Long-term prognosis after a first myocardial infarction: eight years follow up of the case-control study PAROKRANK

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Objective. To explore long-term cardiovascular outcomes and mortality in patients after a first myocardial infarction (MI) compared with matched controls in a contemporary setting. Methods. During 2010–2014 the Swedish study PAROKRANK recruited 805 patients <75 years with a first MI and 805 age-, gender-, and area-matched controls. All study participants were followed until 31 December 2018, through linkage with the National Patient Registry and the Cause of Death Registry. The primary endpoint was the first of a composite of all-cause death, non-fatal MI, non-fatal stroke, and heart failure hospitalization. Event rates in cases and controls were calculated using a Cox regression model, subsequently adjusted for baseline smoking, education level, and marital status. Kaplan–Meier curves were computed and compared by log-rank test. Results. A total of 804 patients and 800 controls (mean age 62 years; women 19%) were followed for a mean of 6.2 (0.2–8.5) years. The total number of primary events was 211. Patients had a higher event rate than controls (log-rank test p < .0001). Adjusted hazard ratio (HR) for the primary outcome was 2.04 (95% CI 1.52–2.73). Mortality did not differ between patients (n = 38; 4.7%) and controls (n = 35; 4.4%). A total of 82.5% patients and 91.3% controls were event-free during the follow up. Conclusions. In this long-term follow up of a contemporary, case-control study, the risk for cardiovascular events was higher in patients with a previous first MI compared with their matched controls, while mortality did not differ. The access to high quality of care and cardiac rehabilitation might partly explain the low rates of adverse outcomes.

Introduction

Cardiovascular disease (CVD) is the globally leading cause of death [1] with its most common manifestation, ischemic heart disease (IHD), still constituting the first single cause of mortality in Europe both in men and women [2]. Encouragingly, and thanks to the widespread use of reperfusion therapy and knowledge about evidence-based risk factor management, the incidence of fatal events after hospitalization from IHD is steadily declining in high-income countries, with a 33% decrease in deaths from 1990 to 2019 [3–5]. However, due to a combination of this increased survival and the general population’s aging and growth, this enhances the high burden of CVD, with IHD accounting for 54% of total lost disability-adjusted life years (DALYs) in Europe [6]. People surviving a myocardial infarction (MI) seem to remain at higher risk than the general population, particularly in older individuals with hypertension, diabetes, peripheral artery disease, or a history of stroke [6]. Moreover, a slight increase in IHD death rates during the last 5 years, and implementation of evidence-based treatment seems to be in decline [3,7,8].

Since most studies have focused on short-term outcomes there is shortage of contemporary data on the long-term prognosis after a MI in high-income countries [9–11]. Furthermore, available information relates to cohorts with both first and recurrent MI, making it hard to determine the actual prognosis without the influence of age and concomitant diseases [9].

The main objective in the present study was to explore the long-term mortality and incident CVD events in patients...
after a previous first MI compared with matched controls in a contemporary setting.

Materials and methods

Study population

The Swedish multicenter case-control study Periodontitis and Its Relation to Coronary Artery Disease (PAROKRANK) protocol has previously been described in detail. Briefly, 805 patients <75 years with a first MI according to international criteria [12] were recruited at 17 Swedish hospitals during the years 2010–2014 [13]. Patients were retrieved through centers with a coronary care unit linked to the Swedish National quality registry SWEDHEART [14]. Controls of the same gender and age (±3 months), free from previous MI and heart valve replacement and who were living in the same postal code areas as the patients were randomly selected from the national population registry. Exclusion criteria were previous myocardial infarction, previous heart valve replacement, and any other condition that, according to the judgment of the investigator, could limit the ability to cope with the protocol. First, a list of potential control candidates was generated and a research nurse at the PAROKRANK coordinating center contacted them by telephone, starting from the candidate who was closest in age to the corresponding patient, providing study information and collecting information on the relevant medical history. Subsequently, contact information to the selected control persons was sent to the local study center, where written informed consent to participate was obtained. The number of persons approached to recruit one control was four on average.

