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Predicting coronary heart disease in remote settings: A prospective, cross-sectional observational study

Running title: Predicting CHD

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Abstract

Background

Coronary heart disease (CHD) places a major burden on the Australian healthcare system.

Determining the likelihood of CHD in a patient presenting with chest pain can be particularly difficult in a remote setting where access to transportation and specialised investigations including myocardial stress studies and coronary angiography can be difficult and delayed.

Objective

To develop a predictive model for determining the risk of CHD, including the value of high sensitivity C-reactive protein (hsCRP), in patients presenting with chest pain with a particular emphasis on resources and information likely to be available in a remote primary health care setting.

Methods

A prospective, cross-sectional observational study of patients with no prior diagnosis of CHD presenting to a specialist chest pain assessment clinic at Cairns Hospital from November 2012 to May 2013.

Results

Out of the 163 participants included in the study analyses, a total of 38 were classified as CHD likely (23.3%, 95% CI 17.1-30.6). Logistic regression modelling identified two factors that were independently associated with likely CHD, namely the presence of typical chest pain (OR 83.7, (95% CI 21.7-322.1)) and an abnormal baseline ECG (OR 12.8, (95% CI 1.9-86.0)).

Conclusion

In this study, it was demonstrated that the presence of typical chest pain and an abnormal resting ECG, remain the cornerstone of predicting a subsequent diagnosis of CHD. This information is easily accessible in remote primary health care and should be utilised to expedite assessment in patients presenting with symptoms suggestive of CHD.

Introduction

Coronary heart disease (CHD) places a major burden on the Australian healthcare system particularly for diagnosing and managing patients who present with symptoms that are suggestive of CHD. For the prompt diagnosis of CHD the issue of remoteness is particularly important as access to diagnostic investigations can be challenging and often delayed.

When considering the risk of CHD, it is important to distinguish between people who are currently asymptomatic and those who have symptoms. A large number of cardiac risk calculators exist to predict the risk of a future cardiovascular event in asymptomatic individuals [1-7]. Whilst such calculators have utility in determining subsequent cardiovascular risk in asymptomatic people, they are not useful or validated for assessing the likelihood of CHD in a patient presenting with a history of suggestive symptoms such as chest pain.

Determining the probability of a subsequently confirmed diagnosis of CHD in a patient presenting with a history of chest pain or other symptoms is less well defined. Typically it involves an *ad hoc* combination of clinician-perceived probability, history (including chest pain type), examination findings, and relevant investigations. The utility of these factors and newer markers of cardiovascular risk, such as the inflammatory marker C-Reactive Protein (CRP), remains poorly understood. This is particularly the case for people living in rural and remote Australia including many Aboriginal and Torres Strait Islander people. In this setting, an improved and validated approach to assessing the risk of CHD in patients presenting with a history of chest pain or associated symptoms is required. This would provide a more rational approach to balancing patient, family and local clinician preferences with the availability of finite transport and distant diagnostic, specialist and management services.

In patients presenting with a history of chest pain or other suggestive symptoms of CHD, the objective of this study was therefore to:

- i. Examine the usefulness of existing data available to primary and remote health care in predicting a subsequent diagnosis of CHD
- ii. Determine whether the addition of high sensitivity CRP (hsCRP) improved this prediction
- iii. Establish if such predictive algorithms were equally useful in Aboriginal and Torres Strait Islander people and non-Indigenous Australians.

Methods

Study design and setting

A prospective, cross-sectional observational study of patients referred to a northern Australian specialist cardiology chest pain clinic.

Eligibility criteria and selection of participants

Participants were a random sample of all men and women aged 18 years or older who were referred to the specialist cardiology chest pain clinic at the Cairns Hospital, Queensland, Australia from November 2012 to May 2013 and who did not have an existing diagnosis of CHD.

Variables measured

Variables assessed included potential predictive factors and outcome variables. Potential predictive factors related to demographics (age, gender, ethnicity), patient history (BMI, systolic and diastolic blood pressure, chest pain type, smoking and alcohol consumption, family history), existing medical diagnoses (including hypertension, diabetes mellitus, dyslipidaemia, chronic kidney disease, heart failure, cerebrovascular and peripheral vascular disease) and standardized baseline pathology (renal function, lipids (total cholesterol, HDL, LDL), percent glycosylated haemoglobin, full blood count including haemoglobin concentration, platelets, mean cell volume, red blood cell count and white cell count (including differential)) and a baseline resting ECG.

