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CLINICAL RESEARCH

Prediction of residual angina after percutaneous coronary intervention

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Aims Angina relief is a major goal of percutaneous coronary intervention (PCI); however, about one in five patients continue to have angina after PCI. Understanding patient factors associated with residual angina would enable providers to more accurately calibrate patients' expectations of angina relief after PCI, may support different follow-up strategies or approaches to coronary revascularization, and could potentially serve as a marker of PCI quality.

Methods and results Among 2573 patients who had PCI at 10 US hospitals for stable angina, unstable angina, or non-ST-elevation myocardial infarction (NSTEMI), 24% reported angina 6 months after PCI, as assessed with the Seattle Angina Questionnaire angina frequency score (categorized as none vs. any angina; score = 100 vs. <100). Post-PCI angina was more common in those patients treated for unstable angina (30 vs. 20% stable angina and 21% NSTEMI, $P < 0.001$). Using a hierarchical logistic regression model, eight variables were independently associated with angina after PCI, including younger age, poor economic status, depression, and greater number of antianginal medications at the time of PCI (*c*-index = 0.75). The amount of angina at the time of PCI was more predictive of post-PCI angina in patients with stable or unstable angina when compared with NSTEMI ($p_{\text{interaction}} = 0.01$). The model demonstrated excellent calibration, both in the original sample (slope 1.04, intercept −0.01, $r = 0.98$) and in bootstrap validation.

Conclusion Based on a large, multicentre cohort of PCI patients, we created a model of residual angina 6 months after PCI that can provide patients realistic expectations of angina relief, guide follow-up strategies, support the use of residual angina as a means of comparing PCI quality, and enable comparative effectiveness research.

Keywords Angina • Percutaneous coronary intervention • Quality of life

Introduction

Relief of ischaemic symptoms is the primary goal of elective percutaneous coronary intervention (PCI) and is also an important outcome of PCI in the setting of myocardial infarction (MI). Chronic angina affects roughly half of the 15.4 million US adults with ischaemic heart disease,[1] substantially worsens patients' quality of life,[2,3] and increases the costs of health care.[4] Although coronary revascularization provides substantial benefits to many patients, about one in five patients have residual angina after revascularization.[5] Understanding the factors that are associated with residual angina could enable providers to more accurately inform patients of the likelihood of achieving complete angina relief after PCI. Furthermore, if patients at high risk for residual angina after PCI could be identified prospectively, this could potentially guide strategies for follow-up or aggressive anti-ischaemic medications in order to maximize their quality of life. This information may even help guide the initial revascularization procedure (e.g. choosing coronary artery bypass graft surgery vs. PCI or performing more complete revascularization with PCI in the setting of multivessel coronary disease).

In addition to its ability to provide realistic, individualized estimates of risk for residual angina at the time of PCI, this model could be used for risk standardization for both quality assessment (i.e. to compare the quality of PCI across centres) and comparative effectiveness research. For example, such a model could be used to examine the impact of alternative treatment strategies (e.g. stent type, smoking cessation, or cardiac rehabilitation) on post-PCI angina. If differences between treatments emerge, this information

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could also be fed back to patients at the time of PCI to potentially influence treatment choices (e.g. risk of angina if patients do or do not stop smoking[6]). Given these broad goals of informing patients, altering treatment choices, and enabling quality comparisons, we aimed to build and validate a prediction model for angina after PCI in a real-world PCI population.

Methods

Study population

Our analytic population was derived from the Outcomes of PCI Study (OPS)/Personalized Risk Information Services Manager™ (PRISM) study, a prospective study designed to test the benefits of providing individualized, evidence-based estimates of the procedural risks prior to PCI. The details of the OPS/PRISM study have been described previously.[7–9] Briefly, between 2009 and 2011, consecutive patients undergoing PCI at 10 US academic medical centres and large community hospitals were invited to participate at the time of PCI. To determine predictors of angina among patients for whom relief of angina would be an important treatment goal, we restricted the cohort to patients who underwent PCI for stable angina, unstable angina, and non-ST-elevation MI (NSTEMI), as the primary goal of PCI for an ST-elevation MI is improved survival. Baseline angina was assessed through a structured interview after the patient underwent PCI and was clinically stable. Clinical data were collected through chart abstraction by trained study coordinators. Detailed follow-up was attempted on all surviving patients at 1, 6, and 12 months after PCI with a standardized telephone interview performed by a central follow-up centre. The study complies with the Declaration of Helsinki, as each hospital obtained Institutional Research Board approval, and all patients provided written informed consent for baseline and follow-up assessments.

