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Considerable differences exist between prevalent and incident myocardial infarction cohorts derived from the same population

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Abstract

Objective: Both prevalent and incident cohorts have been used in epidemiological and prognostic studies of ischemic heart disease (IHD). This study considers the differences between the cohort types.

Study Design and Setting: Using linked primary care, secondary care, and death certification data, prevalent and incident cohorts of people with a first acute myocardial infarction (AMI) were formed from the same population. They were analyzed independently in terms of baseline characteristics and survival to revascularization, another AMI, or death.

Results: 55.7\% of the prevalent cohort members were males, with a mean age of 71.0 years (standard deviation [SD]: 12.0). 59.0\% of the incident cohort members were males, with a mean age of 64.7 years (SD: 13.3). Over 5 years, a greater proportion of prevalent cases died from any cause (31.4\% [95\% confidence interval(CI): 28.6–34.3]) and IHD (18.5\% [95\% CI: 16.2–21.0]) than incident cases (18.0\% [95\% CI: 15.0–21.4] and 12.2\% [95\% CI: 9.7–15.2], respectively). Mean time to death was shorter in prevalent cases. There was a small difference in the numbers of subsequent AMIs between cohorts. In the incident cohort, mean time to AMI was shorter. Fewer prevalent cases underwent coronary artery bypass grafting or percutaneous transluminal coronary angioplasty.

Conclusion: Considerable differences existed between the two cohorts in terms of baseline characteristics and prognosis. Incident cohorts derived from whole populations should be sought for estimation of survival.

Keywords: Cohort studies; Prognosis; Survival; Prevalence; Incidence; Myocardial infarction

1. Background

In health care scenarios, prognosis is expressed in terms of probability, best determined by the observation of populations of patients with similar exposures, adjusted for confounders. Observational cohort studies and clinical trials offer opportunities to estimate such probabilities at a population level to assess the effectiveness of therapeutic and preventative interventions; to establish the incidence of events among the population; and to calculate the absolute and relative effects of exposure to different risk factors [1].

Both incident and prevalent cohorts have been used to study the epidemiology and prognosis of ischemic heart disease (IHD). However, a distinction must be drawn between prevalent and incident cohorts. This study aims to highlight the differences between the cohort types in terms of their characteristics and their effects on estimates of prognosis.

Prevalent cohorts are typically established by identifying everyone in the study population with the exposure or morbidity of interest at a particular point or period in time. Thus, they include both new and existing cases. Such cohorts have the advantage of being relatively easy to assemble, for example, through the use of disease registers. The approach has been used in IHD research to look at the epidemiology, service use, and survival in primary care settings [2–4].
What is new?

Although differences in understanding of the etiology and prognosis of disease obtained from prevalent and incident cohorts is known, few studies have demonstrated this empirically.

Considerable differences in the characteristics and survival were seen between prevalent and incident cohorts of patients with a single acute myocardial infarction assembled from the same primary care population.

Incident cohorts derived from whole populations should be sought for estimation of survival.

However, prevalent cohorts assembled in this way may fail to include those with short-lived, terminal disease, a factor that could result in erroneously optimistic estimates of survival. Conversely, the inclusion of those with long-standing morbidity, who die soon after the baseline date, may have the opposite effect because of truncation of the length of survival from onset to the event of interest, death [2].

Incident cohorts differ from prevalent cohorts in that they should only include new cases that are free of the exposure or condition of interest at the start of the study and who first experience the exposure, or develop the condition, during the defined period of time in which the cohort is assembled. Thus, members of an incident cohort should be more similar than those of a prevalent cohort in terms of disease stage or other prognostic factors. Incident cohorts, however, are more difficult to establish than prevalent ones, especially, if a study requires complex procedures to ensure that everyone is exposure or disease free before entry. Assembly can take a long time, especially, if the exposure or event is rare. Incident cohorts are often established for clinical trials, frequently in secondary care settings. Such cohorts have subsequently been used for prognostic research, even though participation in such studies may be biased because of referral and selection processes [5,6]. It has been shown, for example, that guidance based on prognosis among a trial-based incident cohort may not transfer readily to the general population [7].

Although these methodological concerns have been described in epidemiological textbooks and are clearly important, few studies have demonstrated or quantified the effect empirically, because such work requires the comparison of prevalent and incident cohorts from the same population. Some of the few published studies have highlighted differences in the baseline characteristics and survival in different cohorts of people with epilepsy, incontinence, and human immunodeficiency virus [8—10].

