SCIENTIFIC LETTER

Prediction of mortality of patients with suspected heart failure by brain natriuretic peptide concentrations > 100 pg/ml: comparison of a clinical model with brain natriuretic peptide concentrations

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The test for heart failure with the highest sensitivity (94%) and specificity (70%) is clinical suspicion combined with a brain natriuretic peptide (BNP) concentration > 100 pg/ml.¹ BNP concentration is also an independent predictor of mortality in heart failure.² However, its value in predicting mortality compared with other traditional clinical findings and laboratory parameters has not been reported. This study compared the prediction of heart failure mortality within 30 days by BNP concentrations with a predictive model based on classic clinical findings recently described by Lee *et al.*³

METHODS

Nenagh Hospital is small general hospital in rural Ireland. From 29 September 2000 to 22 January 2002, 3692 patients were admitted to the hospital's 36-bed acute medical unit. BNP was measured in 646 (17.5%) of these patients. The selection of patients for BNP determination was based on the clinical estimate of the likely presence of heart failure. No specific instructions for measuring BNP were issued to the medical staff other than to obtain a concentration if heart failure was suspected. BNP concentrations were measured with the Triage BNP test (Biosite Diagnostics, La Jolla, California, USA). Four hundred and twenty-two patients had BNP concentrations > 100 pg/ml. Twelve patients were excluded because of incomplete data, leaving a final study population of 410 patients. Although the same age (mean 76.2 (SD 10.6) v 76.3 (11.2) years), this patient cohort had several significant differences from the derivation cohort of Lee *et al.*³ The present cohort had fewer women (40.5% v 50.5%) and significantly ($p < 0.05$) lower mean systolic blood pressure (136.5 (29.5) v 148.0 (33.0) mm Hg), pulse rate (90.0 (23.0) v 94.0 (25.0) beats/min), respiratory rate (21.2 (4.6) v 26.0 (7.0) breaths/min) and serum sodium concentration (136.7 (4.9) v 138.0 (5.0) mmol/l) but higher oxygen saturation (94.0 (5.7) v 92.0 (8.0)%) and urea concentration (10.9 (7.1) v 10.5 (6.9) mmol/l). Furthermore, fewer patients had diabetes (27.3% v 34.0%) or a previous myocardial infarction (13.2% v 36.3%), cerebrovascular disease (5.9% v 17.0%) or dementia (1.0% v 8.6%). The validation cohort of Lee *et al.*³ also had similar significant differences from the Nenagh patient cohort.

The ability of several readily available clinical variables, including all those used in the clinical model of Lee *et al.*³ to predict mortality was examined retrospectively. Receiver operating characteristic (ROC) curves were constructed and the area under these curves was compared according to the method of Hanley and McNeil.⁴ Local ethics approval was not required for this study.

RESULTS

Forty-one patients (10%) died within 30 days of hospital admission: 33 before and eight after discharge. These patients had a significantly higher mean (SD) BNP on admission than those discharged alive (806 (437) v 549 (410) pg/ml, $p < 0.0002$) and a significantly higher 30-day mortality risk scores according to the Lee *et al.*³ clinical model (106 (20) v 85 (23) points, $p < 0.0001$).

No patients with a Lee *et al.*³ model score of < 60 points died. The mortality of patients with a score between 61 and 90 points was 5.1%, between 91 and 120 points was 14.3%, and between 120 and 150 points was 28.9%. These rates did not differ significantly from those reported by Lee *et al.*³; indeed, they were almost identical (that is, the p value ranged from 0.44 to 0.94) (fig 1). Only 4.0% of patients with BNP concentrations < 200 pg/ml died, compared with 18.5% of patients with BNP concentrations > 1000 pg/ml. The area under the ROC curve did not differ significantly between the Lee *et al.*³ model score and BNP concentrations (72.2% v 67.4%).

Logistic regression showed that BNP concentration and the 30-day risk score predicted mortality independently of each other. Seven independent predictors of mortality were identified: the presence of cancer, being unwell before the current illness, a white cell count $> 12.5 \times 10^9/l$, a 30-day risk score > 116 points, being unable to stand up unaided, serum sodium ≤ 132 mmol/l and BNP concentration ≥ 700 pg/ml. A simple logistic model assigned one point to each independent predictor: 31.0% patients scored zero points, 33.2% one point, 21.7% two points, 9.5% three points and 4.6% four or more points. All three patients with a score of five or more points died. The area under the ROC curve for the seven-parameter logistic regression model was 84.5% compared with 72.2% for the Lee *et al.*³ model score ($p < 0.01$).

DISCUSSION

Outcomes of patients hospitalised for heart failure vary considerably. Differences in observed mortality may be the result of differences in heart failure severity as well as the treatment given. If the effectiveness of different treatment regimens is to be assessed, it is essential that predictive instruments of heart failure mortality be validated in different populations. ADHERE (Acute Decompensated Heart Failure National Registry), the largest database of hospitalised patients with heart failure, has identified eight parameters that predict in-hospital mortality.⁵ The model of Lee *et al.*³ comprised age, blood urea, systolic blood pressure, blood urea nitrogen and serum sodium concentration; in addition it included respiratory rate and the presence of

Figure 1 Mortality stratified by 30-day risk score categories. The 30-day mortality observed in the derivation and validation cohorts of Lee *et al*³ are compared with the 30-day mortality observed in the Nenagh Hospital cohort.

cerebrovascular disease, dementia, chronic obstructive lung disease, hepatic cirrhosis and cancer. However, it did not include diastolic blood pressure or breathlessness. Even though there were several significant differences between the present study's patients and those of Lee *et al*,³ their clinical model accurately predicted patient mortality. Furthermore, the predictive model and BNP concentration were found to predict mortality independently of each other.

In conclusion, the model of Lee *et al*³ accurately predicts mortality in a patient cohort significantly different from those they originally studied. Moreover, BNP concentration is a comparable and independent predictor of mortality. Although five additional independent predictors of mortality were identified in the particular patient cohort studied, these may not be relevant to other populations.

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REFERENCES

- Wang CS, Fitzgerald JM, Schulzer M, *et al*. Does this dyspneic patient in the emergency department have congestive heart failure? *JAMA* 2005;**294**:1944–56.
- Wang TJ, Larson MG, Levy D, *et al*. Plasma natriuretic peptide levels and the risk of cardiovascular events and death. *N Engl J Med* 2004;**350**:665–3.
- Lee DS, Austin PC, Rouleau JL, *et al*. Predicting mortality among patients hospitalized for heart failure: derivation and validation of a clinical model. *JAMA* 2003;**290**:2581–7.
- Hanley JA, McNeil BJ. A method of comparing the areas under receiver operating characteristic curves derived from the same cases. *Radiology* 1983;**148**:839–43.
- Abraham WT, Adams KF, Fonarow GC, the ADHERE Scientific Advisory Committee and Investigators, and the ADHERE Study Group, *et al*. In-hospital mortality in patients with acute decompensated heart failure requiring intravenous vasoactive medications. *J Am Coll Cardiol* 2005;**46**:57–64.

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