-3) [33], participants were classified into four categories: non-headache, non-migraine headache, probable migraine, and migraine. For the purpose of this study, individuals with either migraine or probable migraine were grouped as migraineurs, while those with non-migraine headaches or no headaches were assigned to the non-migraineur group. In the sensitivity analysis, participants who reported other types of headaches were excluded to assess the robustness of our results.

In accordance with the ICHD-3 criteria [33], migraine participants were categorized into those with aura and those without aura. Migraine with aura is defined by the presence of temporary neurological disturbances, including visual alterations, sensory disruptions, or language impairments, that precede the headache onset. Conversely, migraine without aura refers to migraine attacks that occur without these preceding neurological symptoms. In this study, individuals experiencing both types of episodes interchangeably were included in the "migraine with aura" group [26].

#### Ascertainment of other covariates

All covariate data were collected through questionnaire assessment at baseline. Demographic and socioeconomic covariates included age, menopausal status, employment status, educational level, marital status, and household income. Age was treated as a continuous variable. Menopausal status was classified as "Yes" for postmenopausal participants and "No" for those who were pre-menopausal. Employment status was divided into two groups: participants employed full-time, part-time, or self-employed were categorized as "Yes," while those unemployed for more than a year, recently unemployed (less than a year), retired, or unable to work were categorized as "No." Education level was categorized into three subgroups: "Primary school or lower," "Secondary school or pre-college," and "College or higher." Marital status was categorized as "Married or cohabiting" or "Never married, divorced, separated, or widowed." Monthly household income was classified into three subgroups: "≤14,000 HKD/month," ">14,000 and ≤35,000 HKD/month," and ">35,000 HKD/month."

In addition to the lifestyle factors analysed as part of the LE8 framework, we included two additional lifestyle factors as covariates: fatigue level and drinking status. Fatigue was measured using an 11-point self-perceived fatigue scale ranging from 0 (no fatigue) to 10 (most severe fatigue) [27]. Participants with scores at or above the median were categorized as having high fatigue [27], while those scoring below the median were considered to have no or low fatigue. Drinking status was divided into two categories: "Drinker" and "Never alcohol drinker."

We included several disease-related covariates based on participants' self-reported diagnoses and treatment status. These covariates covered cancer, hypertension, diabetes, and hyperlipidaemia. Hypertension status was categorized as "No hypertension," "Hypertension treated," and "Hypertension untreated." Similarly, diabetes was classified as "No diabetes," "Diabetes treated," and "Diabetes untreated," while hyperlipidaemia was grouped into "No hyperlipidaemia," "Hyperlipidaemia treated," and "Hyperlipidaemia untreated." Cancer status was recorded as "Yes" for participants with a current or past diagnosis, and we assumed all participants with cancer would receive treatment. Those without any cancer diagnosis were recorded as "No."

#### Statistical analysis

Baseline characteristics were compared across subgroups based on migraine status (migraine versus non-migraine) and overall CVH categories (low, moderate, and high). Differences in continuous variables between migraine status groups were evaluated using two-sample t-tests, while differences across CVH categories were assessed using one-way ANOVA. Categorical variables were examined employing Chi-square tests or Fisher's exact tests, selected based on appropriateness. For multiple comparisons across the three CVH subgroups (low, moderate, and high), Bonferroni correction was applied to control for Type I errors. This resulted in an adjusted significance threshold of p-value < 0.0167 (0.05 / 3), covering the three pairwise comparisons: low versus moderate, low versus high, and moderate versus high. To examine differences in the distribution of CVH metric scores from baseline to follow-up, we used Chi-square tests or Fisher's exact tests, grouping the scores into four categories: <30, 30–50, 51–80, and >80. Additionally, paired t-tests were utilized to evaluate changes in the continuous measurements of individual CVH components (e.g., blood pressure, cholesterol) between the two time points.

Multinomial logistic regression analyses were conducted to assess the impact of migraine on changes in CVH profiles from baseline to follow-up. Four models with progressive adjustments were used: ① Model 1 (unadjusted) evaluated the crude association between migraine and CVH changes without adjusting for any covariates; ② Model 2 adjusted for age only; ③ Model 3 further adjusted for multiple factors, including age, menopausal status, educational level, employment situation, marital status, household income, fatigue level, and drinking status; ④ Model 4 (fully adjusted) included

all covariates from Model 2 and 3, along with additional disease-related variables, including cancer, hypertension, diabetes, and hyperlipidaemia. These analyses were conducted for both the overall CVH profile and each individual CVH component.

To further explore whether the impact of migraine on changes in overall CVH categories varied across subgroups, we conducted both subgroup and interaction term analyses, adjusting for relevant covariates. In the subgroup analysis, we stratified the dataset into groups based on various socioeconomic factors (including age, marital status, education level, employment status, and household income), women's menopausal status, and disease-related factors (including cancer, hypertension, diabetes, hyperlipidaemia). Separate analyses were performed for each subgroup, and we calculated subgroup-specific odds ratios (ORs) to assess how migraine's effect on CVH progression differed across these groups. We then used the entire dataset to build a full model with interaction terms to formally test the significance of interaction effects. Combining stratified analyses with interaction term modelling provided a better understanding of potential heterogeneity in the impact of migraine across different population subgroups.

Several sensitivity analyses were undertaken to confirm the results' reliability. First, we adjusted the weights of the overall CVH score by grouping the eight components into two clusters: lifestyle components (dietary habits, PA, nicotine exposure, sleep duration, and stress) and clinical components (blood pressure, BMI, and lipid levels). In the main analysis, equal weights were assigned, with 50% allocated to lifestyle components and 50% to clinical components, and each of the eight individual components contributing equally to the final CVH score. For the sensitivity analysis, we explored several alternative weighting schemes, including 60% lifestyle and 40% clinical, 70% lifestyle and 30% clinical, 40% lifestyle and 60% clinical, and 30% lifestyle and 70% clinical. Second, participants who reported other types of headaches at baseline were excluded from the non-migraineur group. Third, changes in the CVH profile were evaluated using crude scores rather than categorical CVH classifications to define "increases" or "decreases." Statistical significance was defined by a two-sided p-value $<0.05$, and data were analysed utilizing R (version 4.1.2).