We have looked at the effect of the type of cohort on membership profile and on the risk and length of time to outcome events. We have done so by comparing an incident and a prevalent cohort drawn from the same primary care population of patients with a single previous acute myocardial infarction (AMI).

2. Methods

2.1. Sampling frame

The sampling frame for the study was all patients registered with a broadly representative sample of 40 primary care practices spread throughout Scotland. In the United Kingdom, almost the whole of the population is registered with primary care practices, which provide, for most illnesses, the first point of contact with medical health care services and most continuing care for individuals with chronic diseases. The practices have been participating in the Practice Team Information project operated by the Information Services Division (ISD) of National Health Services in Scotland, and contributing data to the Primary Care Clinical Informatics Unit (PCCIU) at the University of Aberdeen [11]. The primary care patient data were linked in May 2007 with deprivation score information (Scottish Index of Multiple Deprivation [12]), secondary care data held on the Scottish Morbidity Record (SMR01) database housed by ISD, and cause of death data collected by the General Register Office for Scotland (GROS), to create a novel PCCIU/SMR/GROS—linked research database. The completeness of capture of primary care contacts and the accuracy of their recording using the Read classification has been found to be greater than 91%. The electronic recording of chronic prescribing by primary care has also been found to be highly accurate and complete [13]. Secondary care data from ISD are coded using the International Classification of Diseases and are reliable from 1981, with completeness and accuracy rates exceeding 90% [14]. The patient population of approximately 238,000 people in the PCCIU/SMR/GROS database is broadly representative of the Scottish population, with respect to age, sex, and social deprivation [15].

2.2. The cohorts

Two cohorts of patients with a single AMI were formed from the same primary care population. For the prevalent single-AMI cohort, every patient was identified who had a record of previous AMI in the linked database on January 1, 2001. The primary care computer records (including the electronic summary record, an accurate lifetime historical record of patients’ major disease events) of each identified patient was checked for the period for which they existed, for Read Codes for AMI, or for history of AMI at any stage before the index AMI record. Secondary care data for each person were also checked for a similar history at any stage, back to 1981, when reliable SMR01 records began. Any patients with a record of a previous AMI at any time before the index record
were removed so that the cohort contained only those believed to have had a single previous AMI at the baseline date. The incident single-AMI cohort was assembled by identifying everyone with a Read Code for AMI between January 1, 2000, and December 31, 2001. For cases in which more than one AMI was recorded in the 2-year period, the first was taken as the index case, and records were checked backward from this date. The primary and secondary care records of these individuals were checked backward in the same way as for the prevalent cohort, to identify and remove anyone with a record of previous AMI. Thus, the incident cohort only contained those believed to have had their first AMI during the 2-year period.

The age and gender of individuals in each cohort were recorded. The records were also examined for a record before cohort entry of ‘baseline’ comorbidities determined a priori as being related to outcome: angina, diabetes, peripheral vascular disease, and stroke. Each condition was recorded as a separate dichotomous variable. Cases with records of current smoking and obesity (defined as body mass index > 30 kg/m²) before the date of cohort entry were recorded as known smokers or known to be obese, using the information closest to cohort entry; those with no record of smoking status or obesity were grouped with those known to be non-smokers or not obese. The primary care records were searched for the issuing of a prescription for a beta-blocker, calcium channel blocker, thiazide, angiotensin-converting enzyme (ACE) inhibitor, statin, and antiplatelet, 90 days before or 60 days after cohort entry. The postcode of each patient was used to assign a deprivation status on a 10-point scale based on the Scottish index of multiple deprivation, which uses 37 indicators of poverty across seven domains (current income, employment, health, education, housing, geographical access, and crime) [12]. This was then converted to quintiles for analysis (1 = most affluent to 5 = most deprived).

Each patient was followed up for all-cause or IHD-related death or exactly 5 years (1,826 days) from entry into the cohort. Subsequent IHD-related events after cohort entry were also recorded: AMI more than 28 days after the index event (to avoid mistaking repeated recording of the incident AMI as a subsequent AMI), coronary artery bypass grafting (CABG) and percutaneous transluminal coronary angioplasty (PTCA).