Angina outcome

Angina was assessed with the Seattle Angina Questionnaire (SAQ); a 19-item self-administered questionnaire that measures five dimensions of health in patients with coronary artery disease.[10,11] It has previously undergone extensive reliability and validity testing.[12,13] The SAQ has a 4-week recall period, and domain scores range from 0 to 100, with higher scores indicating less disease burden. The primary outcome of our study was residual angina at 6 months after PCI, as assessed by the SAQ angina frequency domain, which has been shown to correlate well with patient-reported daily diaries of angina.[14] Congruent with prior work, scores for this domain were categorized as none (score = 100) vs. any (score <100), as this was both clinically interpretable and aligned with the goal of PCI to eliminate angina. The 6-month follow-up assessment was used because it is long enough to enable staged procedures to be completed and early enough so that restenosis or new lesion formation is less likely to have occurred.

Candidate predictors

Candidate variables for the prediction model were selected *a priori* based on clinical judgement and literature review and included 35 sociodemographic, clinical, and health status factors available at the time of admission (Supplementary material online, *Table S1*). For the health status potential predictors, all five SAQ domains and three questions from the Euroqol-5D (EQ-5D; usual activities, pain/discomfort, and anxiety/depression)[15] were included as potential predictors of angina after PCI. For this analysis, we dichotomized the patient responses on the three EQ-5D questions as none vs. moderate/extreme difficulty for each domain.

Because this model was designed to provide estimates of post-PCI angina prior to the procedure and to enable comparative effectiveness research, the model was built exclusively with patient-specific characteristics available prior to PCI. However, we did perform a sensitivity analysis adding number of diseased vessels identified during the coronary angiogram to the model and assessed changes in model performance, as this could be known prior to treatment. Baseline data had a high rate of completion, with 20% of the patients missing only one data element, 3.5% of the patients missing two or more items, and an average of 0.27 missing data items per patient. Missing data were imputed with five imputation data sets (IVEware; Institute for Social Research, University of Michigan, Ann Arbor, MI, USA).

Statistical analysis

The demographic, socioeconomic, clinical, and health status factors at baseline were compared between those patients who did and did not report angina at 6 months after their PCI using the χ^2 test for categorical variables and *t*-tests for continuous variables. We then developed a multivariable logistic regression model to predict angina at 6 months after PCI. To maximize clinical utility of the model, Harrell's backward selection strategy was used to select a parsimonious set of variables for the final model, which supports inclusion of only those variables that provide incremental prognostic value, minimizes over-fitting, and maximizes the potential clinical usefulness of the model.[16] For each of the five imputation data sets, the contribution of each covariate in the multivariable model was ranked by *F*-value. Variables with the smallest contribution to the model were sequentially eliminated until further variable elimination led to a >10% loss in model prediction, when compared with the initial model. This insured that the remaining covariates explained over 90% of the variance of the full model. Variables that were included in at least three of the five reduced variable lists for the imputation data sets were included in the final model. Spline terms were considered for all continuous variables, and, if significant, these variables were plotted against the outcome to examine possible cut-points for categorization. In addition, interactions between the indication for PCI (stable angina, unstable angina, and NSTEMI) and model covariates were examined and included if significant at $P < 0.1$. Hospital was entered in the model as a random effect to adjust for patient clustering by site.

Several approaches were used to describe the final model's performance. Discrimination was assessed with the *c*-index. Model calibration was assessed by plotting deciles of predicted risk against the observed event rate and comparing the regression line with the line of unity (intercept = 0 and slope = 1). Finally, we constructed 1000 bootstrap sample sets (drawn with replacement) of the same size of the original data set to assess internal validation. This has been shown to be superior to split-sample derivation/validation, which tends to provide overly pessimistic estimates of performance, with large variability.[17] We calculated a bootstrap-validated calibration slope from the average of the bootstrapped data sets and also calculated an optimism-adjusted *c*-index, which assesses potential over-fitting.

