Family History as a Risk Factor for Primary Cardiac Arrest

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Background—The hypothesis that a family history of myocardial infarction (MI) or primary cardiac arrest (PCA) is an independent risk factor for primary cardiac arrest was examined in a population-based case-control study. In addition, we investigated whether recognized risk factors account for the familial aggregation of these cardiovascular events.

Methods and Results—PCA cases, 25 to 74 years old, attended by paramedics during the period 1988 to 1994 and population-based control subjects matched for age and sex were identified from the community by random digit dialing. All subjects were free of recognized clinical heart disease and major comorbidity. A detailed history of MI and PCA in first-degree relatives was collected in interviews with the spouses of case and control subjects by trained interviewers using a standardized questionnaire. For each familial relationship, there was a higher rate of MI or primary cardiac arrest (MI/PCA) in relatives of case compared with relatives of control subjects. Overall, the rate of MI/PCA among first-degree relatives of cardiac arrest patients was almost 50% higher than that in first-degree relatives of control subjects (rate ratio [RR] = 1.46; 95% CI = 1.23 to 1.72). In a multivariate logistic model, family history of MI/PCA was associated with PCA (RR = 1.57; 95% CI = 1.27 to 1.95) even after adjustment for other common risk factors.

Conclusions—Family history of MI or PCA is positively associated with the risk of primary cardiac arrest. This association is mostly independent of familial aggregation of other common risk factors. (Circulation. 1998;97:155-160.)

Key Words: risk factors ■ heart arrest ■ myocardial infarction

Out-of-hospital PCA is a major cause of death in the United States. Previous studies have shown that CHD is by far the most common underlying pathology.1 In the general population, the risk factors for sudden death are generally the same as the major risk factors for the development of CHD.2–10

There is ample evidence that CHD tends to cluster in families,11 and the purpose of this report was to evaluate the association between history of MI/PCA in first-degree relatives and the risk of PCA. Because the major documented risk factors for the development of CHD and/or PCA have important genetic determinants, the question arises whether aggregation of PCA is due to the familial aggregation of these known risk factors or to genetic and/or environmental determinants that family members share and that exert their effects through as yet unknown risk factors. In several studies, a family history of MI or CHD was shown to be a strong predictor of CHD, even after adjustment for other risk factors,11 yet this issue has remained controversial.12

In the present investigation, analysis was carried out to assess the significance of family history of MI/PCA as a risk factor for primary cardiac arrest and to test the hypothesis that this association is independent of other potential risk factors.

Methods

The basic design of the present case-control study has been described in detail.13 Briefly, from paramedic incident reports, all cases of out-of-hospital PCA attended by paramedics in Seattle and suburban King County, Washington, during the period October 1988 to July 1994 were identified. PCA cases were eligible if they had a sudden pulseless condition and the absence of evidence of a noncardiac condition as the cause of cardiac arrest.14 In addition to emergency service incident reports, we reviewed death certificates, medical examiner reports, and autopsy reports, when available, to confirm the absence of evidence of a noncardiac condition as the cause of cardiac arrest.15 In addition to emergency service incidence reports, we reviewed death certificates, medical examiner reports, and autopsy reports, when available, to confirm the absence of evidence of a noncardiac condition as the cause of cardiac arrest. The term “primary cardiac arrest” refers to a cardiac arrest that was a result of heart disease and not “secondary” to trauma, drug overdose, respiratory failure, renal failure, end-stage liver disease, cancer, or other noncardiac causes. We specifically are not using the term to refer to patients who have cardiac arrest in the absence of heart disease or cardiac arrest in the presence of heart disease without a precipitating factor. The use of this nomenclature has been discussed elsewhere, and similar diagnostic criteria that define primary cardiac arrest.
arrest in an operational manner were initially proposed by the Joint International Society and Federation of Cardiology–World Health Organization Task Force on Standardization of Clinical Nomenclature. We have used a similar definition in several previous publications.