2.3. Statistical analysis

As some cases were common to both cohorts, they were neither completely dependent nor independent, and statistical tests to compare them directly could not be performed. Descriptive analyses were conducted with the aim of examining the baseline profile of the two cohorts separately, in terms of their demographics, past medical history, and presence of cardiovascular prescribing and risk factors. Chi-squared tests were used to detect significant association of risk factors within cohorts. The proportion of patients experiencing an outcome within the five-year follow-up period was calculated for each cohort along with the median time and interquartile range (IQR) to each event. Cox proportional hazards models were fitted separately for each cohort to determine the effect of different risk factors on the time to each outcome, while controlling for potential confounding variables. Outcomes included all-cause or IHD-related death, or another AMI, CABG, or PTCA. The starting point was taken as the date of first AMI and the end point was exactly 5 years, date of outcome, or death, whichever was earlier. All patients whose end points were not the outcomes of interest were censored. Kaplan–Meier curves of the log of the negative log of the survival function were plotted separately for each baseline characteristic to check for deviation from the proportional hazards assumption.

2.4. Ethical approval

The use of the anonymous PCCIU/SMR/GROS–linked database for this research was approved by the Privacy and Advisory Committee for ISD and by ISD’s Caldicott Guardian.

3. Results

3.1. Descriptive analysis

A total of 999 patients were identified for the prevalent single-AMI cohort, after checking both the primary or secondary care records. There were 551 patients identified for the incident single-AMI cohort. The characteristics of the two cohorts at baseline are shown in Table 1.

Individuals in the prevalent cohort generally had a higher mean age (71 years; standard deviation [SD]: 12.0), a history of angina or hypertension, and were on statins and antiplatelets. Those in the incident cohort were more often males, had a younger mean age (64.7 years; SD: 13.3); had a history of angina, diabetes, stroke, or hypertension; were often on a beta-blocker, ACE inhibitor, statin, or antiplatelet; smoked; or were obese. The two cohorts were similar in their socioeconomic status profiles. In both cohorts, the prescription of ACE inhibitors and thiazides was associated with a history of hypertension ($P < 0.01$) or heart failure ($P < 0.01$); beta-blockers were associated with hypertension ($P < 0.01$). Median time (IQR) since AMI among the prevalent cohort was 8.0 years (3.0, 15.0).

3.2. Progression to events

Data relating to progression to events are presented in Table 2. A high proportion of individuals in the prevalent cohort experienced death from any cause or IHD (31.4% and 18.5%, respectively). A smaller proportion of the incident cohort died (all causes: 18.0%; IHD: 12.2%). Many of these events occurred several years after cohort entry. Median time to death was longer in the incident cohort than...
Table 1
Comparison of demographic, past medical history prescribing, and risk factor profiles of a prevalent and incident cohort of people with first AMI drawn from the same population

<table>
<thead>
<tr>
<th>Baseline variable</th>
<th>Prevalent single-AMI cohort (n=999)</th>
<th>Incident single-AMI cohort (n=551)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>55.7 (556)</td>
<td>59.0 (325)</td>
</tr>
<tr>
<td>Age, mean (SD)</td>
<td>71.0 (12.0)</td>
<td>64.7 (13.3)</td>
</tr>
<tr>
<td>Angina</td>
<td>56.3 (562)</td>
<td>28.7 (158)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>14.3 (143)</td>
<td>11.8 (65)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>1.8 (18)</td>
<td>8.7 (48)</td>
</tr>
<tr>
<td>PVD</td>
<td>11.3 (113)</td>
<td>8.7 (48)</td>
</tr>
<tr>
<td>Stroke</td>
<td>6.7 (67)</td>
<td>10.2 (56)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>44.0 (439)</td>
<td>34.8 (192)</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>35.6 (356)</td>
<td>61.5 (339)</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>29.0 (290)</td>
<td>24.3 (134)</td>
</tr>
<tr>
<td>Thiazides</td>
<td>40.5 (405)</td>
<td>34.8 (192)</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>31.5 (315)</td>
<td>51.2 (282)</td>
</tr>
<tr>
<td>Statins</td>
<td>53.5 (534)</td>
<td>71.0 (391)</td>
</tr>
<tr>
<td>Antiplatelets</td>
<td>68.7 (686)</td>
<td>81.5 (449)</td>
</tr>
<tr>
<td>Smokers</td>
<td>19.6 (196)</td>
<td>26.2 (138)</td>
</tr>
<tr>
<td>Obese</td>
<td>22.1 (221)</td>
<td>28.5 (157)</td>
</tr>
<tr>
<td>Deprivation score</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 (most affluent)</td>
<td>12.1 (121)</td>
<td>13.6 (75)</td>
</tr>
<tr>
<td>2</td>
<td>19.7 (197)</td>
<td>19.4 (107)</td>
</tr>
<tr>
<td>3</td>
<td>24.9 (249)</td>
<td>24.5 (135)</td>
</tr>
<tr>
<td>4</td>
<td>23.8 (238)</td>
<td>22.3 (123)</td>
</tr>
<tr>
<td>5 (least affluent)</td>
<td>19.4 (194)</td>
<td>20.1 (111)</td>
</tr>
</tbody>
</table>

