A Predictive Model for Distinguishing Ischemic from Non-Ischemic Cardiomyopathy

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Objectives: *To develop a predictive model to distinguish ischemic from non-ischemic cardiomyopathy*
Material and Method: *The authors randomly assigned 137 patients with LV systolic dysfunction into two subsets - one to derive a predictive model and the other to validate it. Clinical, electrocardiographic and echocardiographic data were interpreted by blinded investigators to the subsequent coronary angiogram results. Ischemic cardiomyopathy was diagnosed by the presence of significant coronary artery disease from the coronary angiogram. The final model had been derived from the clinical data and was validated using the validating set. The receiver-operating characteristics (ROC) curves and the diagnostic performances of the model were estimated.*
Results: *The authors developed the following model: Predictive score = (3 x presence of diabetes mellitus) + number of ECG leads with abnormal Q waves – (5 x presence of echocardiographic characteristic of non-ischemic cardiomyopathy). The model was well discriminated (area under ROC curve = 0.94). Performance in the validating sample was equally good (area under ROC curve = 0.89). When a cut-off point $\geq$ 0 was used to predict the presence of significant coronary artery disease, the model had a sensitivity, specificity and positive and negative predictive values of 100%, 57%, 74% and 100%, respectively.*
Conclusion: *With the high negative value of this model, it would be useful for use as a screening tool to exclude non-ischemic cardiomyopathy in heart failure patients and may avoid unnecessary coronary angiograms.*

Keywords: *Predictive model, Non-invasive, Distinguish, Ischemic cardiomyopathy*

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Heart failure is a worldwide major public health problem. Morbidity and mortality are extremely high[1,2]. The two most common causes of heart failure with left ventricular systolic dysfunction are ischemic and idiopathic dilated cardiomyopathy[3-7], in which the latter, heart transplant is the only definite treatment. The prevalence of underlying coronary artery disease in patients with heart failure varies from 34%-66%[7-10]. Coronary artery bypass grafting has been shown to improve symptoms and survival in this group of patients, who had angina. Even in those without angina, the observational studies have shown that revascularization could favorably affect left ventricular function in a significant number of patients with impaired yet viable myocardium[11]. Therefore, it would be beneficial to detect the functional significance of coronary artery disease in patients whose etiologies of heart failure are not yet identified.

Coronary angiography is the gold standard for the differentiation of ischemic and non-ischemic cardiomyopathy[1]. However, it is not practical to evaluate all patients with systolic heart failure by coronary angiogram because of the expense and invasive nature of this procedure. It would be of great value to be able to distinguish patients with nonischemic cardiomyopathy from those with ischemic cardiomyopathy noninvasively with sufficient accuracy to avoid unnecessary coronary angiography. Non-invasive techniques such as thallium scintigraphy is costly,while dobutamine stress echocardiography is an operator-dependent procedure and is not generally available.

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The purpose of the present study was to generate the pragmatic non-invasive methods as a diagnostic tool to distinguish ischemic and non-ischemic cardiomyopathy by developing and validating a predictive model, using clinical data, electrocardiography and echocardiography in combination.

Material and Method

All patients, aged > 15 years with clinical heart failure in Maharaj Nakorn Chiang Mai Hospital, were consecutively considered for recruitment. Comprehensive history and physical examination were completed in every patient. Specific cardiac tests and metabolic studies were performed as clinically indicated including, but not limited to: standard 12 leads electrocardiography, chest X-ray, thyroid function tests, electrolytes (including calcium and magnesium), complete blood count, echocardiography.

The echocardiographic measurements were performed in all four standard views. Left ventricular ejection fraction was measured by Modified Simpsons technique, obtained from both apical four-chamber and apical two-chamber views when endocardial definition was clear enough, otherwise Teicholz technique was used. Right ventricular function was evaluated qualitatively by 2D imaging from several different views: parasternal long- and short-axis, right ventricular inflow, apical four-chamber views and was graded as normal or impaired.