Because the focus of the analysis was on persons who appeared healthy until their cardiac arrest, PCA cases were excluded if they had a history of clinically recognized heart disease (such as angina pectoris, MI, coronary artery bypass graft surgery, angioplasty, congestive heart failure, arrhythmias, and cardiomyopathy) or congenital or valvular disease or life-threatening comorbidities, such as cancer or end-stage lung, liver, or renal disease. We further restricted the PCA cases to those who had clinically recognized heart disease or a major comorbidity or who were not married were excluded from the study. The spouses of 357 (85%) of the 418 eligible case patients agreed to participate in an in-person interview.

For each PCA case, control subjects matched for age (within 7 years) and sex were selected from the community by the sampling technique of random-digit dialing. Of the known households, 95% were successfully screened to determine whether a person eligible for the study was resident. Potential control subjects who had previously had clinically recognized heart disease or a major comorbidity or who were not married were excluded from the study. The spouses of 576 (85%) of the 618 eligible case patients agreed to participate in an in-person interview, yielding an overall response rate of 67%.

Data on the subjects’ family health history were collected from both case and control spouses by trained interviewers using a standardized questionnaire. Information about first-degree relatives (each biological parent, brother, or sister) was obtained, including current age or age at death, occurrence of MI, and age at occurrence. In addition, participants were asked to classify the cause of death of their deceased relatives as sudden (if he or she had experienced a sudden unexpected collapse when the heart stopped beating), due to heart attack, or due to some other cause. Complete detailed family history information reported by spouses was available for 235 case and 374 control subjects.

The interview also covered other risk factors for PCA, including age, sex, race, education, weight and height, physician-diagnosed diabetes, hypertension, hypercholesterolemia, cigarette smoking, physical activity, caffeine consumption, and dietary fat intake.

To assess the reliability of spouse reports, we interviewed 58 survivors of primary cardiac arrest and their spouses and 562 control subjects and their spouses independently. The 58 members of our case group were successfully resuscitated in the community by paramedics, were discharged from the hospital, and had no evidence for gross neurological impairment at the time of the study. For the family history variables, the agreement percentages between case subjects and their spouses ranged between 90% and 95%; the $\kappa$ estimates ranged between 0.71 and 0.77. The agreement percentages between control subjects and their spouses ranged between 85% and 98%; the $\kappa$ estimates were between 0.62 and 0.69.

In addition, we examined concordance between study subjects and their spouses with regard to smoking status, hypertension, hypercholesterolemia, and diabetes. In both groups, the agreement percentages ranged between 88% and 100%; the $\kappa$ statistics were between 0.67 and 1.00.

To evaluate the validity of family history and selected covariates, the spouse interview data of 24 case and 57 control subjects were compared with data ascertained through the ambulatory care medical records from Group Health Cooperative of Puget Sound. All medical record data were recorded before the index date. For the family history of MI/PCA variable, the agreement percentages between the two sources were 75% and 84%; the $\kappa$ statistics were 0.51 and 0.65 for case and control subjects, respectively. With regard to smoking status, hypertension, and diabetes, in both groups the agreement percentages were high, ranging between 79% and 100%; the $\kappa$ statistics ranged between 0.64 and 1.00. The concordance for hypercholesterolemia was somewhat lower (% agreement, 0.79 to 0.83; $\kappa=0.41$ to 0.49).

For each participant, person-years accumulated by family members and the number of MI and sudden death events within the family were counted. Person-years of relatives at risk were accumulated from birth until age at interview or age at death, or until age at event for relatives who survived their first MI. For each first-degree relative (parents and siblings), specific incidence rates were calculated and the relative risks were estimated by dividing the rate (history of MI/PCA among relatives per 1000 person-years) among the cardiac arrest cases by the rate among the control subjects; confidence limits for these ratios were also calculated. We used logistic regression analysis to assess the relationship of family history with the risk of PCA while adjusting for differences in familial person-years and for potential confounding and mediating factors.

## Results

Selected characteristics of PCA case and control subjects are summarized in Table 1. PCA cases exhibited a statistically significant higher prevalence of cigarette smoking, hypertension, and diabetes, were less educated, weighed more, and had higher mean caffeine intake than control subjects. PCA cases were, on average, 1.4 years older than the control sample and tended to have a higher prevalence of hypercholesterolemia, to expend fewer kilocalories in leisure-time physical activity, and to consume more fat in their diet.