*Abbreviations: AMI, acute myocardial infarction; SD, standard deviation; PVD, peripheral vascular disease; ACE, angiotensin-converting enzyme.*

in the prevalent cohort. There was little difference in the proportion in each cohort that experienced another AMI, although the median time to AMI in the incident cohort tended to be shorter than that in the prevalent cohort. Few people in the prevalent cohort underwent either a CABG or PTCA; in the incident cohort, nearly 10% of cases had each of these procedures. The median time to either procedure in the incident cohort was considerably very short than that in the prevalent cohort.

Table 3 presents adjusted hazard ratios associated with the baseline variables for each outcome, determined in each cohort independently. Age, preexisting diabetes, stroke or angina, and prescription of thiazides or ACE inhibitors were associated with a significantly increased risk of death from any cause in the incident cohort, and beta-blockers and statins were associated with a significantly decreased risk. In the prevalent cohort, age, diabetes, receipt of a prescription for thiazide, and smoking were significantly associated with an increased risk of all-cause mortality, and none of the variables was protective. Differences in the factors associated with death from IHD, another AMI, CABG, and PTCA were also observed in each cohort type.

4. Discussion

This study, in which both incident and prevalent cohorts were assembled from the same population, demonstrates that the method of cohort identification can have a considerable effect on perceptions of prognosis and factors related to prognosis in IHD. The cohorts were strikingly different in terms of both their baseline characteristics and progression to events. Those in the prevalent cohort were older and more likely to have had angina and hypertension at baseline, and to be prescribed thiazide diuretics. In the incident cohort, more participants had heart failure and previous stroke, had a record of being obese, and were prescribed one of a variety of cardiac drugs. Differences in prescribing patterns observed in each cohort may have resulted from differences in the characteristics of those in each cohort or from changes over time in the provision of care, because many of the prevalent cases would have had their first AMI years before identification for the cohort. The median and IQR times since AMI among the prevalent cohort illustrate the degree of heterogeneity in the primary care AMI population in terms of survival after a single AMI.

The very different nature of the two cohort types is, perhaps, highlighted most clearly by the progression in each from cohort entry to the various outcomes considered. More people in the prevalent cohort died from IHD or any cause than those in the incident cohort, and the median time to death was shorter. These observations may be related to the prevalent cohort’s greater mean age and the inclusion of a higher proportion of individuals with longstanding IHD morbidity, who die soon after the baseline date. In a cohort of older patients, comorbidity at baseline may include a wide range of morbidities and those that are IHD related. Such factors may result in an overestimation of mortality risk in prevalent IHD cohorts. Meanwhile, although the proportion of patients in each cohort who experienced a subsequent AMI was similar, the median time from cohort entry to event was noticeably shorter in the incident cohort. This may reflect the likelihood that more people in the incident cohort had IHD that was at an early point in its progression, whereas individuals at highest risk of a second AMI may have already been lost to the prevalent cohort through a healthy survivor effect. More people in the incident cohort underwent either a CABG or PTCA compared with those in the prevalent cohort, and the time to the procedures was very much shorter. Again, there may be a healthy survivor effect in the prevalent cohort, with considerable time having elapsed since AMI in many cases and only those with more acute or serious disease being offered these cardiac interventions after cohort entry. The much higher numbers of referrals for these procedures in incident cases recorded between 2000 and 2001 may also reflect routine practice in the immediate aftermath of an AMI at that time.