Outpatient visits for study enrolment of participants were carried out at the local departments of cardiology. Patients were scheduled approximately 6–10 weeks after the acute event, in accordance with national routine care. To ascertain that the controls would undergo their investigations during the same season, they were contacted within 10 days after their matched patient had their visit. All study participants had fasted and abstained from smoking during at least 12 h prior to their physical examination and venous blood sampling. Information on medical and family history was collected through a set of standardized questionnaires. The self-assessment version of the Montgomery Åsberg Depression Rating Scale (MADRS) was used to evaluate symptoms of depression [15]. It includes nine questions concerning symptoms of depressive illness, i.e. sadness, inner tension, sleep, appetite, concentration, lassitude, inability to feel, pessimistic thoughts and suicidal thoughts [16].

Laboratory analyses

The following analyses were performed in both patients and controls on blood samples at the local laboratory: complete blood count, P-lipids (total and HDL-cholesterol and triglycerides), P-creatinine, P-fibrinogen, P-glucose, and glycated hemoglobin A1c (HbA1c). Participants without diagnosed type 2 diabetes mellitus (T2DM) underwent an oral glucose tolerance test (OGTT by the ingestion of 75 g glucose dissolved in 200 mL water). P-glucose, measured in the fasting state and 2 h after the glucose intake, was analyzed with the HemoCue® 201 System (HemoCue® AB, Angelholm, Sweden). High-sensitivity C-reactive protein was analyzed at a central laboratory (Redhot Diagnostics, Södertälje, Sweden) through ELISA (MP Biomedicals, New York, USA) a method with a functional sensitivity of 0.1 mg/L. Plasma (6 mL) and whole blood (4 mL) were also collected and stored in a central bio bank at Karolinska Institutet at −70°C.

Follow up

All patients were included at hospitals in Sweden that are participating in the SWEDHEART-registry [3,4]. These hospitals are following patients <75 years for at least 12 months at their cardiology department with access to secondary preventive and physical training programs. Thus, all patients had access to Swedish standards of care.

The primary endpoint was the first of a composite of all-cause death, non-fatal MI, non-fatal stroke, and heart failure (HF) hospitalization (Supplemental Table 1). All study participants were followed until 31 December 2018, through linkage with the National Patient Registry and the Cause of Death Registry.

Statistical analysis

Descriptive statistics was used to characterize patients and controls. Statistical comparisons between patients and controls were made by the Student’s t test for matched pairs. The McNemar’s chi-square test was applied to evaluate hypotheses of variables in 2 × 2 contingency tables for matched pairs. Contingency tables larger than 2 × 2 with ranked ordered alternatives were tested using the Wilcoxon signed rank test. Hazard ratios (HR) and 95% confidence intervals (95% CI) subsequently adjusted for smoking, education level and marital status at baseline were calculated for the primary composite event by means of a Cox regression model. Event curves for the time-to-first event in patients and controls were computed by the Kaplan–Meier estimator and the two groups were compared by means of the log-rank test. A two-sided p < .05 was considered as statistically significant. All statistical analyses were performed by means of the SAS system (SAS system for Windows 9.4, SAS Institute Inc., Cary, NC, USA).

Ethics

All patients provided their informed written consent prior to enrolment. The study was approved by the Regional Ethics Committee in Stockholm (Dnr:2008/152-31/2 and 2019-02871) and conducted according to principles outlined in the Helsinki declaration.
Results

One patient and five controls were lost to follow up due to missing personal or mismatched identification numbers, leaving 804 patients and 800 controls for the present investigation. The mean age was 62 ± 8 years and 81% were males. Clinical characteristics of the participants are presented in Table 1. A history of hypertension, stroke, diabetes mellitus, kidney disease, rheumatic disease and depression did not differ between patients and controls. A family history of CVD and smoking at study entry were more frequent in patients than controls. With regard to anthropometrics, waist circumference and body mass index did not differ between patients and controls. In comparison to controls, patients had on average a higher MADRS score, indicating more depressive symptoms. At baseline, i.e. 6–10 weeks after the MI, patients had lower systolic and diastolic blood pressure, P-cholesterol and P-triglycerides, and they were receiving treatment with statins (97%), renin-angiotensin inhibitors (86%) and beta-blockers (92%) more often compared with controls (17%, 27%, and 13% respectively).