All patients who had left ventricular ejection fraction by echocardiography less than 45%, agreed to participate in the present study and gave informed consent were recruited. Exclusion criteria were previous documented myocardial infarction by WHO criteria, severe valvular heart disease, congenital heart disease, HIV infection, severe debilitating illness, those in which causes of cardiomyopathy were readily identified such as overt hyperthyroid, drug-induced cardiomyopathy, amyloidosis etc.

Coronary angiograms were performed in every patient. Electrocardiography, echocardiography and coronary angiographic results were reviewed by the cardiologists who were unaware of the patients' status. Interobserver variation in assessing qualitative data (right ventricular function and regional wall motion abnormality) was also tested.

Definitions

The presence of abnormal Q wave was defined by the duration of Q waves in the limb and chest leads > 0.03 seconds. The amplitude of Q waves > 25% of R wave in the limb leads and > 0.2 mV in the chest leads[12].

The presence of significant coronary artery disease judged to be the etiology of LV systolic dysfunction was defined from coronary angiographic data as the presence of $\geq$ 75% of area stenosis of left main or proximal LAD or $\geq$ 75% of area stenosis of two or more epicardial vessels[13] and with agreement of two cardiologists to be the cause of left ventricular systolic dysfunction.

Statistical analysis

The authors randomly assigned 137 patients with LV systolic dysfunction who met the eligible criteria into two subsets - one to derive the model and the other to validate it.

The ratio of patients between the first and second subset was 2:1. Independent variables for the model were identified, and then the effect of each potential predictor was examined using univariate logistic regression. Model fitting was done using backward elimination.

After the final model had been obtained, the receiver-operating characteristics (ROC) curve was constructed. The area under the ROC curve and its 95% confidence interval (CI) were then estimated. The model was then validated using the validating set. The ROC curve was reconstructed, and the area under the ROC curve and its 95% CI were estimated. The diagnostic performance of the model for sensitivity, specificity and positive and negative predictive values were estimated together with their 95% CIs.

Statistical analysis was performed with SPSS version 10.0 for Windows (SPSS Inc., Chicago, Illinois) and MedCalc 7.1.0.1 for Windows (MedCalc, Belgium).

Results

Patient characteristics of the deriving set

Among 90 patients in the deriving set, 47 (45.6%) patients had significant coronary artery disease. Eighteen potential predictors of the presence of significant coronary artery disease were prospectively specified. The effect of these predictors was examined in univariate analysis (Table 1-3). The patients with significant coronary artery disease were older and had more risk factors for coronary artery disease than non-ischemic patients. The history of smoking, family history of premature coronary artery disease and alcohol consumption were not different between the two groups. There was a higher prevalence of male patients in the non-ischemic group while the male and female cases in the ischemic group were almost equal.

Table 1. Univariate analysis: unadjusted odds ratio (OR) of each clinical variable in prediction of the presence of significant coronary artery disease

Variables	Non-ischemic N = 49	Ischemic N = 41	OR	95%CI	p value
Age (mean-yrs)	55.6	61.3			0.017
Male	39 (79.6%)	19 (46.3%)	0.48	0.308-0.737	0.001
Chestpain					
- no	34 (69.4%)	16 (39.0%)			
- atypical	14 (28.6%)	12 (29.3%)			
- typical	1 (2.0%)	13 (31.7%)			<0.001
Diabetes mellitus	8 (16.3%)	21 (51.2%)	5.38	2.03-14.25	0.001
Hypertension	11 (22.4%)	25 (61.0%)	5.40	2.15-13.53	<0.001
Dyslipidemia	23 (46.9%)	29 (70.7%)	2.73	1.14-6.56	0.039
Smoking	15 (30.6%)	18 (43.9%)	1.77	0.75-4.22	0.28
Family history of CAD	3 (6.1%)	2 (4.9%)	0.79	0.12-4.95	1.00
Alcohol use	13 (26.5%)	4 (9.8%)	0.30	0.09-1.00	0.08

Table 2. Univariate analysis: unadjusted odds ratio (OR) of each electrocardiographic variable in prediction of presence of significant coronary artery disease