In both cohorts, increasing age was associated with decreased likelihood of PTCA, which may reflect age-based inequity in access to this treatment in line with previous research into inequity in IHD treatments [4]. Alternatively, it may reflect clinical decision making about
the therapeutic viability in older, more seriously ill patients. Diabetes was identified in both cohorts as being highly predictive of subsequent mortality, an effect that has been highlighted in other IHD research [16]. Although heart failure was associated with an increased risk of IHD death in the incident cohort, neither it nor hypertension was associated with any other mortality. Apart from age, diabetes, and prescription of thiazides, no variables were highlighted as significantly associated with any outcome in both incident and prevalent cohorts. Prescription of thiazide diuretics was associated in both cohorts with increased risk of all-cause death, an effect that is likely to be related to the underlying indication for prescription rather than the drugs per se. Similarly, prescription of ACE inhibitors was associated with increased risk of all-cause and IHD death in the incident cohort. Caution must be exercised when considering the effects of drugs in observational studies, because other factors, such as unrecorded comorbidity and length of illness, may be exerting an unknown, confounding effect on prognosis [17].

It is striking that nearly twice as many variables were identified as being significantly associated with outcomes in the incident cohort than those in the prevalent cohort. It is tempting to deduce from this that the incident cohort, with its analysis from actual event to outcome, is more sensitive in its ability to evaluate the effects of risk factors and determine prognosis in newly presenting cases. However, other effects relating to the nature of the prevalent cohort may also contribute to this difference. For example, the records of older patients with long-standing disease included in the prevalent cohort may have been less complete so that recording bias may have occurred. In any research based on medical records, problems can result from data that are missing, whether at random or as a result of clinical, process of care, or patient factors. This is an issue that may affect a prevalent cohort containing a proportion of historical cases more than an incident one in which all cases will be current and active patients. Furthermore, this study considered a cohort based on cases identified at a particular time point—point prevalence. It is possible that if cases were identified over a period of time—period prevalence—more cases who died close to cohort entry, with well-recorded morbidity and prescribing, would have been included. This may have the effect of increasing both the observed risk of mortality in the prevalent cohort and the associations between mortality and baseline morbidity and prescribing.

Although prevalent cohorts may reflect accurately the prevalence of morbidity and an evaluation of care received in a population at a given time, prognostic estimates derived from such cohorts may not accurately reflect the prognosis of newly presenting cases. Large incident cohorts assembled from a broad-enough population to be representative of the underlying population present the best opportunity to determine survival from incident event to outcome event and detect associations between baseline exposures and outcomes.

The potential of different cohort types to influence research results has been illustrated empirically by this study. That being said, it is unclear to what degree the differences in prevalent and incident cohorts of people with a single AMI may be generalizable to different disease populations: the effects may be less in populations with disease that typically develops or progresses over a longer time scale than IHD.

### 4.1. Strengths and limitations

The main strength of this study is the novel linkage of primary care, secondary care, and death certification data, which has enabled prevalent and incident cohorts of people with single previous AMI to be assembled from the same population. The study and the methodology have limitations, however. Importantly, the article should not be considered a prognostic study. The principal consideration in the statistical analysis used was the application of identical tests to the two cohorts and comparison of easily understood results rather than more sophisticated survival analysis. Because some cases were included in both the prevalent and incident cohorts, direct comparisons between

---

**Table 2**

<table>
<thead>
<tr>
<th>Outcome event</th>
<th>Cohort</th>
<th>% of cohort (95% CI) (n)</th>
<th>Median time to events in days (IQR)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All-cause death</td>
<td>Prevalent (n=999)</td>
<td>31.4 (28.6–34.3) (314)</td>
<td>711 (328, 1,221)</td>
</tr>
<tr>
<td></td>
<td>Incident (n=551)</td>
<td>18.0 (15.0–21.4) (99)</td>
<td>974 (502, 1,376)</td>
</tr>
<tr>
<td>IHD death</td>
<td>Prevalent (n=999)</td>
<td>18.5 (16.2–21.0) (185)</td>
<td>738 (371, 1,276)</td>
</tr>
<tr>
<td></td>
<td>Incident (n=551)</td>
<td>12.2 (9.7–15.2) (67)</td>
<td>921 (591, 1,210)</td>
</tr>
<tr>
<td>AMI</td>
<td>Prevalent (n=999)</td>
<td>5.5 (4.3–7.1) (55)</td>
<td>755 (304, 1,274)</td>
</tr>
<tr>
<td></td>
<td>Incident (n=551)</td>
<td>6.2 (4.4–8.4) (34)</td>
<td>295 (124, 855)</td>
</tr>
<tr>
<td>CABG</td>
<td>Prevalent (n=999)</td>
<td>1.6 (1.0–2.6) (16)</td>
<td>907 (275, 1,130)</td>
</tr>
<tr>
<td></td>
<td>Incident (n=551)</td>
<td>8.2 (6.2–10.8) (45)</td>
<td>258 (52, 541)</td>
</tr>
<tr>
<td>PTCA</td>
<td>Prevalent (n=999)</td>
<td>2.1 (1.4–3.2) (21)</td>
<td>463 (297, 891)</td>
</tr>
<tr>
<td></td>
<td>Incident (n=551)</td>
<td>9.1 (7.0–11.8) (50)</td>
<td>75 (7, 309)</td>
</tr>
</tbody>
</table>