Socioeconomic factors are presented in Table 2 showing that a lower income was more common among patients than controls and that divorce rates were higher amongst patients, 15% vs. 10% in controls. There was a difference between the proportion of patients (11%) and controls (6%) born outside of Sweden ($p = .004$).

During a mean follow-up period of 6.2 years (SD 1.1; range 0.2–8.5 years) a total of 211 primary events occurred (patients $n = 141$; controls $n = 70$). The number of the different components of the primary event in the two groups was 61 vs. 24 for MI, 21 vs. three for HF hospitalization and 27 vs. 15 for stroke. Death from any cause did not differ significantly between patients ($n = 38$; 4.7%) and controls ($n = 35$; 4.4%). Cardiovascular death accounted for 18% of the all-cause mortality in patients (seven events) and 11% in

Table 1. Baseline characteristics in patients and controls. Data are presented as mean ± SD or number (%). If not otherwise stated, patient data were retrieved at the follow-up visit 6–8 weeks after the index infarction.

| Variables                                      | Patients ($n = 804$) | Controls ($n = 800$) | $p$ Value |
|------------------------------------------------|----------------------|----------------------|-----------|
| Age (years)                                    | 62 ± 8               | 62 ± 8               |           |
| Female sex                                     | 151 (19)             | 151 (19)             |           |
| Known family history of cardiovascular disease | 302 (38)             | 183 (23)             | <.001     |
| Medical history                                |                      |                      |           |
| Hypertension                                   | 285 (36)             | 268 (34)             | .38       |
| Peripheral artery disease                      | 20 (3)               | 10 (1)               | .099      |
| Stroke                                         | 22 (3)               | 18 (2)               | .64       |
| Diabetes mellitus                              | 79 (10)              | 65 (8)               | .25       |
| Rheumatic disease                              | 164 (21)             | 136 (17)             | .056      |
| Pulmonary disease                              | 106 (14)             | 85 (11)              | .11       |
| Kidney disease                                 | 33 (4)               | 32 (4)               | 1.00      |
| Cancer                                         | 66 (8)               | 58 (7)               | .51       |
| Depression                                     | 76 (9)               | 71 (9)               | .73       |
| Smoking habits                                  |                      |                      |           |
| Current                                        | 70 (9)               | 96 (12)              | <.001     |
| Previous                                       | 440 (55)             | 361 (45)             |           |
| Never                                          | 283 (36)             | 343 (43)             |           |
| Waist circumference (cm)                       | 99 ± 11              | 98 ± 12              | .12       |
| Body mass index (kg/m²)                        | 27 ± 4               | 27 ± 4               | .24       |
| Blood pressure (mm Hg)                         |                      |                      |           |
| Systolic                                       | 129 ± 17             | 137 ± 17             | <.001     |
| Diastolic                                       | 77 ± 10              | 84 ± 10              | <.001     |
| Laboratory                                     |                      |                      |           |
| Cholesterol (mmol/L)                           | 3.9 ± 0.8            | 5.5 ± 1.1            | <.001     |
| Triglycerides (mmol/L)                         | 1.3 ± 0.9            | 1.5 ± 1.3            | .009      |
| Fibrinogen (g/L)                               | 3.4 ± 0.8            | 3.2 ± 0.7            | <.001     |
| High sensitivity CRP (mg/L)                    | 2.3 ± 2.6            | 2.2 ± 2.5            | .48       |
| White blood cell count ($×10^9$/L)            | 6.6 ± 4.8            | 5.7 ± 3.0            | <.001     |
| Platelet count ($×10^9$/L)                     | 240 ± 63             | 234 ± 60             | .11       |
| HbA1c (IFCC mmol/mol; (DCCT %))                | 41 ± 8               | 39 ± 8               | <.001     |
| Glycaemic state (OGTT)                         |                      |                      |           |
| Fasting plasma glucose (mmol/L)                | 6.0 ± 1.4            | 5.6 ± 1.3            | <.001     |
| Post load plasma glucose 120' (mmol/L)         | 7.0 ± 2.4            | 6.2 ± 2.3            | <.001     |
| Diabetes and impaired glucose tolerance (IGT)  |                      |                      |           |
| Newly detected according to OGTT              | 230 (29)             | 141 (18)             | <.001     |
| Previously known + newly detected (%)         | 309 (38)             | 206 (26)             | <.001     |
| MADRS (total score)                            | 6.0 ± 6.2            | 4.4 ± 5.1            | <.001     |
| Pharmacological treatment                      |                      |                      |           |
| Renin-angiotensin inhibitors                   | 687 (86)             | 213 (27)             | <.001     |
| Aspirin                                        | 776 (97)             | 82 (10)              | <.001     |
| Beta-blockers                                  | 735 (92)             | 106 (13)             | <.001     |
| Statins                                        | 775 (97)             | 134 (17)             | <.001     |
| Anti-inflammatory agents                       | 13 (2)               | 32 (4)               | .007      |
| Corticosteroids                                | 24 (3)               | 30 (4)               | .39       |
| Antidepressants                                | 42 (5)               | 51 (6)               | .32       |