Variables	Non-ischemic N = 49	Ischemic N = 41	OR	95%CI	p value
Presence of Q waves	19 (38.8%)	26 (63.4%)	2.74	1.16-6.45	0.034
Number of leads with Q waves (mean)	0.67	1.95			<0.001
ST-T abnormalities	25 (51%)	31 (75.6%)	2.98	1.20-3.77	0.029
Bundle branch block					
- absence	38 (77.6%)	35 (85.4%)			0.13
- LBBB	7 (14.3%)	1 (2.4%)			
- RBBB	4 (8.2%)	5 (12.2%)			
RV6/Rmax (I, II, III)	2.22	1.67			0.029

Table 3. Univariate analysis: unadjusted odds ratio (OR) of each echocardiographic variable in prediction of the presence of significant coronary artery disease

Variables	Non-ischemic N = 49	Ischemic N = 41	OR	95%CI	p value
Ejection fraction%	28.43	30.95			0.112
LVDD (mm)	62.34	60.45			0.246
RWMA	24 (49.0%)	37 (90.2%)	9.64	2.98-31.16	<0.001
Impaired RV function	43 (81.8%)	17 (41.5%)	0.01	0.03-0.28	<0.001
Echo characteristics[a] of non-ischemic cardiomyopathy	40 (81.6%)	8 (19.5%)	0.06	0.02-0.16	<0.001

LVDD = LV diastolic dimension, RWMA = regional wall motion abnormality, a = combination of impaired RV function and either global LV hypokinesia or RWMA with relatively preserved inferoposterior wall motion, Echo = echocardiographic

Most of the patients from both groups had atypical chest pain or no chest pain. Typical angina was found in only 31% of the ischemic patients, and much less in the non-ischemic group (7.1%).

Abnormal Q waves were seen more in the ischemic group (63.4% compared with 38.8% in the

non-ischemic group, $p < 0.05$). There were 8 patients who had left bundle branch block, seven of whom had non-ischemic cardiomyopathy. The non-ischemic group had a higher voltage ratio of RV6/Rmax in I, II, III than the ischemic group.

There were no differences in the ejection fraction and left ventricular diastolic dimension between the two groups (mean EF 29.6%, LVDD 61.5 mm). Eighty six percent of the patients who had global LV hypokinesia and 71.7% of those who had impaired RV systolic function were in the non-ischemic group. The majority of the patients who had a combination of these two findings were in the non-ischemic group (88%).

Although these echocardiographic features were highly predictive for non-ischemic[12] cardiomypathy, half of the non-ischemic patients also had regional wall motion abnormality but with less frequency than ischemic patients. In non-ischemic group, we observed a striking echocardiographic characteristic, which was the presence of regional wall motion abnormalities with relatively preserved inferoposterior wall motion when compared with the more severe hypokinesia of anteroseptal, anterior wall. The authors demonstrated that the combination of overtly impaired RV systolic function and either global hypokinesia or regional wall motion abnormality with relatively preserved motion of inferoposterior LV wall was the echocardiographic characteristics of non-ischemic cardiomyopathy. It was found in 81.6% of the non-ischemic patients and only 19.5% of the ischemic patients in the deriving set.

The interobserver agreement between two blinded observers in assessing RV function and detecting echocardiographic characteristic of non-ischemic cardiomyopathy was 92%.

Development of the predictive model

The statistically significant variables were examined for their efffects in the multivariate logistic regression (Table 4).

The predictive model obtained can be written as:

P = 1/(1 + Exp[13.45 x presence of diabetes mellitus + 4.42 x number of ECG leads with abnormal Q waves – 0.011 x presence of echocardiographic characteristic of non-ischemic cardiomyopathy]).