To demonstrate a potential application of the model, we examined the rates of observed angina by deciles of predicted risk stratified by whether or not the patient had complete revascularization, with the assumption that any potential benefit of complete revascularization on 6-month angina would be most apparent in those at highest risk for residual angina. In addition, we compared the patient-risk-adjusted rate of 6-month angina of patients with and without complete revascularization to determine the effect of this approach on subsequent angina. All analyses were conducted using SAS v9.4 (SAS Institute, Inc., Cary, NC, USA), and statistical significance was determined by a two-sided *P*-value of <0.05 (except for interaction terms).

Results

Patient population

Among 3229 patients who underwent PCI and were enrolled in the PRISM study, 340 patients were excluded as they underwent PCI for an indication other than stable angina, unstable angina, or NSTEMI (e.g. ST-elevation MI, heart failure workup, etc.). There were 28 patients who died prior to 6 months and thus had no opportunity for follow-up. Of the remaining 2861 patients, 2573 patients (89.9%) had both baseline and 6-month assessments of angina and formed our analytic cohort. Patients with missing data were more likely to be younger, non-white, of lower socioeconomic status, current smokers, and were more likely to be undergoing PCI for urgent or emergent indications (Supplementary material online, *Table S2*). Baseline SAQ angina frequency scores were also slightly, but not statistically significantly, lower in patients with missing data (missing vs. not: 68 vs. 71, $P = 0.071$). Patients in the analytic cohort were generally similar to those who were eligible but declined enrolment, although there were modest differences between the two populations (Supplementary material online, *Table S3*). Patients who did not enrol (vs. those in the analytic cohort) were more likely to be older, non-white race, to have more cardiac and non-cardiac comorbidities, and to be undergoing PCI for an NSTEMI. The number of antianginal medications is similar between the groups.

Mean age of the population was 64.7 years, 70.7% were male, 91.9% were white, and 15.8% were current smokers. Stable angina was the indication for PCI in 39.9% of the patients, whereas 38.5% presented with unstable angina and 21.6% with an NSTEMI. Approximately a quarter of patients were on no antianginal medications at the time of PCI, half were on 1, and the remaining quarter of patients were on 2 or more. As expected, the mean number of antianginal medications varied by PCI indication, with NSTEMI patients being on the fewest number of antianginal medications at admission at 0.8 ± 0.8, when compared with patients presenting with unstable angina and stable angina (both with 1.1 ± 0.8 antianginal medications, $P < 0.001$ when compared with NSTEMI patients).

Angina after PCI

Six months after PCI, 24% of the patients reported some angina in the preceding 4 weeks. A comparison of the demographic and clinical factors of patients who did and did not report angina is shown in *Table 1*. Patients with angina were more likely to be younger, female, of lower socioeconomic status, and had more cardiac and non-cardiac comorbidities. Angina after PCI was more common in those with unstable angina (30%) when compared with those who presented with stable angina and NSTEMI (20 and 21%, respectively; $P < 0.001$). Patients with angina were on more antianginal medications at the time of PCI and also reported worse disease-specific and generic health status prior to their PCI.

Angina prediction model

After variable selection, there were eight variables included in the final multivariable prediction model (*Figure 1*). The most important predictor (in terms of *F*-value) was the severity of angina, as assessed by the SAQ angina frequency score, prior to PCI. Patients with more frequent angina at the time of PCI were less likely to be angina-free at 6 months after PCI. However, there was a significant interaction between the severity of angina and the indication for PCI ($p_{\text{interaction}} = 0.010$). Among patients who presented with stable or unstable angina, each 10-point decrease in SAQ angina frequency score was associated with a 26% greater odds of angina after PCI. However, among NSTEMI patients, severity of angina was not as predictive for future angina, with every 10-point decrease in SAQ angina frequency score being associated with a non-significant 8% increased odds of angina after PCI. Independent of SAQ angina frequency scores, both a greater number of antianginal medications taken at the time of PCI and worse SAQ quality of life were strongly associated with the risk of angina after PCI. The spline term for the SAQ quality of life score was significant, and thus the variable was divided into four categories (poor = 0–49, fair = 50–74, good = 75–99, and excellent = 100). A clinical history of depression, self-reported avoidance of care due to costs, self-reported moderate or severe pain/discomfort on the EQ-5D, and younger age were weaker, but still independent predictors of angina 6 months after PCI.