## Result

### Basic characteristics of participants

The final analysis included 2,603 participants, of whom 275 had migraines and 2,328 did not, with a mean age of $56.5 \pm 8.5$ years. Baseline characteristics were categorized by migraine status (migraine vs. no migraine) and overall CVH profiles (low, moderate, and high CVH), see in in Table 2. In comparison with non-migraineurs, individuals with migraine were younger, had higher level of education, were unlikely to be menopausal, were more prone to fatigue, had a higher proportion of drinkers, and had a lower prevalence of diabetes (all p-value $<0.05$). No significant differences were found in marital status, household income, employment status, hypertension, hyperlipidaemia, or cancer prevalence between migraineurs and non-migraineurs.

Demographic characteristics varied across CVH profile groups. Participants with moderate CVH were significantly younger than those with low CVH (p-value $<0.0167$). Women with low CVH were less likely to be married or cohabiting, had higher employment rates, experienced more fatigue, and had a higher prevalence of hypertension and hyperlipidaemia compared to individuals within the moderate and high CVH categories (all p-value $<0.0167$). Moreover, the prevalence of hypertension and hyperlipidaemia was highest in the high CVH group among the three groups (p-value $<0.0167$). No significant difference was found in educational level, household income, proportion of drinkers, diabetes, or cancer prevalence across the CVH groups.

### Changes in cardiovascular health profile from baseline to follow-up

At baseline, migraineurs had poorer CVH profiles compared to non-migraineurs (Table S1), with a median CVH score of 71.9, significantly lower than the 73.1 observed in non-migraineurs (Fig. 2A). By the follow-up, both groups showed a significant decline in their overall CVH scores, with migraineurs' median score dropping to 70.6 and non-migraineurs' to 71.9 (all p-value $<0.05$, Fig. 2A).

Both groups exhibited significant changes in individual CVH component score distributions at follow-up, particularly in physical activity, nicotine exposure, and blood lipid levels (all p-value $<0.05$, Table 1). When examining continuous measurements of individual CVH components, we found that average moderate physical activity decreased from 308.4 to 260.6 min in migraineurs and from 316.8 to 289.3 min in non-migraineurs (all p-value $<0.05$, Table S3). Average sleep duration decreased from 6.4 to 6.2 h for migraineurs and from 6.5 to 6.4 h for non-migraineurs (all p-value $<0.05$). Both groups also experienced increases in non-HDL cholesterol concentrations, with migraineurs' levels rising from 133.3 to 138.4 mg/dL and non-migraineurs' from 133.2 to 138.4 mg/dL (all p-value $<0.05$).

The CVH profiles were further classified into three categories: low CVH (0–49 points), moderate CVH (50–79 points), and high CVH (80–100 points). Analyses focused on changes within these categories to determine whether participants' CVH profiles increased, remained stable, or decreased by the follow-up. As illustrated in Fig. 2B, a decline in overall CVH scores was observed in 18% (50

**Table 2** Baseline characteristics of participants by migraine status and CVH profile categories

| Characteristics [a] | Total subjects (*N* = 2603) | Migraineurs (*N* = 275) | Non-migraineurs (*N* = 2328) | *p*-value | Low CVH (*N* = 57) | Moderate CVH (*N* = 1896) | High CVH (*N* = 650) | *p*-value |
|---|---|---|---|---|---|---|---|---|
| Age, years | 56.5 ± 8.5 | 53.9 ± 8.9 | 56.8 ± 8.4 | < 0.001 | 56.5 ± 8.8 | 54.4[†] ± 9.2 | 56.7 ± 8.4 | 0.033 |
| Marital status | | | | 0.643 | | | | 0.001 |
| Married or cohabiting | 1956 (75.14%) | 72 (26.18%) | 575 (24.7%) | | 31 (54.39%) | 1433[†] (75.58%) | 492[†] (75.69%) | |
| Never married/Divorce/separation/ widowhood | 647 (24.86%) | 203 (73.82%) | 1753 (75.3%) | | 26 (45.61%) | 463[†] (24.42%) | 158[†] (24.31%) | |
| Educational level | | | | 0.020 | | | | 0.242 |
| Primary school or lower | 191 (7.34%) | 17 (6.18%) | 174 (7.47%) | | 3 (5.26%) | 149 (7.86%) | 39 (6%) | |
| Secondary school or pre-college | 1547 (59.43%) | 146 (53.09%) | 1401 (60.18%) | | 33 (57.89%) | 1138(60.02%) | 376 (57.85%) | |
| College or higher | 865 (33.23%) | 112 (40.73%) | 753 (32.35%) | | 21 (36.84%) | 609 (32.12%) | 235 (36.15%) | |
| Household income, HKD/month | | | | 0.147 | | | | 0.075 |
| ≤ 14,000 | 733 (28.16%) | 64 (23.27%) | 669 (28.74%) | | 11 (19.3%) | 546 (28.8%) | 176 (27.08%) | |
| > 14,000 and ≤ 35,000 | 993 (38.15%) | 109 (39.64%) | 884 (37.97%) | | 24 (42.11%) | 739 (38.98%) | 230 (35.38%) | |
| > 35,000 | 877 (33.69%) | 102 (37.09%) | 775 (33.29%) | | 22 (38.6%) | 611 (32.23%) | 244 (37.54%) | |
| Employed | 1292 (49.64%) | 150 (54.55%) | 1142 (49.05%) | 0.097 | 44 (77.19%) | 946[†] (49.89%) | 302[†] (46.46%) | < 0.001 |
| Menopause | 2001 (76.87%) | 186 (67.64%) | 1815 (77.96%) | < 0.001 | 45 (78.95%) | 1391 (73.36%) | 490 (75.38%) | 0.413 |
| High Fatigue [b] | 1330 (51.09%) | 187 (68%) | 1143 (49.1%) | < 0.001 | 37 (64.91%) | 1022[†] (53.9%) | 271[†] (41.69%) | < 0.001 |
| Never alcohol drinker | 1079 (41.45%) | 96 (34.91%) | 983 (42.23%) | 0.024 | 19 (33.33%) | 780 (41.14%) | 280 (43.08%) | 0.312 |
| Hypertension | | | | 0.247 | | | | < 0.001 |
| No hypertension | 2199 (84.48%) | 241 (87.64%) | 1958 (84.11%) | | 37 (64.91%) | 1542[†] (81.33%) | 620[†‡](95.38%) | |
| Hypertension treated | 250 (9.6%) | 19 (6.91%) | 231 (9.92%) | | 12 (21.05%) | 223[†] (11.76%) | 15[†‡] (2.31%) | |
| Hypertension untreated | 154 (5.92%) | 15 (5.45%) | 139 (5.97%) | | 8 (14.04%) | 131[†] (6.91%) | 15[†‡] (2.31%) | |
| Diabetes | | | | 0.038 | | | | 0.050 |
| No Diabetes | 2431 (93.39%) | 266 (96.73%) | 2165 (93%) | | 52 (91.23%) | 1758 (92.72%) | 621 (95.54%) | |
| Diabetes treated | 86 (3.3%) | 6 (2.18%) | 80 (3.44%) | | 4 (7.02%) | 68 (3.59%) | 14 (2.15%) | |
| Diabetes untreated | 86 (3.3%) | 3 (1.09%) | 83 (3.57%) | | 1 (1.75%) | 70 (3.69%) | 15 (2.31%) | |
| Hyperlipidaemia | | | | 0.803 | | | | < 0.001 |
| No Hyperlipidaemia | 1800 (69.15%) | 189 (68.73%) | 1611 (69.2%) | | 20 (35.09%) | 1254[†] (66.14%) | 526[†‡] (80.92%) | |
| Hyperlipidaemia treated | 274 (10.53%) | 32 (11.64%) | 242 (10.4%) | | 12 (21.05%) | 225[†] (11.87%) | 37[†‡] (5.69%) | |
| Hyperlipidaemia untreated | 529 (20.32%) | 54 (19.64%) | 475 (20.4%) | | 25 (43.86%) | 417[†] (21.99%) | 87[†‡] (13.38%) | |
| Cancer | 151 (5.8%) | 14 (5.09%) | 137 (5.88%) | 0.692 | 2 (3.51%) | 111 (5.85%) | 38 (5.85%) | 0.899 |