Chest pain type was based on the Diamond and Forrester classification with three domains assessed: retrosternal component; brought on by stress or exercise; and relieved promptly by rest or glyceryl trinitrate (GTN) [8]. Chest pain was defined as typical if all three criteria were met, atypical if two out of three criteria were met and, finally, non-anginal if one or no criteria was met. Smoking and alcohol consumption were classified as dichotomous variables, defined by whether the patient had never smoked/regularly drunk alcohol or was a past/current smoker or regular consumer of alcohol.

The primary outcome variable was a specialist cardiology assessment of the likelihood of CHD classified as likely or unlikely. Whilst an additional classification of 'indeterminate' was provided, this was not utilized in the classification of any patient.

Data measurement and bias

Demographic details were collected from patients at the time of enrolment and prior to chest pain clinic review. The consulting specialist cardiologists collected clinical and past history data using

standard data definitions. Primary outcome data (CHD likelihood) were obtained from the cardiologists' final clinical summaries relating to participants' diagnosis and management plans. Laboratory results were obtained from the hospital electronic laboratory database and results of additional cardiac investigation, when performed, were retrieved from patient health records. Reporting bias was possible due to the fact that there were three consultant cardiologists consulting in the chest pain clinic and possible variability in interpreting and reporting additional cardiac investigations.

Sample size

Sample size estimations were based on the recommendations of Katz [9] and Peduzzi *et al.* [10] who recommended 20 observations for each potential predictive variable. In this case it was assumed, based on earlier studies of CHD prediction, that 5-10 predictive factors might be encountered requiring a sample size of 100-200 patients.

Ethics and statistical analysis

Ethics approval was obtained from the Human Research Ethics Committees of the Cairns and Hinterland Health Service District and James Cook University. All participants provided written informed consent. Where necessary language and intercultural interpreters were used for Aboriginal and Torres Strait Islander patients.

Descriptive univariate analyses were performed on independent variables. A bivariate correlation matrix was developed for potential predictive and outcome variables to identify predictive factors associated with outcomes and the inter-relationship between predictive variables. Bivariate analyses included Student t-test, Wilcoxon rank-sum test, chi square, Fisher's exact test, linear regression and Spearman rank correlation test, as appropriate, based on data format and distribution.

Logistic regression models were developed to explore the relationship between predictive and outcome variables. Predictive variables incorporated in these models included those included in earlier studies and where bivariate analyses were associated with a p value <0.1. Where substantial co-linearity between predictive variables was identified, the factor with the highest statistically significant correlation was included. Significant correlation between potential predictive factors was further tested in models by inclusion as an additional interaction term. A p value <0.05 was considered statistically significant and all tests were two-sided.

Results

The study enrolled 189 participants. Of these, 26 (13.8 %) had a history of CHD and were excluded from subsequent analysis. Of the remaining 163 participants, a total of 38 were classified as CHD likely (23.3%, 95% CI 17.1-30.6). Table 1 outlines the demographic and clinical features (including chest pain type) of these participants stratified by whether they were Aboriginal and Torres Strait Islander people or non-Indigenous Australians. The results of baseline laboratory and cardiac investigations are outlined in Table 2.

A comparison of selected patient, clinical and baseline investigation findings stratified by whether CHD was felt to be likely or unlikely are outlined in Table 3. Aboriginal and Torres Strait Islander people were more likely to have a final diagnosis of CHD (40.9% (95% CI 20.7-63.7)) compared with 20.6% ((95% CI 14.2-28.2), $p=0.036$). Logistic regression modelling identified two factors that were independently associated with likely CHD, namely the presence of typical chest pain (OR 83.7, (95% CI 21.7-322.1)) and an abnormal baseline ECG (OR 12.8, (95% CI (1.9-86.0))). The Nagelkerke R-square value for the final prediction model was 0.62 indicating nearly two thirds of the variability in the clinical likelihood of CHD was explained by these two factors. The probability of the presence of likely CHD in patients with one or both of these factors is outlined in Table 4.