Overall, the model demonstrated good discrimination (*c*-index = 0.75) and calibration with the observed outcomes, having an intercept of −0.01 (*P*-value for difference from 0 = 0.63), a slope of 1.04 (*P*-value for difference from 1 = 0.69), and an R^2 of 96%. The observed vs. predicted risk of angina after PCI within risk deciles is shown in *Figure 2*. The range of predicted risk of angina was very broad, with the lowest decile of risk having only a 5% predicted (4% observed) prevalence of angina at 6 months and the highest decile of risk having a 60% predicted (64% observed) rate. In the 1000 bootstrap samples, the optimism-adjusted *c*-index was 0.74, indicating that over-fitting was not a substantial issue with the model, and the calibration was also good, with an intercept of −0.004 (*P*-value for difference from 0 = 0.60) and a slope of 0.98 (*P*-value for difference from 1 = 0.54).

As a sensitivity analysis, to assure that we were able to effectively predict post-PCI angina using pre-procedural variables alone, we added a number of diseased vessels noted on the coronary angiogram to the final model. In this multivariable model, the number of diseased vessels was not significantly associated with angina after PCI [odds ratio (OR) 1.13, 95% confidence interval (CI) 0.99–1.29, $P = 0.066$], and the discrimination of the model did not change (*c*-index = 0.75).

Application of the model

Using the model containing only pre-procedural factors, we examined whether complete revascularization affected the observed rate of angina among patients with different levels of predicted angina (*Figure 3*). There was a significant effect of complete revascularization on the observed rate of risk-adjusted angina, which was most prominent among patients with higher predicted rates of angina. For example, among patients with a predicted risk of 6-month angina after PCI of 29–35%, those who had incomplete revascularization had an observed rate of angina of 41 vs. 27% among those with complete revascularization. After adjusting for the patient risk of angina, complete revascularization was associated with a significantly lower risk of angina 6 months after PCI (OR 0.44, 95% CI 0.38–0.49, $P = 0.010$).

Table 1 Baseline characteristics of patients with vs. without angina at 6 months after PCI

	Angina after PCI, $n = 616$	No angina after PCI, $n = 1957$	*P*-value
Age (years)	63.1 ± 10.7	65.2 ± 10.8	<0.001
Male sex (%)	66.2	72.1	0.005
White race (%)	90.4	92.3	0.134
High school or greater education (%)	88.1	92.1	0.002
Insurance for medications (%)	91.9	93.7	0.116
Self-reported avoidance of care due to cost (%)	20.4	10.7	<0.001
Hypertension (%)	87.2	83.0	0.013
Dyslipidaemia (%)	87.7	81.7	<0.001
Diabetes mellitus (%)	40.3	31.1	<0.001
Insulin use (%)	18.1	11.8	<0.001
Current smoker (%)	20.3	14.4	<0.001
Prior myocardial infarction (%)	33.9	23.4	<0.001
Prior PCI (%)	50.3	37.6	<0.001
Prior bypass graft surgery (%)	29.9	18.7	<0.001
Chronic heart failure (%)	13.5	8.4	<0.001
Atrial fibrillation (%)	9.6	8.4	0.379
Peripheral artery disease (%)	9.6	8.9	0.604
Prior stroke (%)	5.8	4.0	0.058
Body mass index (kg/m^2)	31.2 ± 6.5	30.2 ± 6.1	<0.001
Creatinine pre-PCI (mg/dL)	1.1 ± 0.7	1.1 ± 0.8	0.935
On dialysis (%)	1.1	0.9	0.633
Chronic lung disease (%)	16.6	11.4	<0.001
Depression (%)	14.4	6.7	<0.001
Haemoglobin pre-PCI (g/dL)	13.5 ± 1.6	13.7 ± 1.7	0.024
PCI indication			<0.001
Stable angina (%)	33.4	41.9	
Unstable angina (%)	47.6	35.7	
NSTEMI (%)	19.0	22.4	
Elective PCI (%)	68.7	67.0	0.452
No. of antianginal medications			<0.001
0 (%)	18.5	28.8	
1 (%)	45.7	48.9	
2 (%)	25.4	19.2	
3 (%)	9.0	2.9	
4 (%)	1.3	0.2	
Seattle Angina Questionnaire			
Angina frequency	57.3 ± 25.3	75.0 ± 23.0	<0.001
Angina stability	29.1 ± 27.7	39.3 ± 27.6	<0.001
Physical limitation	65.7 ± 25.8	79.9 ± 22.4	<0.001
Quality of life	43.9 ± 23.3	59.0 ± 25.2	<0.001
Treatment satisfaction	91.7 ± 11.9	94.6 ± 9.7	<0.001
Euroqol-5D			
No problem performing usual activities (%)	50.7	66.4	<0.001
No pain or discomfort (%)	42.7	59.8	<0.001
Not anxious or depressed (%)	56.0	71.5	<0.001

PCI, percutaneous coronary intervention; NSTEMI, non-ST-elevation myocardial infarction.