*Abbreviations: IQR, interquartile range; IHD, ischemic heart disease; AMI, acute myocardial infarction; CABG, coronary artery bypass grafting; PTCA, percutaneous transluminal coronary angioplasty.*
Table 3
Association of independent variables with mortality and IHD outcomes by cohort type—hazard ratios (95% CI)*

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Mortality and IHD outcome</th>
<th>All-cause death (95% CI)</th>
<th>IHD death (95% CI)</th>
<th>AMI (95% CI)</th>
<th>CABG (95% CI)</th>
<th>PTCA (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Incident cohort</td>
<td>Prevalent cohort</td>
<td>Incident cohort</td>
<td>Prevalent cohort</td>
<td>Incident cohort</td>
<td>Prevalent cohort</td>
</tr>
<tr>
<td>Age (each 1.06)</td>
<td>1.06 (1.03–1.09)</td>
<td>1.06 (1.04–1.07)</td>
<td>1.06 (1.03–1.09)</td>
<td>1.06 (1.03–1.08)</td>
<td>1.02 (0.99–1.06)</td>
<td>1.04 (1.00–1.08)</td>
</tr>
<tr>
<td>Male</td>
<td>1.23 (0.77–1.97)</td>
<td>1.20 (0.91–1.58)</td>
<td>1.18 (0.68–2.06)</td>
<td>1.14 (0.80–1.62)</td>
<td>2.70 (1.12–6.48)</td>
<td>1.50 (0.78–2.97)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.90 (1.09–3.33)</td>
<td>1.92 (1.38–2.68)</td>
<td>2.14 (1.12–4.10)</td>
<td>1.77 (1.18–2.67)</td>
<td>2.09 (0.70–6.22)</td>
<td>1.82 (0.88–3.79)</td>
</tr>
<tr>
<td>Heart failure</td>
<td>1.74 (0.97–3.17)</td>
<td>1.47 (0.61–3.51)</td>
<td>2.00 (1.03–3.90)</td>
<td>1.70 (0.64–4.56)</td>
<td>3.44 (0.86–13.78)</td>
<td>1.65 (0.45–6.03)</td>
</tr>
<tr>
<td>Previous stroke</td>
<td>2.16 (1.23–3.75)</td>
<td>1.27 (0.76–1.97)</td>
<td>2.59 (1.38–4.85)</td>
<td>1.34 (0.74–2.41)</td>
<td>0.24 (0.30–1.93)</td>
<td>2.26 (0.99–5.12)</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>1.04 (0.53–2.07)</td>
<td>0.88 (0.55–1.41)</td>
<td>1.43 (0.69–3.00)</td>
<td>1.04 (0.60–1.81)</td>
<td>0.59 (0.13–2.69)</td>
<td>4.91 (2.37–10.19)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.13 (0.68–1.87)</td>
<td>0.85 (0.64–1.12)</td>
<td>1.16 (0.64–2.11)</td>
<td>0.90 (0.63–1.28)</td>
<td>2.17 (0.94–5.04)</td>
<td>1.33 (0.71–2.48)</td>
</tr>
<tr>
<td>Angina</td>
<td>1.64 (1.02–2.64)</td>
<td>0.80 (0.60–1.05)</td>
<td>1.70 (0.97–2.99)</td>
<td>0.95 (0.66–1.37)</td>
<td>0.74 (0.28–2.01)</td>
<td>0.93 (0.47–1.86)</td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>0.57 (0.35–0.93)</td>
<td>0.78 (0.59–1.05)</td>
<td>0.67 (0.38–1.20)</td>
<td>0.90 (0.63–1.30)</td>
<td>1.10 (0.43–2.82)</td>
<td>1.10 (0.58–2.08)</td>
</tr>
<tr>
<td>Calcium channel blockers</td>
<td>0.66 (0.38–1.15)</td>
<td>0.84 (0.62–1.13)</td>
<td>0.66 (0.34–1.27)</td>
<td>1.00 (0.69–1.44)</td>
<td>0.80 (0.26–2.43)</td>
<td>1.20 (0.63–2.27)</td>
</tr>
<tr>
<td>Thiazides</td>
<td>1.96 (1.16–3.29)</td>
<td>1.70 (1.28–2.26)</td>
<td>1.61 (0.97–2.97)</td>
<td>1.62 (1.12–2.33)</td>
<td>0.58 (0.20–1.72)</td>
<td>1.64 (0.84–3.18)</td>
</tr>
<tr>
<td>ACE inhibitors</td>
<td>3.27 (1.31–8.12)</td>
<td>1.09 (0.72–1.65)</td>
<td>3.23 (1.07–9.78)</td>
<td>1.57 (0.94–2.62)</td>
<td>3.16 (0.58–17.14)</td>
<td>2.00 (0.75–5.22)</td>
</tr>
<tr>
<td>Statins</td>
<td>0.37 (0.15–0.93)</td>
<td>0.74 (0.50–1.10)</td>
<td>0.34 (0.11–0.94)</td>
<td>0.99 (0.58–1.70)</td>
<td>0.33 (0.07–1.67)</td>
<td>0.51 (0.19–1.39)</td>
</tr>
<tr>
<td>Antiplatelets</td>
<td>0.79 (0.37–1.70)</td>
<td>1.35 (0.96–1.88)</td>
<td>1.07 (0.41–2.81)</td>
<td>1.43 (0.87–2.35)</td>
<td>0.12 (0.03–0.49)</td>
<td>0.64 (0.28–1.45)</td>
</tr>
<tr>
<td>Current smoker</td>
<td>1.64 (0.89–2.91)</td>
<td>1.44 (1.05–1.99)</td>
<td>1.71 (0.84–3.46)</td>
<td>1.53 (1.01–2.32)</td>
<td>0.84 (0.35–2.06)</td>
<td>0.82 (0.36–1.87)</td>
</tr>
<tr>
<td>Obese</td>
<td>0.92 (0.54–1.58)</td>
<td>0.83 (0.59–1.17)</td>
<td>0.84 (0.44–1.62)</td>
<td>0.75 (0.48–1.18)</td>
<td>0.45 (0.17–1.20)</td>
<td>0.68 (0.31–1.52)</td>
</tr>
</tbody>
</table>