[†] Significant differences ($p < 0.0167$) were observed between the Moderate CVH or High CVH groups compared to the Low CVH group

[‡] Significant differences ($p < 0.0167$) were observed between the Moderate CVH group and the High CVH group

[a] Continuous and categorical variables were shown as mean ± standard deviation and number (percentage), respectively; Pearson's Chi-squared test, Fisher's exact test, Two-Sample t-test, or One-Way ANOVA were used, as appropriate

[b] High fatigue was defined as scores at or above the median level

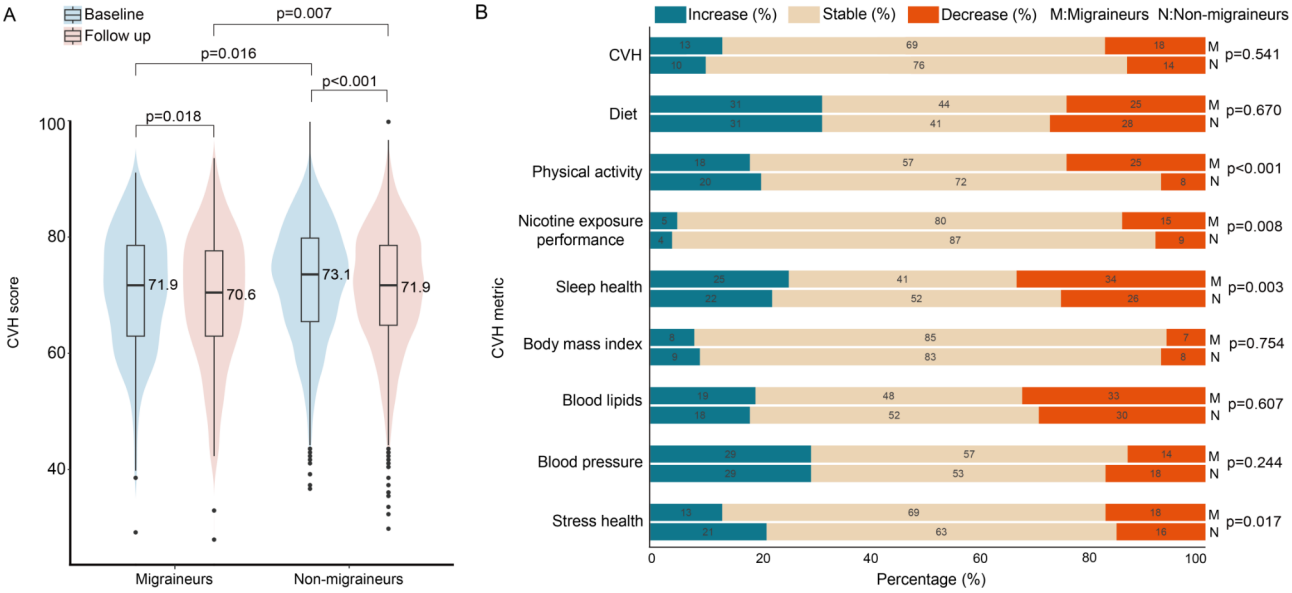


**Fig. 2** Overall CVH scores and changes in CVH profile from baseline to the follow-up for migraineurs and non-migraineurs. (**A**) Violin plot for the distribution of overall CVH scores for migraineurs and non-migraineurs at baseline and follow-up. (**B**) Bar plot for the percentage of participants whose overall CVH category or individual components either increased, remained stable, or decreased from baseline to follow-up

out of 275) of migraineurs and 14% (326 out of 2,328) of non-migraineurs. Migraine participants were more likely to experience reductions in physical activity and sleep health, along with higher nicotine exposure, whereas non-migraine individuals showed greater increases in stress health compared to those with migraines (Fig. 2B).

### Impact of migraine on changes in cardiovascular health profile

Table 3 showed the association between shifts in CVH categories and migraine. In the fully adjusted model (Model 4), migraine was significantly associated with a decrease in overall CVH category compared to non-migraineurs, with an odds ratio (OR) of 1.36 (95% CI: 1.33, 1.39), indicating that migraineurs had a 36% higher likelihood of experiencing a reduction in CVH compared to non-migraineurs. This result closely aligned with the unadjusted model, which reported an OR of 1.35 (95% CI: 1.18, 1.54).