Discussion

In a large, multicentre cohort of patients undergoing PCI for stable angina, unstable angina, and NSTEMI, we identified a set of covariates and created a prediction model to estimate a patient's risk for angina 6 months after PCI. Importantly, as recommended by the American Heart Association,[18] this model included only pre-procedural factors—without the use of treatments,

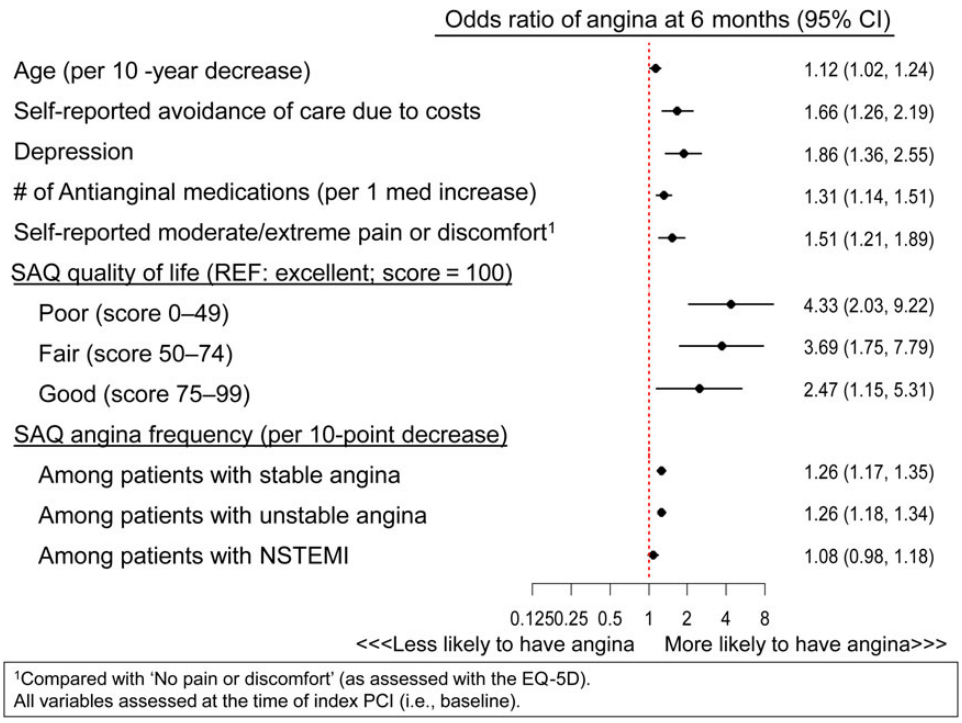

Figure 1 Factors independently associated with angina 6 months after PCI.

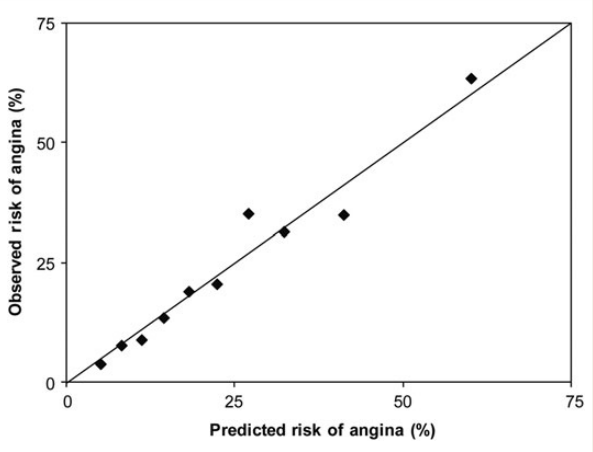

Figure 2 Calibration plot for prediction of angina at 6 months after PCI. Intercept of −0.01 [*P*-value (for difference from 0) = 0.63], a slope of 1.04 [*P*-value (for difference from 1) = 0.69], and R^2 of 96%.