CRP: C-reactive protein; DCCT: diabetes control and complications trial; IFCC: international federation of clinical chemistry; MADRS: Montgomery Åsberg depression rating scale; OGTT: oral glucose tolerance test. The bold text indicates statistically significant $p$-values ($<.05$).
controls (four events). Most of the non-CV death were due to cancer. Non-CV deaths not related to cancer were: diabetes, suicides/accidents, cirrhosis, pulmonary, neurologic conditions and rheumatoid arthritis and infections (sepsis). Six patients and seven controls had another hospitalization prior to their death.

Unadjusted HR for the primary outcome was 2.08 (95% CI 1.56–2.77). After adjusting for smoking, education level and marital status at baseline the adjusted HR was 2.04 (95% CI 1.52–2.73).

Computed Kaplan–Meier plots showed that a total of 82.5% of the patients and 91.3% of the controls were event-free during follow up (log-rank test \( p < .0001 \); Figure 1).

**Discussion**

In this long-term follow-up of a case-control study, patients with a prior first MI had a two-fold higher risk for a subsequent event during on average 6 years of follow up, including non-fatal MI, non-fatal stroke, HF hospitalization and all-cause death, compared with controls. Although this difference was statistically significant, the event rate in both groups was lower than expected. The number of deaths were few and did not differ significantly between patients and controls; similarly, few developed HF in need of hospitalization. Thus, the difference in event rates between patients and controls were largely attributable to a higher number of non-fatal cardiovascular events.

Interestingly, cardiovascular death accounted for a remarkably low proportion of all deaths, suggesting that individuals surviving the acute phase of a first MI are not at high risk of dying from a subsequent vascular event, reasonably because they receive appropriate treatment and rehabilitation after the event. Several studies have investigated morbidity and mortality trends in patients after an acute MI. One large Swedish study reported that the risk of subsequent non-fatal MI, non-fatal stroke and cardiovascular death in 97,254 patients discharged after MI was 18% during the first year after the index event and an additional 20% during the following 3 years [9]. These results suggest a somewhat higher risk compared with the present study, especially since HF and total mortality were not included as outcomes and that the follow up was shorter. The inclusion of re-infarctions and the higher age of the population (mean age 72 years) might explain this discrepancy. The nationwide study did not include a control group, excluding the possibility to compare the prognosis in people with and without MI. Our estimates do not include mortality during the first

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**Table 2.** Socioeconomic factors. Data are presented as number (%).