* Adjusted for gender and age; diabetes; heart disease; previous stroke; peripheral vascular disease; hypertension or angina recorded before baseline; prescription of beta-blockers, calcium channel blockers, thiazides, ACE inhibitors, statins, antiplatelets at or near baseline; and record of smoking or obesity.

Bold values indicate statistical significance.
the two cohorts could not be made. We have used patient record data and have no details of the diagnostic criteria used by the different clinicians who recorded the AMIs or of any investigations used to make the diagnosis. Therefore, it is possible that different diagnostic standards were used for different subgroups within the population. Furthermore, in the search for records for each dichotomous variable missing data were recorded as negative results; although completeness of records in primary and secondary care in Scotland have been found to be high in previous research, missing data have resulted in unrecorded confounders. Finally, as with all studies in which multiple comparisons are made, some significant findings might have occurred by chance.

5. Conclusions

This study has been able to compare prevalent and incident cohorts assembled from the same population to consider the effects of the different methods of cohort assembly. It is confirmed that the method of cohort assembly affects the profile of the cohorts identified and produces important differences in estimates of prognosis. Although convenient for the study of diseases that are common in primary care populations, such as IHD, the limitations associated with prevalent cohorts should be recognised. Prevalent cohorts may be most appropriate for assessing the likelihood of disease development in a population receiving care, but they may overestimate the risk of mortality because of the inclusion of those with long-standing illness and unrecorded confounders. On the other hand, incident cohorts may be more suitable for determining the effectiveness of care in newly presenting cases. For prognostic research, representative, incident cohorts should be sought.

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References