complications, or repeat procedures occurring after the start of the initial PCI—and, as such, may be useful in calibrating patients' expectations of angina relief at the time of PCI. Furthermore, clinicians could use the results of this model to alter treatment decisions. For example, patients with appropriate coronary anatomy who are at high risk for residual angina after PCI might be offered bypass graft surgery instead of multivessel PCI,[19] or interventionalists may be more aggressive in completely revascularizing these patients, including the treatment of chronic total occlusions.[20,21] In fact, we were able to show that patients with high risks for residual angina had lower rates of observed angina if they received complete revascularization when compared with those who were incompletely revascularized and that complete revascularization had little effect on the rate of observed angina among patients who had a low risk of predicted angina. Such an analysis provides strong evidence of the validity of this model and its potential to serve as a foundation for future comparative effectiveness studies.

Beyond calibrating patient expectations and contributing to treatment decisions at the time of PCI, this model could support quality comparisons between hospitals or providers. Currently, PCI quality is primarily assessed by the short-term success of the procedure—both in terms of improvement in coronary flow and minimization of complications. However, the quality of a PCI should not be measured simply by technical success. Ideally, the success of PCI would also be judged by whether the goal of the procedure is actually attained, which in the majority of cases is the relief of angina. This outcome cannot be fairly compared across hospitals without accounting for the underlying risk of the patient, for which our model was designed. Using the presence of 6-month angina as a measure of healthcare quality is directly aligned with recent calls for improving the value of health care[22,23] and has been endorsed by the International Consortium for Health Outcomes Measurement. It occurs much more frequently than other outcomes currently used to assess the quality of PCI, such as mortality, and, even more importantly, is potentially modifiable by clinical care that is under the locus

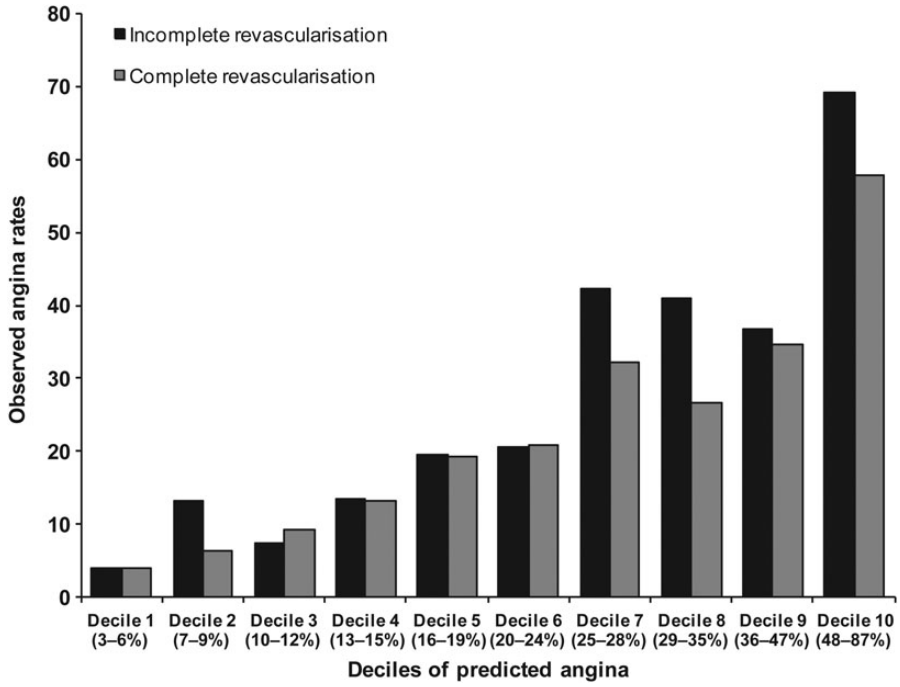

Figure 3 Observed rates of angina by decile of predicted risk, stratified by complete revascularization at the time of PCI.

of control of hospitals and physicians. Prior to implementation, further validation of this model is needed in addition to defining the proper statistical methods, including minimal sample sizes, for comparing hospitals and providers. We have previously published a similar risk-standardization model for disease-specific quality of life after an MI.[24] We are hopeful that these models will allow for the fair comparison of the quality of care from the patient's perspective and serve as a foundation for improving the quality of patient-centred care.