### Table 1. Risk Factors for Primary Cardiac Arrest Among Case Patients and Control Subjects

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>Case Patients (n=235)</th>
<th>Control Subjects (n=374)</th>
<th>P*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>59.2±10.0</td>
<td>57.8±10.4</td>
<td>.09</td>
</tr>
<tr>
<td>Male sex, %</td>
<td>82.5</td>
<td>80.5</td>
<td>.55</td>
</tr>
<tr>
<td>White race, %</td>
<td>97.0</td>
<td>94.1</td>
<td>.10</td>
</tr>
<tr>
<td>Education, ≤high school</td>
<td>34.5</td>
<td>19.5</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Current smoker, %</td>
<td>32.3</td>
<td>8.8</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hypertension, %</td>
<td>26.8</td>
<td>15.2</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Diabetes mellitus, %</td>
<td>12.3</td>
<td>2.9</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hypercholesterolemia, %</td>
<td>26.8</td>
<td>21.4</td>
<td>.13</td>
</tr>
<tr>
<td>Body mass index, kg/m²</td>
<td>26.6±4.73</td>
<td>25.6±3.79</td>
<td>.008</td>
</tr>
<tr>
<td>Physical activity, kcal/wk</td>
<td>1115±1685</td>
<td>1309±1700</td>
<td>.169</td>
</tr>
<tr>
<td>Coffee intake, cups/wk</td>
<td>3.0±3.56</td>
<td>2.3±3.12</td>
<td>.010</td>
</tr>
<tr>
<td>Fat intake scale</td>
<td>22.1±3.55</td>
<td>21.3±3.92</td>
<td>.100</td>
</tr>
</tbody>
</table>

*Plus-minus values are mean±SD.

*Based on the two-sample $t$ test or the $\chi^2$ test.

Selected Abbreviations and Acronyms

- CHD = coronary heart disease
- HDL-C = HDL cholesterol
- $\kappa$ = kappa statistics
- MI = myocardial infarction
- MI/PCA = myocardial infarction or primary cardiac arrest
- OR = odds ratio
- PCA = primary cardiac arrest
- RR = rate ratio
- TC = total plasma cholesterol
Further analysis of family history, therefore, was based on the history of MI/PCA among first-degree relatives of cardiac arrest case and control subjects. For each familial relationship, there was a substantial excess rate of MI/PCA in family members of PCA cases compared with family members of control subjects (Table 2). The RR was 1.47 (95% CI 1.14 to 1.89) in fathers, 1.50 (95% CI 1.08 to 2.07) in mothers, and 1.47 (95% CI 1.21 to 1.80) in the pooled parents groups. The rate of MI/PCA among siblings of PCA cases was 1.64 per 1000 person-years, compared with a rate of 0.98 per 1000 person years in siblings of control subjects, resulting in an RR = 1.67 (95% CI = 1.22 to 2.29). Overall, the rate of MI/PCA among first-degree relatives of cardiac arrest cases was almost 50% higher than that in first-degree relatives of controls (RR = 1.46, 95% CI = 1.23 to 1.72).

Table 3 shows the results of multivariate logistic modeling of the risk of PCA associated with other risk factors. In model A (which excludes family history variables), diabetes, hypertension, low level of education, and cigarette smoking were significant positive predictors of primary cardiac arrest. Moderate or vigorous physical activity (defined as expenditure of kilocalories in leisure-time activity above the 20th percentile of the control group’s distribution) was negatively associated with PCA risk. Model B shows the OR with its 95% CI from a multivariate model on the inclusion of family history. On the basis of this sample, the estimated odds of cardiac arrest occurring in a subject increases by 1.57 times with each additional first-degree relative affected with MI/PCA, after adjustment for other risk factors and person-years at risk among first-degree relatives. The 95% CI for the family history OR was 1.27 to 1.95. This regression coefficient for family history is nearly identical to that obtained from the univariate logistic model (OR = 1.58; 95% CI = 1.29 to 1.95). The introduction of the family history variables into the logistic model also did not considerably change the coefficients for diabetes, hypertension, cigarette smoking, and physical activity, whereas a modest change was observed in the coefficient for low level of education.