| Variables                        | Patients (n = 804) | Controls (n = 800) | \( p \) Value |
|----------------------------------|-------------------|-------------------|-----------|
| Education                        |                   |                   |           |
| 1–12 years                       | 533 (67)          | 494 (62)          | .052      |
| University                       | 269 (34)          | 307 (38)          |           |
| Occupation                       |                   |                   |           |
| Working                          | 420 (52)          | 395 (49)          | .051      |
| Retired                          | 353 (44)          | 370 (46)          |           |
| Sick leave                       | 10 (1)            | 4 (1)             |           |
| Other                            | 21 (3)            | 36 (4)            |           |
| Annual income (household; SEK/y) |                   |                   |           |
| <180,000                         | 100 (13)          | 90 (11)           | .048      |
| 180,000–300,000                  | 226 (28)          | 192 (24)          |           |
| >300,000                         | 468 (59)          | 516 (65)          |           |
| Marital status                   |                   |                   |           |
| Single                           | 86 (11)           | 83 (10)           | .046      |
| Married                          | 597 (74)          | 642 (80)          |           |
| Divorced/widowed                 | 121 (15)          | 79 (10)           |           |
| Place of birth outside of Sweden | 85 (11)           | 46 (6)            | .004      |

SEK indicates Swedish crowns.

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**Figure 1.** Kaplan–Meier plots for the composite study outcome (the first of all-cause mortality, non-fatal myocardial infarction, non-fatal stroke, and hospitalization for heart failure) during the long-term follow up. Patients in red and controls in blue. Log-rank test, \( p < .0001 \). Follow-up of patients started 6–10 weeks after the myocardial infarction.
6–10 weeks after the index MI. The mortality after an acute MI including all patients, and not as in the present study only those with a first MI, is on average 4% among those below the age of 80 years in Sweden during the period 2010–2018 (https://www.ucr.uu.se/swedeheart/). The fact that few in our cohort developed serious HF also possibly contributed to the overall low mortality, as HF has a detrimental effect on patient outcome after MI. Gerber et al. reported that 5-year survival after a MI between 2001 and 2010 was 82% among HF-free patients and 61% in HF-patients [17].

The overall number of events was low in our study, although somewhat higher among patients. This may be attributed to high quality of care, which has remarkably improved over the last 20 years in Sweden [14]. This encompasses the increase of in-hospital reperfusion therapy, including percutaneous coronary interventions/coronary artery bypass graft, as well as the implementation of evidence-based treatments at discharge from acute cardiac care, including beta blockers, lipid-lowering therapy, double antiplatelet therapy, angiotensin-converting enzyme inhibitors/angiotensin receptor blockers [3,14]. In addition, programs for cardiac rehabilitation in Sweden, i.e. Heart School, almost halved all-cause and cardiovascular mortality after first-time MI [18]. This supports the importance of sustained initiatives to improve and report quality of care, including careful selection of patients who should have access to early revascularization procedures [19,20].

Despite such encouraging improvements over time, our first MI patients remained at higher risk for subsequent events and death compared with controls, consistently with what was observed in numerous other studies summarized by Johannson [6]. Moreover, time-trends of a meticulous application of evidence-based treatments including lifestyle adjustments have been stagnant or even decreasing over the last 5 years, in Europe as well as in Sweden [3,8]. Therefore, there is a considerable potential for improvement in patient management and investment in education of health care personnel besides identifying new treatment concepts.

The cardiovascular profile [21] of patients and controls in the present study only differed with regard to higher proportions of patients with a family history of cardiovascular disease and smoking being more common among patients, while a history of hypertension, dyslipidemia and known T2DM had a similar prevalence in both groups. When further investigating the glycemic state by means of OGTT the true proportion of T2DM and impaired glucose tolerance was 38% in patients vs. 26% in controls (p < .001). This is still a somewhat lower proportion compared with previous investigations, reporting that approximately two thirds of coronary patients with T2DM and impaired glucose tolerance [22,23]. This difference is likely due to different population characteristics: previous reports included older patients and reinfections, while the current study only recruited younger patients with a first MI [16]. In any case, disclosing previously unknown glucose perturbations by screening with an OGTT might have led to better management, considering that dysglycemic patients with MI have significantly poorer prognosis [24]. The significant differences found in pharmacological treatment are reasonably attributable to the prescription of guideline-recommended postinfarction treatment in patients.

Regarding non-traditional risk factors, we observed that patients had a generally worse socioeconomic status, as indicated by lower income, higher divorce rates and more signs of depression at the MADRS score [16]. Thus, such aspects should be considered when handling patients who suffer a MI event, as they might have a negative prognostic impact [16,25]. Moreover, patients had higher values of fibrinogen and white blood cell counts, but C-reactive protein did not differ between the two groups, therefore no firm conclusions can be drawn on the potential impact of inflammatory mechanisms [26].