Further analysis of individual CVH components revealed a significant correlation between migraine and unfavourable progression in physical activity, nicotine exposure, and stress levels. In the unadjusted model, the ORs for these associations were 1.15 (95% CI: 1.00, 1.33), 4.35 (95% CI: 2.80, 6.76), and 1.33 (95% CI: 1.14, 1.55), respectively. After adjusting for additional covariates, these associations were slightly attenuated, with ORs of 1.09 (95% CI: 1.06, 1.11), 4.27 (95% CI: 3.87, 4.71), and 1.23 (95% CI: 1.19, 1.26) in the fully adjusted model.

Migraine was inversely related to declines in diet, BMI, and blood pressure categories, with fully adjusted ORs of 0.77 (95% CI: 0.75, 0.78), 0.70 (95% CI: 0.68, 0.72), and 0.85 (95% CI: 0.83, 0.87), respectively. For sleep and blood lipids, migraineurs were more likely to experience both increases and decreases in these categories with both fully adjusted ORs larger than 1 (all p-value < 0.05), indicating greater variability and less stability in these metrics among migraineurs. For all these outcomes, similar findings were consistently observed in Models 2 and 3. In addition, the effects of other covariates in the adjusted models were provided in the supplementary material (Table S7).

### Impact of aura on changes in cardiovascular health profile among migraineurs

Among the 275 migraine sufferers, 73 experienced migraine with aura. At baseline, no significant difference was detected in the distribution of scores across each CVH metric component between those with and without aura (Table S2). By follow-up, both groups showed reductions in their overall CVH scores. For those with aura, the median score decreased from 71.9 to 69.4 points, while those without aura saw a decrease from 71.9 to 71.3 points (Fig. S1, all p-value < 0.05).

Table 4 showed the impact of aura on changes in overall CVH and each CVH component among migraineurs. The results indicated that migraine with aura was associated with a slightly higher risk of decline in the overall CVH category compared to those without aura, with an OR of 1.02. Migraineurs with aura were significantly linked to a higher risk of decreases in PA, nicotine exposure, BMI, and stress categories in comparison with migraine without aura, with ORs of 2.81 (95% CI: 2.61, 3.02), 1.25 (95% CI: 1.11, 1.41), 1.85 (95% CI: 1.63, 2.11), and 1.06 (95% CI:

**Table 3** Impact of migraine on changes in overall CVH and individual components categories (non-migraine as the referent)

| Outcome | Exposure | Model 1 [a] | | Model 2 [b] | | Model 3 [c] | | Model 4 [d] | |
|---|---|---|---|---|---|---|---|---|---|
| | | OR (95% CI) | *P*-value | OR (95% CI) | *P*-value | OR (95% CI) | *P*-value | OR (95% CI) | *P*-value |
| CVH category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.35 (1.18, 1.54) | < 0.001 | 1.33 (1.31, 1.34) | < 0.001 | 1.35 (1.33, 1.38) | < 0.001 | 1.36 (1.33, 1.39) | < 0.001 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.36 (0.93, 1.99) | 0.11 | 1.33 (0.58, 3.05) | 0.502 | 1.28 (0.98, 1.67) | 0.075 | 1.28 (0.98, 1.68) | 0.071 |
| Diet category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 0.71 (0.65, 0.79) | < 0.001 | 0.74 (0.73, 0.75) | < 0.001 | 0.76 (0.75, 0.78) | < 0.001 | 0.77 (0.75, 0.78) | < 0.001 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.03 (0.76, 1.38) | 0.862 | 1.07 (0.56, 2.08) | 0.83 | 1.12 (0.88, 1.42) | 0.374 | 1.13 (0.88, 1.44) | 0.340 |
| Physical activity category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.15 (1, 1.33) | 0.051 | 1.06 (1.04, 1.07) | < 0.001 | 1.09 (1.06, 1.11) | < 0.001 | 1.09 (1.06, 1.11) | < 0.001 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.16 (0.77, 1.77) | 0.476 | 1.03 (0.45, 2.35) | 0.947 | 0.98 (0.75, 1.27) | 0.859 | 0.98 (0.75, 1.27) | 0.867 |
| Nicotine exposure category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 4.35 (2.8, 6.76) | < 0.001 | 3.74 (3.55, 3.94) | < 0.001 | 4.55 (4.13, 5.02) | < 0.001 | 4.27 (3.87, 4.71) | < 0.001 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 2.61 (1.04, 6.56) | 0.041 | 2.16 (0.27, 17.11) | 0.464 | 2.17 (0.62, 7.58) | 0.226 | 2.05 (0.59, 7.15) | 0.26 |
| Sleep health category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.73 (1.55, 1.93) | < 0.001 | 1.73 (1.71, 1.75) | < 0.001 | 1.79 (1.76, 1.83) | < 0.001 | 1.8 (1.77, 1.84) | < 0.001 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.51 (1.09, 2.1) | 0.013 | 1.53 (0.75, 3.11) | 0.238 | 1.56 (1.24, 1.97) | < 0.001 | 1.56 (1.24, 1.97) | < 0.001 |
| BMI category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 0.75 (0.63, 0.9) | 0.002 | 0.72 (0.71, 0.74) | < 0.001 | 0.72 (0.7, 0.74) | < 0.001 | 0.7 (0.68, 0.72) | < 0.001 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.25 (0.75, 2.08) | 0.395 | 1.3 (0.38, 4.39) | 0.677 | 1.26 (0.84, 1.91) | 0.266 | 1.27 (0.84, 1.91) | 0.257 |
| Blood lipids category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 0.97 (0.84, 1.12) | 0.672 | 1 (0.99, 1.01) | 0.739 | 1.03 (1.01, 1.04) | 0.001 | 1.03 (1.01, 1.05) | 0.001 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.1 (0.72, 1.67) | 0.665 | 1.26 (0.44, 3.6) | 0.668 | 1.3 (1.06, 1.59) | 0.01 | 1.28 (1.05, 1.57) | 0.016 |
| Blood pressure category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 0.77 (0.69, 0.85) | < 0.001 | 0.82 (0.81, 0.83) | < 0.001 | 0.85 (0.83, 0.87) | < 0.001 | 0.85 (0.83, 0.87) | < 0.001 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1 (0.73, 1.36) | 0.983 | 1.08 (0.53, 2.17) | 0.838 | 1.11 (0.83, 1.49) | 0.467 | 1.12 (0.84, 1.5) | 0.439 |
| Stress category | | | | | | | | | |
| Decrease [e] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 1.33 (1.14, 1.55) | < 0.001 | 1.29 (1.26, 1.31) | < 0.001 | 1.22 (1.19, 1.26) | < 0.001 | 1.23 (1.19, 1.26) | 0.015 |
| Increase [f] | Non-migraine | Referent | | Referent | | Referent | | Referent | |
| | Migraine | 0.99 (0.61, 1.61) | 0.98 | 0.93 (0.37, 2.38) | 0.887 | 0.81 (0.56, 1.17) | 0.272 | 0.82 (0.57, 1.19) | 0.297 |