Table 2. Familial Person-Years at Risk, No. of Events, and Rates of Events From MI or PCA in First-Degree Relatives

<table>
<thead>
<tr>
<th>Relationship</th>
<th>Case Patients</th>
<th>Control Subjects</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Person-Years</td>
<td>Number of Events</td>
<td>Rate†</td>
<td>Rate</td>
</tr>
<tr>
<td>Father</td>
<td>19 513</td>
<td>114</td>
<td>5.84</td>
<td>33 443</td>
</tr>
<tr>
<td>Mother</td>
<td>22 403</td>
<td>69</td>
<td>3.08</td>
<td>37 440</td>
</tr>
<tr>
<td>Parents</td>
<td>41 916</td>
<td>183</td>
<td>4.37</td>
<td>70 883</td>
</tr>
<tr>
<td>Brothers</td>
<td>26 307</td>
<td>66</td>
<td>2.51</td>
<td>36 862</td>
</tr>
<tr>
<td>Sisters</td>
<td>25 642</td>
<td>19</td>
<td>0.74</td>
<td>35 485</td>
</tr>
<tr>
<td>Siblings</td>
<td>51 949</td>
<td>85</td>
<td>1.64</td>
<td>72 347</td>
</tr>
<tr>
<td>First-degree relative</td>
<td>93 865</td>
<td>268</td>
<td>2.85</td>
<td>143 230</td>
</tr>
</tbody>
</table>

*Years were accumulated from birth until age at interview or age at death, or until age at event for those who survived their first MI.
†Per 1000 person-years.

Table 3. Risk of Primary Cardiac Arrest Associated With Selected Risk Factors With and Without Adjustment for Family History in First-Degree Relatives

<table>
<thead>
<tr>
<th></th>
<th>Model A</th>
<th></th>
<th>Model B</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR</td>
<td>95% CI</td>
<td>OR</td>
<td>95% CI</td>
</tr>
<tr>
<td>Age*</td>
<td>1.13</td>
<td>0.94—1.36</td>
<td>1.11</td>
<td>0.91—1.34</td>
</tr>
<tr>
<td>Male sex</td>
<td>1.37</td>
<td>0.84—2.22</td>
<td>1.33</td>
<td>0.81—2.17</td>
</tr>
<tr>
<td>Hypertension</td>
<td>1.66</td>
<td>1.06—2.60</td>
<td>1.62</td>
<td>1.02—2.58</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>1.22</td>
<td>0.80—1.86</td>
<td>1.21</td>
<td>0.78—1.86</td>
</tr>
<tr>
<td>Low education (&lt;high school)</td>
<td>1.62</td>
<td>1.08—2.45</td>
<td>1.49</td>
<td>0.97—2.30</td>
</tr>
<tr>
<td>Current smoking</td>
<td>4.81</td>
<td>2.94—7.85</td>
<td>4.90</td>
<td>3.00—8.00</td>
</tr>
<tr>
<td>High coffee intake (≥5 cups/d)</td>
<td>1.29</td>
<td>0.79—2.10</td>
<td>1.33</td>
<td>0.80—2.19</td>
</tr>
<tr>
<td>Physical activity (&gt;20th percentile)</td>
<td>0.55</td>
<td>0.37—0.82</td>
<td>0.54</td>
<td>0.37—0.81</td>
</tr>
<tr>
<td>High fat intake (&gt;80th percentile)</td>
<td>0.97</td>
<td>0.63—1.48</td>
<td>1.03</td>
<td>0.67—1.58</td>
</tr>
<tr>
<td>High blood mass (BMI &gt;29.0)</td>
<td>1.12</td>
<td>0.73—1.71</td>
<td>1.10</td>
<td>0.71—1.69</td>
</tr>
<tr>
<td>Family history in first-degree relatives†</td>
<td>...</td>
<td>...</td>
<td>1.57</td>
<td>1.27—1.95</td>
</tr>
<tr>
<td>Person years of first-degree relatives‡</td>
<td>...</td>
<td>...</td>
<td>1.01</td>
<td>0.87—1.17</td>
</tr>
</tbody>
</table>