Strengths and limitations

This study has several strengths. To the best of our knowledge, no study has recently evaluated a truly long-term prognosis after a first MI. The characterization of the study population was carried out in a standardized manner by trained hospital personnel. Moreover, the case-control study offered a possibility of a direct comparison of event rates between well-characterized patients and matched controls with complete follow-up data.

There are also some limitations to be acknowledged. First of all, subjects willing to participate in clinical studies are usually healthier, with higher compliance to medical treatment, and this might be particularly true for the control subjects [27]. Such selection bias may lead to an overestimation of the true quality of care. Moreover, centers that participated in the PAROKRANK study are supposedly more motivated in guideline-directed treatment of patients. Therefore, although in accordance with previous and larger reports, the good quality of care might not be fully representative of contemporary Swedish healthcare and the prognosis may be overstated. Third, early post-discharge deaths (up to 10 weeks after the index MI) were not captured by our study, possibly leading to an underestimation of the overall mortality among the patients.

Finally, even though adjustments for several covariates were performed, there might be residual unknown confounders not considered.

Conclusion

The present study illustrates that patients surviving the initial phase of a first MI have comparable survival rates to controls, while their risk of experiencing a non-fatal CV event remains higher than in MI-free controls, although at low absolute rates. The access to high quality of care, secondary prevention and cardiac rehabilitation might partly explain such low rates of CV outcomes in patients during this long-term follow up. However, there is still a need for further improvement in post-MI management, especially in treatment and prevention of atherosclerosis.
Disclosure statement

G.F. has no conflicts of interest related to this work; has received grant support from the Erling-Persson family foundation and speaker fees from the European Society of Cardiology, outside of the present work. M.A., K.B., U.D.F., B. Kjellström, B. Klinge, Å.N., P.N. and E.S. have no conflicts of interest related to this work; has received research grants from the Swedish Heart- and Lung foundation, Stockholm County, Erling-Persson family foundation and Private Foundations. A.N. has no conflict of interest related to this work; has received research grants from the Swedish Heart- and Lung foundation, Stockholm County Council and honorarium from advisory board meetings outside this work from Astra Zeneca, Novo Nordisk, MSD Sweden, and Boehringer Ingelheim.

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Data availability statement

The data that support the findings of this study are available from the senior author L.R., upon reasonable request.