Discussion

There are few well-validated tools for assessing the eventual risk of CHD in patients with a history of suggestive symptoms such as chest pain and none that have been validated in remote or Aboriginal and Torres Strait Islander populations. In addition, there are no published studies that have looked at the utility of hsCRP at diagnosing CHD in patients with a history of chest pain. Our study aimed to address these identified knowledge gaps.

The only two factors that were consistently and independently associated with an eventual high likelihood of CHD in our population group (as determined by a cardiologist following clinical review and further investigation) were an abnormal baseline ECG, when chest pain was no longer present, and a history of chest pain typical for cardiac ischaemia. Of note, it was the chest pain type that substantially increased the odds of eventual CHD, suggesting that clinical skills and history taking remain extremely relevant in this setting. The Nagelkerke R-square value of 0.62 for the final logistic regression model, demonstrates that the two independent variables explained a substantial amount of the variation in the eventual likelihood of CHD.

Interestingly, in our study sample, other factors that have been identified in the few existing validated tools were not associated with an increased likelihood of CHD. Diamond and Forrester developed one of the earliest and most commonly cited models for assessing the likelihood of CHD in patients presenting with a history of chest pain [8]. In line with our study, it was typical anginal chest pain, which had the greatest impact on the probability of CHD. However, additional factors identified by Diamond and Forrester, which were not significant in our study included increasing age and male gender [8]. Other studies have also identified other significant factors associated with an increased likelihood of CHD, which were not demonstrated in our study. The more recent Duke risk score identified similar factors to our study (symptoms of angina pectoris and ECG changes) [11]. Nonetheless it also highlighted that age, tobacco use, diabetes, history of myocardial infarction and hypercholesterolaemia had additional and independent predictive utility [11]. Additional predictive factors identified by the Morise risk score included BMI, obesity (BMI>27), family history of CHD and oestrogen status [12]. Despite the increased risk of CHD borne by Aboriginal and Torres Strait Islander people this did not add any additional predictive power to our model.

There are a number of possible factors that could explain why variables identified in other studies were not associated with an increased likelihood of CHD in our study. In comparison to the other models outlined above, the sample size of this study was smaller (n=163), however, an advantage was the target population of Aboriginal and Torres Strait Islander and non-Indigenous Australians as no other studies have validated their tools in this population. Also, there was possible reporting bias as there were three different consultant cardiologists involved in the study giving the opportunity for

variability in final assessment of likelihood and interpreting and reporting additional cardiac investigations.

The primary outcome of this study was based on a specialist cardiology assessment of the likelihood of CHD. The treating cardiologist defined patients as CHD likely or unlikely following patient history, clinical examination, routine investigations such as an ECG and any further investigations including myocardial stress studies and angiography. This is in contrast to the other existing tools which all defined the pre-test probability of CHD based on at least one coronary artery stenosis, ranging from $\geq 50\%$ to $\geq 75\%$ stenosis, on coronary angiogram [8, 11, 12]. Nonetheless, we believe our clinical endpoint more closely reflects a practical gold standard for CHD diagnosis that reflects usual clinical care, namely a combination of specialist review and additional investigations when or if indicated.

The implications of this study for health care delivery and the broader health service are highlighted by the final logistic regression model. It is apparent that if a patient had atypical or non-anginal chest pain with a normal baseline ECG, they had a low probability of a subsequent diagnosis of CHD (5.3%). This is in contrast to participants in the study with typical chest pain and an abnormal baseline ECG, who were all found to have CHD (100%). It can be further appreciated that it was the type of chest pain that had a greater impact on the outcome, as over 80% of people with typical chest pain and a normal ECG were likely to have CHD (Table 5). The findings of this study support the current literature. They reinforce that clinical skills, namely history taking, and the use of basic investigations including ECG, which is typically available in even the most remote primary health care setting, should be prioritised when determining the timing and nature of further clinical review.

To date, there have been no published studies that have examined the utility of hsCRP in this setting. Our study did not support the use of this emerging biomarker to improve the prediction of a subsequent diagnosis of CHD in patients presenting with a history of chest pain. Whilst hsCRP may have an important future role in directing primary and secondary prevention of CHD, we have not demonstrated utility in facilitating diagnosis [13-18].