The final model was simplified to the predictive score as (Table 5):

Table 4. The initial multivariate logistic regression model:odds ratio (OR) of each variable, adjusted for all other variables in prediction of the presence of significant coronary artery disease

Variables	Coefficient	Standard error	p value
Sex	3.753	0.993	0.093
Age	1.055	0.042	0.194
Diabetes mellitus	13.448	1.213	0.032
Hypertension	2.601	0.947	0.313
Dyslipidemia	1.477	0.920	0.679
Chest pain	3.845	0.721	0.062
Number of ECG leadswith Q waves	4.419	0.455	0.001
RV6/Rmax ratio	0.570	0.458	0.219
Echo characteristic of non-ischemic cardiomyopathy	0.011	1.519	0.003
Constant	0.003	3.083	0.055

Echo = echocardiographic

Table 5. The final model

Variables	Coefficient	Standard error	Exp. Coefficient	p value
Diabetes mellitus	13.448	1.213	2.6	0.032
Number of ECG leads with Q waves	4.419	0.455	1.4	0.001
Echo characteristic of non-ischemic cardiomyopathy	0.011	1.519	-4.7	0.003
Constant	0.003	3.083		0.055

Echo = echocardiographic, Predictive score = 2.6 (diabetes mellitus 0, 1) + number of ECG leads with Q waves – 4.7 (echocardiographic characteristic of non ischemic cardiomyopathy 0.1)

Predictive score = (3 x presence of diabetes mellitus) + number of ECG leads with abnormal Q waves – (5 x presence of echocardiographic characteristic of non-ischemic cardiomyopathy).

The area under the ROC curve of the final model was 0.94 (95%CI 0.89-0.98) (Fig. 1a). The model fit the data well (goodness-of-fit test, p = 0.96).

Validation of the model

The validating set of 47 patients was used to validate the model. The area under the ROC curve was 0.89 (95%CI 0.71-0.96) (Fig. 1b). The performance of the model in the validating sample was not significantly different from that of the deriving set (p = 0.90). The model fit the data well (goodness-of-fit test, p = 0.85).

The optimal cut-off point of predictive score, as suggested by the ROC curve, was 0.

Assessing the diagnostic performance of the model

The performance of the model was tested against the validating set by substituting the key characteristics of each patient which consisted of

1) presence of diabetes mellitus (0 or 1)

2) number of ECG leads with abnormal Q waves (0 – 11, exclude lead aVR)

3) presence of echocardiographic characteristics of non-ischemic cardiomyopathy (0 or 1) into the model to obtain predictive score. The authors used a cut-off point of score ≥ 0 to predict the presence of significant coronary artery disease, and then the authors compared the outcome predicted by the model (diagnostic test) with the true outcome (reference standard). The model had a sensitivity of 100% (95%CI 87-100%), specificity of 57% (95%CI 37-75%), positive predictive value of 74% (95%CI 58%-85%) and negative predictive value of 100% (95%CI 87-100%).

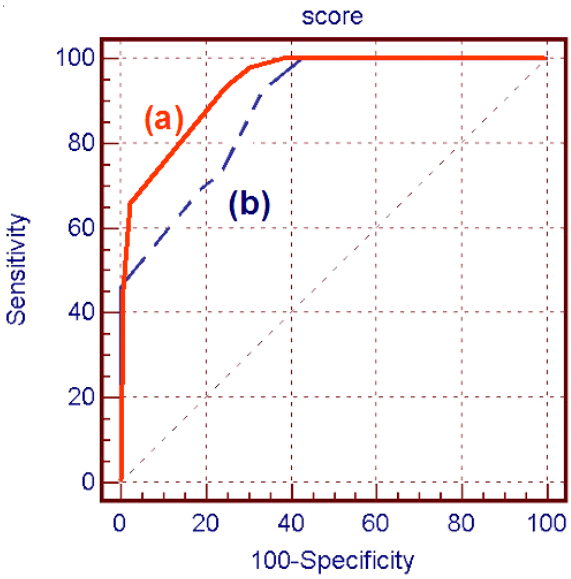

Fig. 1 Receiver-operating characteristics (ROC) curves for prediction model derived from (a) the deriving set (solid line;area under the ROC curve = 0.94) and (b) the validating set (dotted line;area under the ROC curve = 0.89). Test for equivalence of the two area under the ROC curves, p = 0.23

Discussion

The presentation of non-ischemic and ischemic cardiomyopathy may be clinically indistinguishable. In view of the differing natural history, prognosis and treatment of the two conditions, accurate differentiation between them is important.

