

EVALUATING A NURSE LED TRIAGE PROCESS IN TREATING PATIENTS WITH LEFT BUNDLE BRANCH BLOCK (LBBB) REFERRED FOR PRIMARY PERCUTANEOUS CORONARY INTERVENTION (PPCI)

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Background The Freeman Hospital (FRH) performs over 900 pPCI a year. Patients with suspected Acute Myocardial Infarction (AMI) are referred either by paramedics or networked hospitals for consideration of pPCI via a Telmed system, which is triaged by experienced CCU nurses. The pPCI Pathway can be activated in patients with LBBB suspected of having an AMI. However, there remains considerable variation in the clinical utility of new or presumed new LBBB as a ST-elevation myocardial infarction (STEMI)-equivalent ECG diagnostic criterion. The major discriminators the triage staff use in this population are ECG findings and symptoms suggestive of AMI. Our aim was to evaluate outcomes in patients with LBBB accepted to FRH or referred to local hospitals for assessment.

Methods Consecutive patients referred to FRH with LBBB and suspected AMI from 1st August 2009 to 30th November 2009 were analysed by recording: 1) Peak Troponin Level 2) Angiographic findings 3) Revascularisation rates.

Results 1069 patients were referred for consideration of pPCI. 177 (16.6%) of patients had new or presumed new LBBB. 33 (18.6%) patients were accepted by FRH and 144 patients (81.4%) were declined and referred to their local hospitals for assessment. Abstract 10 Table 1 Troponin levels in patients with LBBB referred for consideration of pPCI. 26.5% of patients with LBBB referred for consideration of pPCI had moderately to highly raised troponin. Of the 33 patients admitted to FRH, 13 underwent inpatient angiography and 9 patients had significant coronary disease (coronary stenosis 70%–100% in at least one coronary artery). Of those, 5 had PCI and 1 required urgent CABG. Only one patient had a 100% coronary occlusion believed to be an acute occlusion. 4 patients had unobstructed coronaries and were managed medically. Of the 132 patients declined for pPCI only 2 (1.5%) were referred back to FRH for PCI. Neither of these patients was found to have a 100% acute occlusion of a coronary artery.

Abstract 10 Table 1

	FRH Assessed	FRH Declined
Number of patients	33	144
Number analysed	33	132 (Biochemistry data not found for 12 patients)
Troponin levels		
Trop I<0.04 or Trop T<0.01 (Normal)	19 (57.6%)	84 (63.6%)
Trop I 0.04—0.1 or Trop T 0.01—0.1 (Mildly raised)	2 (6.1%)	25 (18.9%)
Trop I or Trop T 0.1—1.0 (Moderately raised)	1 (3.0%)	17 (12.9%)
Trop I or Trop T>1.0 (High)	11 (33.3%)	6 (4.5%)

Conclusion Revascularisation was performed in only 6/33 (18.2%) accepted for assessment and only 2/132 (1.5%) were referred back to the centre for PCI. The sensitivity of the triage process in detecting patients with LBBB requiring urgent revascularisation is 75% and the specificity is 83%. The sensitivity of detecting patients with an acutely occluded artery diagnosed at angiography is 100% with a specificity of 81%. In a high volume Heart Attack Centre a nurse led triage is effective at discriminating patients with LBBB requiring immediate coronary intervention.



COPEPTIN IMPROVES EARLY RISK STRATIFICATION BY GRACE SCORE IN NON ST-ELEVATION MYOCARDIAL INFARCTION; NT-PROBNP DOES NOT

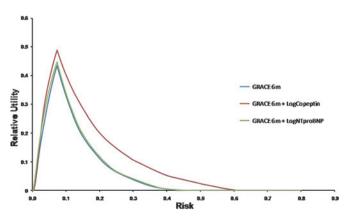
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Background Risk stratification is vital to the optimal management of patients with non ST-elevation myocardial infarction (NSTEMI) however, current tools are not fully discriminatory. Copeptin, the stable 39 amino acid C-terminal portion of pro-vasopressin, is a recognised prognostic marker in ST elevation myocardial infarction (STEMI) that is also useful to exclude MI as levels rise early after onset. Copeptin has not been evaluated in a NSTEMI population to date. **Aims** We hypothesised that copeptin is an independent predictor of mortality following NSTEMI and, in accordance with AHA criteria for the evaluation of novel biomarkers, assess whether copeptin adds

mortality following NSTEMI and, in accordance with AHA criteria for the evaluation of novel biomarkers, assess whether copeptin adds prognostic information to GRACE risk score (GRACE-RS). We use NT-proBNP for comparison. **Methods and Results** In this prospective observational study plasma copeptin and NT-proBNP was measured in 754 NSTEMI patients (519 men, median age 70±13 years) within 36 h of symptoms. The primary endpoint of all cause mortality at 6 months was reached by

primary endpoint of all-cause mortality at 6 months was reached by 56 (7.4%) patients. Median copeptin levels were 7.9 range 0.3 to 523.0 pmol/l and were significantly higher in those that reached the primary endpoint than the event free survivors; median (IQR), 32.0 (12.0-88.7) vs 7.2 (4.0-16.7) respectively p<0.001. Both copeptin and NT-proBNP were predictive of the primary endpoint on univariate Cox regression analysis (HR 5.98 p<0.0005 and HR 6.07 p<0.0005 respectively). On adjustment for baseline clinical and biochemical variables copeptin remained predictive (HR 3.03 p=0.009) but NTproBNP did not (p=0.70). Kaplan-Meier analysis revealed that supramedian levels of copeptin were associated with increased mortality (log rank 28.4 p<0.001). ROC curve c-statistics for GRACE-RS of 0.799 increased to 0.835 when combined with copeptin (0.785), when combined with NT-proBNP (0.730) increased to 0.802. Re-classification analysis shows that copeptin improves accuracy of risk stratification when combined with the GRACE-RS as determined by net reclassification improvement (NRI 13.3% p=0.008) whereas, NTproBNP does not (NRI -4.9% p=0.21). The relative utilities for logistic regression models using GRACE-RS alone, GRACE-RS + copeptin and GRACE-RS + NTproBNP as covariates are shown in Abstract 11 figure 1. The relevant region was the region to the right of the sample risk for 6 months mortality, 0.074. The relative utility for GRACE-RS + copeptin was consistently more than the relative utility for GRACE-RS + NTproBNP across a range of risks; for example at a risk threshold of 15% the additional utility of adding copeptin to the GRACE-RS was 0.097 compared to 0.009 for NTproBNP.



Abstract 11 Figure 1

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