*OR associated with a difference of 10 years.
†Total number of first-degree relatives with positive history of MI/PCA.
‡OR associated with a difference of 100 person-years.
The results of our case–control study suggest a differential pattern of familial clustering of PCA risk with respect to the sex and age of the case and control subjects. The family history ORs for women and men were OR = 2.97 and 1.49, respectively, and OR = 1.91 and 1.47 for men <55 and women <60 years old and older subjects, respectively. These differences, however, were not statistically significant (probability values from the multivariate logistic regression models were .29 for the family history × sex interaction term coefficient and .31 for the family history × age interaction term).

Discussion

Various studies have indicated that the risk factor profile of persons with an increased risk for PCA consists of essentially the same factors as those associated with an increased risk for coronary heart disease. The findings of the present study, that diabetes, hypertension, smoking and habitual physical activity are significant risk factors for PCA, are consistent with the findings of numerous other epidemiological investigations.

This shared constellation of risk factors for CHD and sudden death suggest that a family history of MI or PCA may be associated with PCA risk. In our univariate analysis, MI and sudden death in first-degree relatives were found to be equally strong and significant predictors of PCA. In multivariate analysis, family history maintained its predictive strength in the presence of the other risk factors. Our findings are in agreement with many retrospective studies, prospective studies, and angiography studies, which have all clearly demonstrated a familial aggregation for CHD.

A major objective in epidemiological studies is to investigate the effect of single or multiple risk factors on the risk of developing a disease or disorder. The control of confounding to obtain measures of effect that are free of this bias is one of the main challenges in nonexperimental research. In our study, case and control subjects were matched for age within 7 years (difference in mean age, 1.4 years; P = .09), and incomplete matching was introduced as covariate into the logistic regression to adjust for the residual difference in this matching factor. Studies that have examined the option of partial matching have indicated that complete matching is not always necessary to produce most of the efficiency benefit of matching.

The introduction of the family history variable as the last term in the stepwise logistic regression model (model B, Table 3) did not alter the coefficients for the risk factors in the antecedent model. Although this indicates that the PCA risk associated with family history was independent of other risk factors, it does not imply lack of a familial influence in the values for diabetes, blood pressure, smoking, or physical activity in our data. What this does indicate is that familial aggregation of these risk factors accounts for only a small part of the clustering of PCA in families. The clustering would therefore appear to reflect characteristics other than the classic risk factors that were measured in our study. These findings, that the aggregation of PCA is not explained by familial patterns in these risk factors, are consistent with other retrospective studies focusing on CHD, and they were also confirmed through several prospective studies. Yet, at least one retrospective study concluded that the risk of CHD associated with a positive family history appears to be fully mediated by familial aggregation of common risk factors.

Several alternative interpretations of the independent effect of a positive family history of MI or sudden death in the prediction of PCA risk may be considered. First, the association may be operating via unobserved risk factors. We were able to determine TC and HDL-C on nonfasting specimens obtained from a subsample of case (n = 74) and control (n = 179) subjects. Blood specimens from PCA cases were taken in the field immediately after essential emergency medical care had been provided and either the patient was clinically stable or resuscitative efforts had proved ineffective. Specimens from control subjects were obtained at the time of the in-person spousal interview. Although no significant difference between the two groups for plasma TC were observed, case patients had significantly lower mean levels of HDL-C (39.2 mg/dL) than the group of control subjects (47.5 mg/dL). The adjustment for TC/HDL-C ratio had a trivial effect on the strength of the association between family history and the risk of primary cardiac arrest. Nevertheless, it is possible that residual uncontrolled confounding accounts for our findings.