In terms of the model itself, we found that the most important predictors of whether a patient will be free of angina at 6 months after PCI were his/her angina burden (including number of antianginal medications required) prior to PCI and his/her quality of life related to that angina.[25,26] It is not surprising that these factors are predictors of angina after PCI, but it is rather noteworthy that these three factors were all independently and strongly associated with angina after PCI, as it would be expected that these factors would be correlated. These results highlight that although these two concepts (angina burden and disease-specific quality of life) are related, they are distinct and are both important predictors of whether or not a patient will have angina after PCI.

In addition, we found that the burden of angina in the preceding 4 weeks, as assessed with the SAQ, was less predictive of whether or not the patient would have angina after PCI in the setting of NSTEMI. This makes clinical sense, as in the setting of NSTEMI, the ruptured plaque often causes a marked acute worsening of chest pain, but this severity of chest pain is not necessarily reflective of chronic disease. Interestingly, though, while unstable angina is considered to be similar in pathophysiology to an NSTEMI, with a rupture-prone vulnerable plaque and accelerating angina, the association of pre-PCI angina with angina after PCI was very similar to stable angina patients without vulnerable plaque. In addition, we found higher rates of angina among patients who underwent PCI for unstable angina when compared with both NSTEMI and stable angina patients. This finding of more angina in patients with unstable angina compared with NSTEMI contradicts one prior study that found similar 1-year rates of angina among these two groups of patients (with ST-elevation MI patients have less angina).[26] The reasons for this are unclear but may reflect the difficulty of making the diagnosis of unstable angina (vs. progressive stable angina). As there are rarely any objective ischaemic markers with unstable angina, there may be some misclassification of patients with severe, progressive, difficult-to-treat stable coronary disease as unstable angina.

The other identified predictors, in particular younger age and depressive symptoms, have been shown to be associated with angina after an MI.[27,28] This prior work, however, has been largely descriptive and included procedural and post-procedural factors.[25] Our study built upon this prior work by creating a model on the basis of pre-procedural factors only, so that this information could be shared with patients at the time of PCI. In addition, we are hopeful that the model can serve as the basis for comparing treatments, such as stent type or smoking cessation, and that the patient-specific information gleaned from this model could not only inform patients but also influence treatment decisions (e.g. attending cardiac rehabilitation). We have already provided personalized risks of procedural complications to patients at the time of PCI,[29] but these personalized consent forms were not able to share the benefits of treatment, as could now be provided with this model.

There are several potential limitations to our analysis that should be acknowledged. First, although this study included 10 hospitals

across the USA, they were mostly large hospitals and academic medical centres. In addition, we were unable to externally validate the model. For both these reasons, the generalizability of the model should be tested in an independent data set. However, one of the strengths of the model is that it was developed in a real-world patient population, which enhances its generalizability. Secondly, although sustained relief of angina is also a goal of PCI in ST-elevation MI patients, there was an insufficient number of these patients to include them in the model, and the primary indication for the procedure was for improved survival. Moreover, it is highly unlikely that such a model would be run pre-procedurally in these patients, thereby decreasing its clinical usefulness. However, a model that demonstrates how angina varies with different interventions after PCI, such as smoking cessation and cardiac rehabilitation, could be quite useful in ST-elevation MI patients, even if run post-procedurally, and thus future work in a data set that includes more patients with ST-elevation MIs should be done. Finally, we found four important predictors of angina after PCI, which are not typically collected in routine clinical care (SAQ angina frequency, SAQ quality of life, EQ-5D pain/discomfort question, and self-reported avoidance of care due to cost). Although this represents a limitation to the implementation of the model in clinical care, we think that these results highlight the need to collect data such as health status and socioeconomic status. Recent introduction of a seven-item SAQ[30] means that only nine questions, requiring 3–5 min, are needed to predict patients' risks of angina at 6 months.

In conclusion, using a multicentre cohort of patients undergoing PCI for stable angina, unstable angina, and NSTEMI, we created a prediction model using pre-procedural factors alone that can reliably estimate a patient's individualized risk of angina at 6 months after PCI. Use of this model can not only help calibrate patients' expectations of angina relief after PCI but also serve as a foundation for studies to compare the effect of different treatments, during and after PCI, on angina relief. It also has the potential to become a patient-centred outcome for comparing the quality of PCI treatments across centres and operators. Future work to externally validate the model can help ensure its generalizability beyond large US hospitals and examine whether providing this information to patients changes their treatment choices or satisfaction.

Supplementary material

Supplementary material is available at *European Heart Journal—Quality of Care and Clinical Outcomes* online.

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