A major strength of this study was that there have been no previously published studies to date which have examined the utility of hsCRP at diagnosing CHD in patients with a history of chest pain. In addition, the study sample was reflective of regional and remote Australia as participants were recruited from a northern Australian specialist cardiology clinic. A limitation that could be identified is the relatively small sample size, particularly the number of Aboriginal and Torres Strait Islander participants. However, the sample size comprised 13.5% Aboriginal and Torres Strait Islander participants, which is an over-representation of this population in north Queensland, where the study was based.

Conclusion

This study aimed to develop an algorithm for predicting an eventual diagnosis of CHD in patients presenting with chest pain. It also investigated the utility of the emerging biomarker, hsCRP, to determine if this provided any addition predictive power. We demonstrated that in this setting, it is the presence of typical chest pain and an abnormal resting ECG, which remains the cornerstone of predicting a subsequent diagnosis of CHD in a patient presenting with chest pain.

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Table 1. *Demographic and clinical features of participants stratified by Indigenous status*

	Aboriginal and/or Torres Strait Islander n = 22		Non-Indigenous Australian n = 141		P value
	N		N		
Age (years): mean (SD [#])	22	52.68 (11.08)	141	57.18 (12.19)	0.015
Gender (%): female, (95% CI)	14	63.6 (40.66-82.80)	71	50.4 (41.82-58.88)	0.246
BMI (kg/m ²): mean (SD)	18	30.78 (7.36)	109	30.98 (6.57)	0.906
BP systolic (mmHg): mean (SD)	18	134.06 (21.64)	127	133.77 (16.71)	0.948
BP diastolic (mmHg): mean (SD)	18	77.06 (14.98)	127	80.19 (11.42)	0.298
ETOH (standard drinks/week): mean (SD)	16	6.19 (12.01)	102	7.42 (17.03)	0.781
Never smoked: % (95% CI)	6	27.3 (10.73-50.22)	60	48.4 (39.3-57.5)	0.140
HTN: % (95% CI)	17	77.3 (54.63-92.18)	61	44.2 (35.76-52.90)	0.004
Diabetes: % (95% CI)	9	40.9 (20.71-63.65)	17	12.4 (7.40-19.12)	0.001

Dyslipidaemia: % (95% CI)	12	54.5 (32.21-75.61)	63	44.7 (37.44-54.70)	0.455
Chronic kidney disease (eGFR<60mL/min/1.73m ²): % (95% CI)	2	11.8 (1.46-36.44)	8	7.5 (3.31-14.33)	0.555
Cerebrovascular disease: % (95% CI)	2	9.1 (1.12-29.16)	2	1.5 (0.18-5.17)	0.092
Heart Failure: % (95% CI)	1	4.5 (0.12-22.84)	0	0 (0-2.66)	0.138
Peripheral vascular disease: % (95% CI)	0	0 (0-15.44)	1	0.7 (0.02-4.00)	1.000
Family history in 1 st degree relative <60yo: % (95% CI)	7	43.8 (19.8-70.1)	42	38.9 (29.7-48.8)	0.171
Typical chest pain: % (95% CI)	7	31.8 (13.9-54.9)	23	16.3 (10.6-23.5)	0.351

SD – standard deviation

Table 2. Baseline investigations of participants stratified by Indigenous status

	Aboriginal and/or Torres Strait Islander n = 22		Non-Indigenous Australian n = 141		P value
	N		N		
hsCRP (mg/L): median (IQR*)	20	3.45 (1.6, 4.9)	130	1.5 (0.7, 2.9)	0.002
<i>Blood glucose profile</i>					
Random BSL (mmol/L): mean (SD [#])	16	7.26 (1.91)	103	6.5 (1.65)	0.093
HbA1C (%): median (IQR)	15	6.10 (5.3, 7.6)	97	5.7 (1.05)	0.020
<i>Full blood count</i>					
Haemoglobin (g/L): mean (SD)	17	142.4 (10.77)	106	140.7 (15.23)	0.664
WBC (x10 ⁹ /L): mean (SD)	17	9.9 (2.77)	106	7.84 (2.19)	0.001
<i>Lipid profile</i>					
Total cholesterol (mmol/L): mean (SD)	15	4.54 (0.87)	105	5.06 (1.20)	0.109
HDL (mmol/L): mean (SD)	15	1.21 (0.27)	105	1.25 (0.37)	0.694
LDL (mmol/L): median (IQR)	15	2.60 (1.7, 2.9)	97	2.70 (2.0, 3.6)	0.276
Troponin T: median (IQR)	17	0.00 (0.0, 0.01)	112	0.01 (0.0, 0.01)	0.706
Urea/creatinine: mean (SD)	18	72.80 (15.71)	114	82.3 (22.7)	0.090