Abbreviations: OR, Odds ratio; CI, Confidence interval; CVH, Cardiovascular health

[a] Model 1 unadjusted model

[b] Model 2 adjusted for age

[c] Model 3 adjusted for age, menopause status, marital status, household income, educational level, employment status, fatigue level, and drinking status

[d] Model 4 adjusted for variables in Model 3, hypertension, diabetes, hyperlipidaemia, and cancer

[e] Both the overall CVH profile and individual CVH components were categorized into three levels: Low (0–49 points), Moderate (50–79 points), and High (80–100 points). Decrease indicates that the overall CVH or individual component category at follow-up was lower than at baseline

[f] Increase means that the overall CVH or individual component category at the follow-up was higher than that at baseline

**Table 4** Impact of aura on changes in overall CVH and individual components categories in migraineurs (migraineurs without aura as the referent)

| Outcome | Exposure | Model 1 [a] | | Model 2 [b] | | Model 3 [c] | | Model 4 [d] | |
|---|---|---|---|---|---|---|---|---|---|
| | | OR | *P*-value | OR | *P*-value | OR | *P*-value | OR | *P*-value |
| CVH category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 1.1 (0.74, 1.64) | 0.638 | 1.13 (1.1, 1.17) | < 0.001 | 1.04 (0.98, 1.11) | 0.233 | 1.02 (0.96, 1.09) | 0.490 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.65 (0.27, 1.56) | 0.332 | 0.67 (0.08, 5.36) | 0.702 | 0.53 (0.24, 1.14) | 0.105 | 0.52 (0.23, 1.13) | 0.098 |
| Diet category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.87 (0.63, 1.2) | 0.391 | 0.86 (0.83, 0.89) | < 0.001 | 0.87 (0.81, 0.93) | < 0.001 | 0.85 (0.79, 0.91) | < 0.001 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.85 (0.45, 1.62) | 0.622 | 0.85 (0.15, 4.75) | 0.852 | 0.97 (0.43, 2.18) | 0.942 | 1.03 (0.45, 2.33) | 0.946 |
| Physical activity category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 1.84 (1.15, 2.97) | 0.012 | 2.07 (2, 2.15) | < 0.001 | 2.42 (2.25, 2.6) | < 0.001 | 2.81 (2.61, 3.02) | < 0.001 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 1.53 (0.65, 3.6) | 0.331 | 1.88 (0.21, 17.02) | 0.573 | 1.84 (0.8, 4.27) | 0.153 | 1.72 (0.74, 4) | 0.21 |
| Nicotine exposure category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 1.25 (0.85, 1.84) | 0.263 | 1.22 (1.14, 1.3) | < 0.001 | 1.36 (1.21, 1.53) | < 0.001 | 1.25 (1.11, 1.41) | < 0.001 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 1.03 (0.49, 2.19) | 0.938 | 1.05 (0.15, 7.5) | 0.964 | 1.06 (0.25, 4.49) | 0.941 | 1.05 (0.24, 4.48) | 0.951 |
| Sleep health category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.82 (0.58, 1.17) | 0.28 | 0.84 (0.82, 0.87) | < 0.001 | 0.93 (0.88, 0.98) | 0.01 | 0.85 (0.8, 0.89) | < 0.001 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.6 (0.29, 1.26) | 0.177 | 0.61 (0.09, 4.09) | 0.61 | 0.62 (0.31, 1.24) | 0.178 | 0.61 (0.3, 1.22) | 0.163 |
| BMI category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 1.56 (0.92, 2.64) | 0.099 | 1.51 (1.41, 1.63) | < 0.001 | 1.3 (1.15, 1.48) | < 0.001 | 1.85 (1.63, 2.11) | < 0.001 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.55 (0.15, 1.94) | 0.35 | 0.52 (0.02, 14.36) | 0.701 | 0.53 (0.13, 2.22) | 0.383 | 0.52 (0.12, 2.25) | 0.384 |
| Blood lipids category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.88 (0.56, 1.38) | 0.568 | 0.86 (0.83, 0.88) | < 0.001 | 0.89 (0.84, 0.93) | < 0.001 | 0.88 (0.84, 0.93) | < 0.001 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.78 (0.31, 1.96) | 0.599 | 0.73 (0.04, 12.45) | 0.825 | 0.7 (0.37, 1.32) | 0.268 | 0.81 (0.43, 1.54) | 0.519 |
| Blood Pressure category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.59 (0.42, 0.83) | 0.002 | 0.56 (0.53, 0.59) | < 0.001 | 0.56 (0.51, 0.61) | < 0.001 | 0.61 (0.55, 0.66) | < 0.001 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 0.9 (0.46, 1.76) | 0.761 | 0.85 (0.11, 6.33) | 0.876 | 0.92 (0.34, 2.54) | 0.877 | 0.93 (0.33, 2.59) | 0.892 |
| Stress category | | | | | | | | | |
| Decrease [e] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 1.13 (0.64, 1.98) | 0.678 | 1.17 (1.12, 1.22) | < 0.001 | 1.11 (1.03, 1.2) | 0.009 | 1.06 (1.01, 1.12) | 0.014 |
| Increase [f] | Migraine without aura | Referent | | Referent | | Referent | | Referent | |
| | Migraine with aura | 1.56 (0.59, 4.1) | 0.366 | 1.78 (0.16, 19.31) | 0.635 | 1.88 (0.65, 5.42) | 0.245 | 1.95 (0.65, 5.84) | 0.234 |