References

[1] Roth GA, Johnson C, Abajobir A, et al. Global, regional, and national burden of cardiovascular diseases for 10 causes, 1990 to 2015. J Am Coll Cardiol. 2017;70(1):1–25.
[2] Timmis A, Townsend N, Gale CP, et al. European Society of Cardiology: cardiovascular disease statistics 2019. Eur Heart J. 2020;41(1):12–85.
[3] Szummer K, Wallentin L, Lindhagen L, et al. Improved outcomes in patients with ST-elevation myocardial infarction during the last 20 years are related to implementation of evidence-based treatments: experiences from the SWEDHEART registry 1995-2014. Eur Heart J. 2017;38(41):3056–3065.
[4] Szummer K, Wallentin L, Lindhagen L, et al. Relations between implementation of new treatments and improved outcomes in patients with non-ST-elevation myocardial infarction during the last 20 years: experiences from SWEDHEART registry 1995 to 2014. Eur Heart J. 2018;39(42):3766–3776.
[5] Gersh BJ. The changing prognosis of myocardial infarction in the reperfusion era: implications for evaluation and management of ventricular arrhythmias. Revista Española de Cardiología (English Edition). 2003;56(6):535–542.
[6] Johansson S, Rosengren A, Young K, et al. Mortality and morbidity trends after the first year in survivors of acute myocardial infarction: a systematic review. BMC Cardiovasc Disord. 2017;17(1):53.
[7] Roth GA, Mensah GA, Johnson CO, et al. Global burden of cardiovascular diseases and risk factors, 1990-2019: update from the GBD 2019 study. J Am Coll Cardiol. 2020;76(25):2982–3021.
[8] Kotseva K, De Backer G, De Bacquer D, et al. Lifestyle and impact on cardiovascular risk factor control in coronary patients across 27 countries: results from the European Society of Cardiology ESC-EORP EUROASPIRE V registry. Eur J Prev Cardiol. 2019;26(8):824–835.
[9] Jernberg T, Hasvold P, Henriksson M, et al. Cardiovascular risk in post-myocardial infarction patients: nationwide real world data demonstrate the importance of a long-term perspective. Eur Heart J. 2015;36(19):1163–1170.
[10] Jernberg T, Lindholm D, Hasvold LP, et al. Impact of ischaemic heart disease severity and age on risk of cardiovascular outcome in diabetes patients in Sweden: a nationwide observational study. BMJ Open. 2019;9(4):e027199.
[11] Rapsomaniki E, Shah A, Perel P, et al. Prognostic models for stable coronary artery disease based on electronic health record cohort of 102 023 patients. Eur Heart J. 2014;35(13):844–852.
[12] Thygesen K, Alpert JS, Jaffe AS, et al. Third universal definition of myocardial infarction. Circulation. 2012;126(16):2020–2035.
[13] Rydén L, Buhlin K, Ekstrand E, et al. Periodontitis increases the risk of a first myocardial infarction: a report from the PAROKRANK study. Circulation. 2016;133(6):576–583.
[14] SWEDHEART. Annual report. 2019, p. 6.
[15] Montgomery SA, Asberg M. A new depression scale designed to be sensitive to change. Br J Psychiatry. 1979;134:382–389.
[16] Kjellström B, Gustafsson A, Nordendal E, et al. Symptoms of depression and their relation to myocardial infarction and periodontitis. Eur J Cardiovasc Nurs. 2017;16(6):468–474.
[17] Gerber Y, Weston SA, Enriquez-Sarano M, et al. Mortality associated with heart failure after myocardial infarction: a contemporary community perspective. Circ Heart Fail. 2016;9(1):e002460.
[18] Wallert J, Olsson EM, Pingel R, et al. Attending Heart School and long-term outcome after myocardial infarction: a decennial SWEDHEART registry study. Eur J Prev Cardiol. 2020;27(2):145–154.
[19] Alabas OA, Jernberg T, Pujades-Rodriguez M, et al. Statistics on mortality following acute myocardial infarction in 842 897 Europeans. Cardiovase Res. 2020;116(1):149–157.
[20] Chung SC, Gedeborg R, Nicholas O, et al. Acute myocardial infarction: a comparison of short-term survival in national outcome registries in Sweden and the UK. Lancet. 2014;383(9925):1305–1312.
[21] Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364(9438):937–952.
[22] Bartnik M, Rydén L, Ferrari R, et al. The prevalence of abnormal glucose regulation in patients with coronary artery disease across Europe. The Euro Heart Survey on diabetes and the heart. Eur Heart J. 2004;25(21):1880–1890.
[23] Norhammar A, Tenerz A, Nilsson G, et al. Glucose metabolism in patients with acute myocardial infarction and no previous diagnosis of diabetes mellitus: a prospective study. Lancet. 2002;359(9324):2140–2144.
[24] Hermannides RS, Kennedy MW, Kedhi E, et al. Impact of elevated HbA1c on long-term mortality in patients presenting with acute myocardial infarction in daily clinical practice: insights from a 'real world’ prospective registry of the Zwolle Myocardial Infarction Study Group. Eur Heart J Acute Cardiovasc Care. 2020;9(6):616–625.
[25] Harshfield EL, Pennells L, Schwartz JE, et al. Association between depressive symptoms and incident cardiovascular diseases. JAMA. 2020;324(23):2396–2405.
[26] Kaptoge S, Di Angelantonio E, Pennells L, et al. C-reactive protein, fibrinogen, and cardiovascular disease prediction. N Engl J Med. 2012;367(14):1310–1320.
[27] Storms W. Clinical trials: are these your patients? J Allergy Clin Immunol. 2003;112(Suppl 51):S107–111.