Coronary angiography is the gold standard for the differentiation of these conditions. However, the invasive nature of the procedure and the expense are the limitations of this method. Several studies have evaluated the non-invasive methods to differentiate ischemic and non-ischemic cardiomyopathy but the results were inconclusive. Figulla et al[14] showed that non-invasive tests were inadequate to predict ischemic from idiopathic dilated cardiomyopathy. The diagnosis of idiopathic dilated cardiomyopathy in the present study was derived from clinical data (gender, age, patient history of exertional dyspnea and no angina pectoris on exertion), electrocardiography (absence of Q wave, presence of left bundle branch block, absence of ST segment abnormality during stress test) and echocardiography (left ventricular end-diastolic enlargement > 56 mm). The sensitivity and positive predictive value of noninvasive diagnostic methods were 59% and 66%, respectively.

The reasons for the contradiction to the present study might be because the present study was retrospectively analyzed and some data may be incomplete. Moreover, the echocardiographic data in the study of Figulla did not include either the presence of the LV regional wall motion abnormality or the presence of impaired RV systolic function. The presence of regional wall motion abnormality from echocardiogram was used to predict coronary artery disease in previous studies, the results were conflicting, because it could also be detected in patients with non-ischemic cardiomyopathy[15,16]. The presence of impaired RV systolic function to predict these two entities was also inconclusive[17]. Importantly, the echo-cardiographic characteristics to predict non-ischemic cardiomypathy,

which was the combination of overtly impaired RV function and either global LV hypokinesia or regional wall motion abnormality with relatively preserved inferoposterior wall observed in the present study and being the most significant variable in the present predictive model could distinctly explain the different results from other previous studies.

In the present study, the authors observed that typical angina was found more in ischemic patients, but chest pain was not a statistically significant predictor for ischemic cardiomyopathy in multivariate analysis, possibly since most of the patients did not have typical angina.

The patients with significant coronary artery disease had abnormal Q waves in more ECG leads and had a lower voltage ratio of RV6/Rmax in I, II, III than non-ischemic patients, comparable to the study of Momiyama et al[18]. The Momiyama study showed that ischemic patients had lower RV6 voltage than non-ischemic patients because of myocardial apical scarring which was usually associated with coronary artery disease and non-ischemic patients had lower R wave voltages in the limb leads, which were possibly affected by interstitial or replacement fibrosis in non-ischemic cardiomyopathy. The RV6/Rmax in I, II, III ratio was a useful index for the diagnosis of non-ischemic cardiomyopathy with 100% positive predictive value, 80% negative predictive value and 85% accuracy. Contrastingly, the present study showed that only the number of ECG leads with abnormal Q waves was a significant variable in multivariate analysis, not RV6/Rmax in I, II, III ratio.

The clinical diagnosis of ischemic cardiomyopathy is made in patients with LV dysfunction who have a history of myocardial infarction or based on objective evidence of coronary artery disease such as angiography or functional testing. In the present study, the authors used the coronary angiography as a gold standard to differentiate ischemic from non-ischemic cardiomyopathy. However, the appropriate classification of ischemic cardiomyopathy is not clear. In previous studies[15-17], an ischemic etiology of cardiomyopathy by angiography was defined as the presence of any epicardial coronary vessels with $\geq$ 75% stenosis. With this definition, the extent of coronary artery disease in some patients with the clinical diagnosis of ischemic cardiomyopathy does not seem to correlate with the degree of LV dysfunction. Recently, Felker et al[13] have proposed the new definition of ischemic cardiomyopathy by angiography. They demonstrated that more extensive coronary artery disease is independently associated with a shorter survival, and patients with single-vessel disease with no history of myocardial infarction or revascularization had a similar prognosis to those with nonischemic etiology and should be classified as nonischemic cardiomyopathy unless they have left main or proximal LAD disease. The authors chose the definition proposed by Felker et al for the ischemic etiology in the present study based on the accepted knowledge that ischemic etiology is independently associated with worse long-term outcome in patients with LV dysfunction.