Abbreviations: OR, Odds ratio; CI, Confidence interval; CVH, Cardiovascular health

[a] Model 1 unadjusted model

[b] Model 2 adjusted for age

[c] Model 3 adjusted for age, menopause status, marital status, household income, educational level, employment status, fatigue level, and drinking status

[d] Model 4 adjusted for variables in Model 3, hypertension, diabetes, hyperlipidaemia, and cancer

[e] Both the overall CVH profile and individual CVH components were categorized into three levels: Low (0–49 points), Moderate (50–79 points), and High (80–100 points). Decrease indicates that the overall CVH or individual component category at follow-up was lower than at baseline

[f] Increase means that the overall CVH or individual component category at the follow-up was higher than that at baseline

1.01, 1.12), respectively. Conversely, migraine with aura was inversely linked to declines in diet, sleep, blood lipids, and blood pressure categories (all p-value < 0.05).

### Subgroup analysis

The subgroup analysis (Table 5) showed no significant variation in the effect of migraine on CVH progression across most covariates, as most interaction terms were not statistically significant ($p > 0.05$), except for household income, education level, and hypertension status. The impact of migraine on the deterioration of CVH is more severe in participants with higher household income and hypertension, while it is less pronounced in participants with a secondary school or pre-college education level (see details in supplementary results).

### Sensitivity analysis

To evaluate the robustness of the associations observed, we performed three sensitivity analyses. After adjusting the weights of lifestyle and clinical components in

**Table 5** Stratified analyses for the effect of migraine (non-migraineurs as the referent) on the decrease of overall CVH category. Adjusted for age, marital status, educational level, household income, employment status, menopause, hypertension, diabetes, hyperlipidaemia, and cancer, if not already stratified

| Covariate subgroup | *N* | Effect of migraine OR (95% CI) | Interaction coefficient (Migraine*covariate) | Interaction *p*-value |
|---|---|---|---|---|
| Age | | | -0.58 (-1.19, 0.03) | 0.061 |
| <=55 years† | 994 | 1.71 (1.12–2.61) | | |
| >55 years | 1609 | 0.98 (0.69–1.4) | | |
| Marital status | | | -0.26 (-0.87, 0.35) | 0.401 |
| Never married/Divorce/separation† | 647 | 1.65 (1.58–1.72) | | |
| Married or cohabiting | 1956 | 1.24 (1.21–1.27) | | |
| Educational level | | | | |
| Primary school or lower† | 201 | 2.3 (2.07–2.56) | | |
| Secondary school or pre-college | 1537 | 0.78 (0.76–0.8) | -0.88 (-1.04, -0.72) | < 0.001*** |
| College or higher | 865 | 2.32 (2.24–2.4) | 0.15 (-1.33, 1.63) | 0.840 |
| Household income, HKD/month | | | 0.69 (0.05, 1.33) | 0.033* |
| <=35,000† | 1726 | 1.07 (1.04–1.1) | | |
| >35,000 | 877 | 1.86 (1.8–1.94) | | |
| Employed | | | 0.07 (-0.54, 0.68) | 0.819 |
| No† | 1311 | 1.35 (1.3–1.39) | | |
| Yes | 1292 | 1.41 (1.37–1.83) | | |
| Menopause | | | -0.30 (-0.91, 0.31) | 0.335 |
| No† | 602 | 1.27 (1.56–1.68) | | |
| Yes | 2001 | 1.22 (1.18–1.25) | | |
| Drink | | | -0.02 (-0.63, 0.59) | 0.942 |
| No† | 1079 | 1.5 (1.45–1.55) | | |
| Yes | 1524 | 1.32 (1.28–1.36) | | |
| Hypertension | | | 0.62 (0.01, 1.23) | 0.048* |
| No† | 2199 | 1.31 (1.28–1.34) | | |
| Yes | 404 | 2.14 (1.99–2.3) | | |
| Diabetes | | | 0.58 (-0.03, 1.19) | 0.064 |
| No† | 2431 | 1.33 (1.3–1.36) | | |
| Yes | 172 | 3.2 (2.84–3.61) | | |
| Hyperlipidaemia | | | -0.13 (-0.74, 0.48) | 0.670 |
| No† | 1800 | 1.44 (1.4–1.47) | | |
| Yes | 803 | 1.28 (1.22–1.35) | | |
| Cancer | | | 0.40 (-0.24, 1.04) | 0.220 |
| No† | 2452 | 1.34 (1.31–1.37) | | |
| Yes | 151 | 1.02 (0.92–1.13) | | |
| Fatigue | | | -0.16 (-0.77, 0.45) | 0.609 |
| No† | 1269 | 1.57 (1.52–1.62) | | |
| Yes | 1334 | 1.26 (1.22–1.29) | | |

*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$

† Referent group when calculate the interaction coefficient

the CVH score, we consistently found that migraine remained significantly associated with a decrease in CVH (Table S4). Additionally, excluding participants who reported other types of headaches from non-migraine group did not alter the study findings (Table S5). Furthermore, when CVH crude score changes were used as outcomes instead of categorical CVH changes, the results were consistent with the original analysis (Table S6).

## Discussion

### Summary of findings

This cohort study investigated changes in CVH profiles over time among Hong Kong Chinese women with and without migraine. The findings revealed that both groups experienced a decline in CVH profiles; however, migraineurs were more likely to have a reduction in overall CVH categories, with an OR of 1.36. This decline was particularly notable among participants with higher household income and hypertension. Specifically, migraine was positively associated with declines in individual CVH components such as physical activity (OR=1.09), nicotine exposure (OR=4.27), sleep quality (OR=1.80), blood lipid levels (OR=1.03), and stress (OR=1.23). Conversely, migraine was inversely related to decreases in diet, BMI, and blood pressure categories. Among migraineurs, those with aura were specifically linked to decreased physical activity, increased nicotine exposure, higher BMI, and elevated stress levels compared to those without.