Alternatively, those persons with a positive family history may be more susceptible to the deleterious effects of the traditional risk factors. In other words, individuals from a family prone to MI/PCA may experience a greater risk of PCA by smoking or by developing hyperlipidemia than someone with a similar exposure without such a family history.

It has been noted in several cardiovascular studies that the independent effects of family history may be most important in individuals who are otherwise at low risk for the disease. Yet in our study, no significant differences in ORs have been shown when the study participants were stratified according to a risk score based on diabetes, hypertension, hypercholesterolemia, low education level, smoking, and physical activity (data not shown). Nonetheless, a family history of MI/PCA could modify the risk associated with other risk factors not measured in the present study. In attempts to explore the reasons why men develop CHD despite being at low risk on the basis of established risk factors, it was suggested that family history may be of particular importance because it modifies the degree of risk associated with other factors such as HDL-C. Analyses conducted on a random sample of Israeli men examined in the Jerusalem Lipid Research Clinic Prevalence Study have also suggested potential interaction between family history and HDL-C on CHD risk. One possible mechanism for such a relationship is the existence of interactions between genotypes at different loci and levels of apolipoprotein A-I and HDL-C, which may modify the risk for CHD or PCA. For example, a potential interaction between apolipoprotein A-I and cholesterol ester transfer protein genes has been described. HDL-C, however, was not measured in all study subjects, and in a subsample of 74 case and 179 control subjects, this potential modification could not be demonstrated.

A number of limitations are inherent in the present study. Despite attempts to obtain a complete assessment of parental and sibling history of MI and PCA, a substantial number of the spouses of case and control subjects were unable to provide information on the disease status of first-degree relatives and about the ages of their spouses’ relatives at the time of an
MI/PCA event. However, the characteristics of study participants with and without family history information were similar among both the case and the control subjects (data not shown). To study the impact of missing data on the estimates of effect, models A and B (Table 3) were fitted by use of imputed values estimated by the use of the method of multiple imputation.41 No meaningful changes in OR estimates were observed, whereas the CIs tended to be narrower. In model B, the OR for family history was 1.48, with 95% CI=1.23 to 1.79. Another potential drawback of these data is the lack of a full validation of parental or sibling disease history by medical record review. On the basis of a subsample of case patients and on the total group of control subjects, our data support the reliability of family history of MI/PCA when provided through a spouse interview. A small validation study indicated that spouses accurately provide information about common risk factors, such as smoking, hypertension, and diabetes. Information concerning a more complex item, such as the family history variable, may have a somewhat lower validity. Other investigators have detected a relatively strong concordance between reported family MI and medical record evidence (a sensitivity of 70% to 80% and a specificity of 95%), suggesting that despite some degree of imprecision, the reported history gives a reasonably accurate estimate of family history for the diseases we assessed in this article.42–45 If, however, sudden deaths that were related to noncardiac causes were identified by the spouses as sudden cardiac deaths, such a misclassification would tend to reduce the OR estimates toward the null value, assuming that there is no differential misclassification among case and control subjects. In addition, in such retrospective studies, patients with a recent episode of PCA, or their relatives, may selectively report more familial disease (both true and supposed PCA) compared with control subjects. Although the agreement percentages between the spouse interview data and the ambulatory care medical records were similar for case and control subjects and similar associations between family history and CHD have been seen in case-control studies and prospective studies in which information on family history is obtained before the onset of the disease and is therefore not susceptible to recall bias, we cannot rule out the possibility that such bias may have exaggerated our findings. In a recent prospective study of 7,735 middle-aged British men who were followed up for 8 years, the age-adjusted relative risk for PCA associated with a positive parental history of death from heart disease was similar in magnitude (RR = 1.4; 95% CI = 0.9 to 2.0) to that observed in our study.10 The data accumulated from the present study support an overall significant independent association between family history of MI or PCA and the risk for PCA. At present, no well-defined mechanism has been proposed to explain the process by which a positive family history is associated with PCA risk. Identification of these genetic and/or environmental factors will provide a major tool for the understanding and prevention of PCA, especially among susceptible subpopulations with a positive family history of the disease.

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References


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