The limitations of the study

The echocardiographic characteristics of non-ischemic cardiomyopathy, especially impaired RV function was qualitatively assessed, therefore it is prone to vary among individual operators. However, the qualitative evaluation of RV function is a simple, practical and generally accepted method[19] and inter-observer agreement in estimating RV function and detecting characteristic of non-ischemic cardiomyopathy was very high. The sample size of the validating set was rather small. Further validating studies with a larger sample size in different health care settings to confirm this predictive model should be considered.

This model has a specificity of 57%. Therefore, it is not a highly predictive model to predict ischemic cardiomyopathy. However, the very high sensitivity and high negative predictive values of 100% make this model very useful as a screening tool to exclude non-ischemic cardiomyopathy and may avoid unnecessary coronary angiograms.

Conclusion

The authors have developed a predictive model to differentiate ischemic from non-ischemic cardiomyopathy, derived from clinical data (presence of diabetes mellitus), ECG (number of ECG leads with abnormal Q waves) and echocardiographic data (presence of echocardiographic characteristic of non-ischemic cardiomyopathy).

Using only 3 variables to derive a predictive score and with such a high negative predictive value, this simple model could be useful for clinicians to avoid unnecessary coronary angiograms.

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Predictive model เพื่อวินิจฉัยแยก ischemic cardiomyopathy จาก non-ischemic cardiomyopathy

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วัตถุประสงค์: *ต้องการสร้าง predictive model เพื่อใช้ช่วยในการวินิจฉัยแยกโรคหัวใจล้มเหลวที่เกิดจากเส้นเลือดโคโรนารีตีบ จากโรคหัวใจล้มเหลวจากสาเหตุอื่น*

วัสดุและวิธีการ: *ศึกษาในผู้ป่วยหัวใจล้มเหลวที่เกิดจากการบีบตัวของห้องหัวใจเวนตริเคิลซ้ายลดลง 137 คน โดยข้อมูลทางคลินิกของผู้ป่วยทั้งหมด ได้แก่ ประวัติการเจ็บป่วย ผลตรวจคลื่นไฟฟ้าหัวใจ ผลตรวจหัวใจด้วยคลื่นเสียงสะท้อนความถี่สูง จะได้รับการประเมินจากแพทย์ผู้ซึ่งไม่ทราบผลการตรวจสวนหลอดเลือดโคโรนารี และจะแบ่งผู้ป่วยเป็น 2 กลุ่ม ข้อมูลทางคลินิกของผู้ป่วยกลุ่มที่ 1 จะนำมาใช้ในการสร้าง predictive model และนำ model นี้มาทดสอบความน่าเชื่อถือในผู้ป่วยกลุ่มที่ 2*

ผลการศึกษา: *Predictive score = (3 x การมีหรือไม่มีเบาหวาน (0 หรือ 1)) + จำนวน ECG leads ที่มี Q waves ที่ผิดปกติ – (5 x การมีหรือไม่มีลักษณะเฉพาะของหัวใจล้มเหลวที่ไม่ได้เกิดจากหลอดเลือดโคโรนารีตีบจากการตรวจด้วยคลื่นเสียงสะท้อนความถี่สูง (0 หรือ 1)) ค่าพื้นที่ใต้ receiver-operating characteristics (ROC) curve จากกลุ่มที่ 1 และ 2 เท่ากับ 0.94 และ 0.89 ตามลำดับ โดยค่าการทำนายที่มากกว่าหรือเท่ากับ 0 จะวินิจฉัยว่าผู้ป่วยมีสาเหตุหัวใจล้มเหลวจากหลอดเลือด โคโรนารีตีบ โดยมี sensitivity 100%, specificity 57%, positive predictive value 74% และ negative predictive value 100%*

สรุป: *Predictive model ซึ่งใช้ข้อมูลทางคลินิก ได้แก่ ประวัติการเจ็บป่วย คลื่นไฟฟ้าหัวใจ และการตรวจหัวใจด้วยคลื่นเสียงสะท้อนความถี่สูง สามารถใช้ในการแยกโรคหัวใจล้มเหลวจากสาเหตุอื่นออกจากโรคหลอดเลือด โคโรนารี และอาจช่วยลดการตรวจสวนหลอดเลือดโคโรนารีโดยไม่จำเป็นในผู้ป่วยกลุ่มนี้*