There is currently a lack of prospective research exploring the impact of migraine on changes in CVH profiles over time; most research is cross-sectional [19, 22–25]. While a few prospective studies exist, such as a recent study involving 332,895 participants from the UK Biobank without migraine, which found that maintaining an ideal BMI, engaging in PA, and achieving adequate sleep were associated with reductions in the risk of new-onset migraine by 5.65%, 0.81%, 10.16%, and 16.39%, respectively [15], none have specifically investigated the effects of migraine on changes in CVH profiles. Our prospective cohort study is the first to examine CVH progression among migraineurs and non-migraineurs in a population of Hong Kong Chinese women. This research is crucial for understanding the interplay between migraine and CVH, ultimately aiming to reduce the risk of CVD.

Diet patterns and migraine exhibited a bidirectional relationship: dietary factors could trigger migraine, while migraine might influence appetite and food choices [34]. Our study indicated that women with migraine had significantly lower diet scores than non-migraineurs at baseline, indicating poorer overall diet quality. This finding aligned with previous studies [22, 35, 36], such as a cross-sectional study by Martins et al., which found that migraineurs tend to have lower diet quality scores and higher dietary inflammatory index scores [35]. Additionally, our study observed that diet scores of migraineurs were more stable by the follow-up compared to non-migraineurs. One possible explanation was that migraineurs, particularly those with aura, may be more inclined to avoid potential trigger foods, such as alcohol, chocolate, soybeans, and certain meats [37]. Restricting these trigger foods was shown to reduce migraine frequency, duration, and severity [38]. Although this selective diet may be limited in variety, it likely contributed to the observed stability in their diet scores by the follow-up.

Our study showed that migraine was associated with decreased physical activity, consistent with previous research indicating that migraine sufferers tend to be less physically active than non-migraineurs [19, 39, 40]. Many migraine sufferers intentionally avoid physical activity, particularly at higher intensities, due to concerns that it might trigger or worsen attacks [41]. Additionally, symptoms like fatigue and weakness experienced by migraine patients might diminish their motivation and endurance to engage in regular physical activity. Notably, our study found that migraine patients with aura were at an even higher risk of reduced physical activity, possibly due to the heightened sensory disturbances and neurological symptoms they experience, which may increase their sensitivity to physical exertion. However, the relationship between exercise and migraine is complex. A review study highlighted that while physical activity can sometimes act as a trigger, it may also serve as a preventive measure [42]. Strenuous exercise has been reported to provoke migraine attacks in some individuals [43], yet regular moderate aerobic exercise might reduce both the severity and frequency of migraines, potentially through modulation of the endocannabinoid system [44, 45].

Individuals with migraines are more likely to exhibit a higher smoking intensity and a longer duration of smoking history than those without migraines [18]. A review study reported that tobacco use is linked to a higher prevalence of migraine and may exacerbate migraine-related consequences [18]. Consistent with these findings, our study observed that nicotine exposure was higher among migraineurs at baseline, and we further found that nicotine exposure increased more among migraineurs compared to non-migraineurs by the follow-up. One possible explanation is that migraine sufferers, particularly women, often experience higher levels of stress [46], and smoking is commonly used as a strategy to mitigate these emotional stresses [47]. Additionally, the acute pain-relieving effects of nicotine are mediated by the activation of nicotinic acetylcholine receptors, which may help alleviate migraine symptoms [48]. However, the relationship between nicotine and migraine remains complex and not fully understood. Parasympathetic mechanisms

involving acetylcholine and its analogues may also contribute to migraine pathophysiology [49].

Our study observed that migraineurs' sleep health became more unstable at follow-up compared to non-migraineurs. This instability suggests that migraineurs' sleep health tends to fluctuate, with some experiencing improvements while others show deterioration, resulting in a more dynamic and unpredictable sleep pattern over time. Previous studies have found that individuals with migraine are predisposed to poor sleep [20, 24, 25], it is likely that their sleep health will deteriorate further without timely intervention. Conversely, some migraineurs may experience improvements in sleep quality, likely due to the effects of migraine treatments and lifestyle adjustments. For example, medications like amitriptyline, known for their sedative properties, have been shown to improve sleep when used prophylactically [50]. Similarly, recent findings suggest that OnabotulinumtoxinA treatment can enhance sleep quality in chronic migraineurs, offering dual benefits for both migraine management and sleep improvement [51]. The relationship between migraine and sleep is complex, potentially involving common pathophysiological mechanisms in the brainstem and hypothalamus [52, 53]. Future research should explore these connections to develop personalized treatments, as stabilizing sleep may aid in managing migraines and improving CVH.

The presence of significant positive ORs for both increase and decrease in specific CVH components (e.g., sleep heath) does not indicate contradictory results but rather reflects the dynamic health trajectories and unpredictable courses of migraineurs in certain components, with both positive and negative shifts over time depending on treatment adherence and individual behaviour. These findings highlight the need for personalized treatment strategies, as stabilizing these components could improve long-term outcomes for migraine sufferers.

The correlation between BMI and migraine showed mixed results across studies. Several studies have found a positive association between obesity and migraine risk [54, 55], with some indicating a J-shaped relationship [56]. However, the association may be modified by factors such as age, gender, and stress levels [57]. In our study, migraineurs' BMI was less likely to increase by the follow-up after adjusting the effects of other covariates. This suggests that migraine sufferers may place greater emphasis on weight management. Weight loss interventions have shown promise in improving migraine symptoms; for example, behavioural weight loss programs have demonstrated efficacy in reducing headache frequency, pain intensity, and duration [44]. Therefore, migraineurs may benefit from engaging in weight management to alleviate headache symptoms. Additionally, certain migraine medications, such as topiramate, may induce weight loss and improve metabolic parameters [58]. The mechanisms linking obesity and migraine may involve inflammatory processes, adipocytokines, and shared behavioural and psychological factors [59]. Further research is warranted to explore these relationships longitudinally and across diverse populations, which may enhance clinical guidelines for migraine management through tailored weight control strategies.