ECG abnormal: % (95% CI)	2	10.5 (1.3-33.1)	7	5.6 (2.26-11.11)	n/a*
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*IQR – inter-quartile range # SD – standard deviation

Table 3. Selected patient, clinical and baseline investigation findings stratified by CHD likelihood

	Aboriginal and/or Torres Strait Islander					Non-Indigenous Australian				
	n = 22					n = 141				
	N	CHD likely	N	CHD unlikely	P value	N	CHD likely	N	CHD unlikely	P value
Age (years): mean (SD [#])	9	51.4 (8.2)	13	53.5 (13.0)	0.674	29	61.3 (9.2)	112	56.1 (12.7)	0.039
Gender: % (95% CI)										
Female	5	55.6 (21.2-86.3)	9	69.2 (38.6-90.9)	0.662	17	58.6 (38.9-76.5)	54	48.2 (38.7-57.9)	0.318
ETOH (standard drinks/week):	5	1.0 (0, 21)	11	0 (0, 1)	0.380	21	3.00 (1.0, 1.4)	81	1.0 (0, 5.50)	0.006
Median (IQR*)										
Never smoked: % (95% CI)	2	22.2 (2.8-6.0)	4	30.8 (90.9-61.4)	1.000	10	41.7 (22.1-63.4)	50	50.0 (39.8-60.2)	0.463
Hypertension: % (95% CI)	9	100.0 (66.4-100.0)	8	61.5 (31.6-86.1)	0.054	15	53.6 (33.9-72.5)	46	41.8 (32.5-51.6)	0.264
Diabetes: % (95% CI)	4	44.4 (13.7-78.8)	5	38.5 (13.9-68.4)	0.779	5	17.9 (6.1-36.9)	12	11.0 (5.8-18.4)	0.327
Dyslipidaemia: % (95% CI)	6	66.7 (29.9-92.5)	6	46.2 (19.2-74.9)	0.342	15	53.6 (33.9-72.5)	48	44.0 (34.5-53.9)	0.401
Family history in 1 st degree relative	2	40.0 (5.3-85.3)	5	45.5 (16.8-76.6)	n/a*	8	34.8 (16.4-57.3)	34	72.3 (29.5-51.2)	0.649
<60yo: % (95% CI)										
Typical chest pain: % (95% CI)	7	77.8 (40.0-97.2)	0	0 (0-24.7)	0	10	34.5 (18.0-54.3)	108	96.4 (91.1-99.0)	0

hsCRP (mg/L): Median (IQR)	7	2.3 (0.9, 6.2)	13	3.6 (2.0, 4.8)	0.451	27	1.2 (0.7, 2.03)	103	1.6 (0.7,3.1)	0.402
<i>Blood glucose profile</i>										
Random BSL (mmol/L): mean	7	7.8 (2.5)	9	6.9 (1.3)	0.404	20	6.8 (2.1)	83	6.4 (1.5)	0.376
(SD)	6	6.3 (5.4, 11.3)	9	6.2 (1.0)	0.442	19	5.9 (1.4)	78	5.7 (1.0)	0.362
HbA1C (%): mean (SD)										
<i>Renal function test</i>										
Urea/creatinine: mean (SD)	7	74.7 (13.0)	11	71.6 (17.7)	0.698	23	90.0 (21.7)	91	80.4 (22.7)	0.069
Abnormal ECG: % (95% CI)	1	16.7 (0.4-64.1)	1	7.7 (0.2-36.0)	1.000	5	20.8 (0.1-0.4)	2	2 (0.2-6.9)	0.003

*IQR – inter-quartile range [#] SD – standard deviation

Table 4. *Multivariate logistic regression analyses showing the probability of CHD likelihood based on chest pain type and ECG*

PROBABILITY OF CHD	ECG Normal	ECG Abnormal
(%, 95% CI)		
Atypical/non-anginal chest pain	5.3% (2.0-11.1)	40.0% (5.3-85.3)
Typical chest pain	81.8% (59.8-94.8)	100.0% (40.0-100.0)