Blood lipid levels in migraineurs have been a topic of debate. In our study, no significant differences in lipid levels were observed between migraine suffers and non-migraineurs at baseline. However, by follow-up, blood lipid levels among migraineurs showed greater variability. On one hand, some evidence suggests that lipid profiles in migraine patients may deteriorate over time. For example, a meta-analysis by Liampas et al. reported elevated low-density lipoprotein (LDL) cholesterol levels among migraine patients [16]; similarly, another study suggested that migraineurs often have less favourable lipid profiles [60]. Lifestyle factors, such as reduced physical activity, may further influence lipid metabolism, as migraineurs are less likely to engage in moderate-to-vigorous exercise [41]. On the other hand, some research has noted improvements in lipid profiles with certain chronic migraine treatments; for example, migraine treatment with sodium valproate has been associated with reductions in total cholesterol and LDL levels [61]. These findings indicate that therapeutic interventions may influence lipid metabolism in chronic migraine patients. However, the clinical significance of lipid level differences between migraineurs and non-migraineurs remains uncertain, highlighting the need for further research to determine optimal lipid monitoring strategies for migraine patients [62].

The association between blood pressure and migraine has yet to be clearly defined. Some studies suggest that migraine is linked to a higher risk of developing hypertension [17, 63], while others report lower systolic blood pressure in individuals with headaches [64]. In our study, no significant difference in baseline blood pressure was observed between migraineurs with and without aura. Additionally, migraineurs' blood pressure remained more stable over time, showing less variation at follow-up compared to non-migraineurs. This stability may be partly explained by common migraine treatments, such as beta-blockers, which are known to lower blood pressure as a side effect [65]. Additionally, some research has linked increased pulse pressure to a decreased prevalence of headaches [66], possibly due to hypertension-associated hypoalgesia, which involves the baroreflex system's influence on pain perception [67]. The complex relationship between migraine and blood pressure underscores the need for personalized treatment strategies when managing patients with both migraine and hypertension,

highlighting careful consideration in clinical practice [68].

Our study found that migraineurs had higher stress levels at baseline, and by the follow-up, their stress levels were more likely to worsen compared to non-migraineurs. This finding aligns with previous research showing that migraineurs experience higher levels of perceived stress than those without migraine [69]. This relationship is especially pronounced in patients with high-frequency migraines [70]. Stress acts as both a trigger and a consequence of migraine attacks, creating a cyclical relationship that can exacerbate the condition over time [71]. The interplay between stress and migraine is complex, involving factors such as anxiety, depression, and insomnia, which can all significantly contribute to migraine severity and frequency [69].

### Advantages and limitations

This study may be the first cohort study to explore the impact of migraine on the progression of CVH over time among Chinese women. However, there are certain limitations. First, the follow-up duration, averaging 1.27 years, was relatively short. Previous research on populations aged 8 to 55 has shown that CVH decline accelerates at two key inflection points: 16.9 and 37.2 years, with an estimated 18-point drop in CVH score between the ages of 37 and 55 [72]. However, data on CVH decline after the age of 55 remain limited. Considering that the average age in this study was 56.5, if individuals with migraines do experience a more rapid decline in CVH, they may see a drop greater than 18 points between the ages of 55 and 73. Therefore, personalized interventions targeting this older migraine population would be essential. Prolonging the follow-up duration in future studies could yield more comprehensive data on the long-term impacts of migraine on CVH. Second, the reliance on self-reported surveys may introduce potential recall bias. To mitigate this bias, the interviewer administered the questionnaire in person instead of relying solely on self-administered responses. This approach aimed to obtain more accurate and reliable responses by allowing researchers to clarify questions and ensure understanding. Third, Fasting plasma glucose scores were excluded from the analysis because data collection was not performed in a fasting state. Nonetheless, the CVH metrics used in our study remain robust, as our findings were consistent in the reweighting of component analysis. Future studies should include this variable for a more comprehensive evaluation. Fourth, the study focused exclusively on women of Chinese descent in Hong Kong. While this focus reduced confounders related to sex-specific and hormonal differences, it restricts the applicability of the findings to males and individuals from other ethnic backgrounds. Fifth, we recognize that socio-political factors/events may have impacted participants' stress levels. Our study did not directly measure exposure to these events, and we assumed that these effects was balanced between migraine and non-migraine groups. We acknowledge this assumption as a limitation and recommend that future studies account for such influences when examining stress-related health outcomes particularly in the context of public emergencies. Finally, although this is a cohort study, which allows for observing relationships over time, it remains observational and cannot definitively establish causal relationships. Despite these limitations, the study provides valuable insights and sets the stage for future research that can build on these findings to explore the broader impact of migraine on CVH progression.

## Conclusion

This cohort study observed a decline in CVH profiles over time in both migraineurs and non-migraineurs among Hong Kong Chinese women, with migraineurs experiencing a significantly elevated risk of deterioration. Migraine was associated with adverse changes in physical activity, nicotine exposure, sleep health, blood lipids, and stress levels. Notably, women with migraine with aura were more susceptible to reductions in physical activity, increased nicotine use, higher BMI, and elevated stress compared to those without aura. These findings underscore a faster rate of CVH decline in women with migraine, emphasize the importance of timely, tailored interventions to mitigate cardiovascular health deterioration in this vulnerable population.

### Abbreviations

| | |
|---|---|
| CVDs | Cardiovascular diseases |
| CVH | Cardiovascular health |
| MECH-HK | Migraine Exposures and Cardiovascular Health in Hong Kong Chinese Women cohort study |
| BMI | Body mass index |
| LE8 | Life's Essential 8 |
| IPAQ | International Physical Activity Questionnaire |
| PSS | Perceived Stress Scale |
| ICHD-3 | International Classification of Headache Disorders 3 |
| HDL | High-density lipoprotein |
| LDL | Low-density lipoprotein |
| DBP | Diastolic blood pressure |
| SBP | Systolic blood pressure |

## Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s10194-024-01911-2.

Supplementary Material 1

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**Author contributions**
YJX conceptualized and designed this study. YJX was responsible for data collection. JBL and YJX conducted the data analysis, interpreted the results, and prepared the initial draft of the manuscript. JJM, HHW, QLY, TYM, QS, LY, and YJX reviewed the data and provided critical revisions to the manuscript. All authors reviewed and approved the final version for submission.

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**Data availability**
The datasets utilized and/or examined in this study are accessible upon reasonable request from the corresponding author.

## Declarations

**Ethics approval and consent to participate**
Ethical approval for this study was obtained from the Human Subjects Research Ethics Committee at The Hong Kong Polytechnic University (Ref.: HSEARS20171229004). All participants provided written informed consent.

**Competing interests**
The authors declare no competing interests.

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