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Does plasma copeptin level at admission predict final infarct size in ST-elevation myocardial infarction

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ABSTRACT

Background: Copeptin is a novel biomarker of potential diagnostic and prognostic value in patients with ST-elevation myocardial infarction (STEMI). This study was conducted to investigate the relationship between plasma copeptin levels at admission and final infarct size in STEMI patients.
Materials and methods: This observational study was conducted in Sher-i-Kashmir Institute of Medical sciences, Srinagar, for a period of 1 year. 60 patients with STEMI admitted within 24 h of symptom onset were included in the study. Plasma copeptin concentrations were determined by ELISA from blood samples drawn at the time of admission. Infarct size was estimated on cardiac MRI after 5–14 days of admission, in successfully reperfused patients. Correlations between plasma copeptin levels, infarct size and various clinico-hemodynamic variables were studied.
Results: Plasma copeptin concentrations showed a significant positive correlation with MRI determined infarct size ($r = 0.957$; $p \leq 0.0001$). Copeptin levels were significantly higher in patients with anterior wall infarction ($p \leq 0.0001$), longer symptom duration ($p = 0.018$), advanced Killip class ($p \leq 0.0001$), higher body mass index ($p = 0.019$) and extensive coronary artery disease ($p \leq 0.0001$). On multivariate analysis, copeptin levels at admission independently predicted final infarct size, irrespective of the clinico-hemodynamic profile of patients or mode of reperfusion ($p \leq 0.0001$). The only independent predictor of copeptin level was symptom duration ($p = 0.018$).
Conclusion: Copeptin level at admission predicts final infarct size in STEMI patients. Further evidence is however needed before implementation of this biomarker into routine clinical practice.
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1. Introduction

Copeptin, the C-terminal part of the vasopressin prohormone, is secreted in stoichiometric proportions with vasopressin during precursor processing [1,2]. Since vasopressin is unstable and cleared rapidly from the circulation, there is considerable ambiguity regarding the reliability and usefulness of measuring its plasma concentrations.[3] Copeptin, on the other hand, is stable and easily measurable, thus serving as a reliable surrogate of vasopressin secretion [4]. In recent years, copeptin has gained attention as a novel biomarker of predicting adverse prognosis in various acute illnesses like lower respiratory tract infections, sepsis, stroke, and acute pancreatitis [5–9]. Copeptin levels have also been demonstrated to rise early after the onset of acute myocardial infarction (AMI) [10]. Mechanisms believed to be responsible for triggering its release in AMI include acute endogenous stress, changes in hemodynamic and fluid status, and direct release from the infarcting myocardium [11–14]. Studies on temporal release kinetics of copeptin in AMI have demonstrated that unlike CK-MB and cardiac troponins, copeptin levels rise immediately after the onset of AMI and decline rapidly thereafter, reaching baseline values in about 24 h [12–14]. This distinct release pattern makes copeptin a potentially attractive biomarker for early diagnosis of AMI, particularly in ruling out AMI within first 3 h of symptom onset (period of "troponin blindness") [15–17].

Copeptin has also been demonstrated to be of utility in early risk stratification and prognostication of AMI patients. Studies have shown that copeptin is an independent predictor of mortality and major

Abbreviations: CK, creatine kinase; CK-MB, creatine kinase MB fraction; eGFR, estimated glomerular filtration rate; ELISA, enzyme linked immunosorbent assay; GRACE, Global Registry of Acute Coronary Events; MRI, magnetic resonance imaging.

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adverse cardiovascular events (MACE) in patients with ST elevation myocardial infarction (STEMI) as well as non ST elevation myocardial infarction (NSTEMI) [10,18,19]. Echocardiographic and cardiac MRI studies have revealed that increased copeptin concentrations, measured 2–5 days post-AMI, show a positive correlation with left ventricular (LV) dysfunction as well as late LV remodelling after the acute event [20–22]. It is well known that in patients with STEMI, infarct size is a stronger outcome predictor than LV function and volumes [23]. Cardiovascular magnetic resonance imaging (CMR) is the current gold standard for measurement of infarct size [24]. It has been hypothesized that larger infarct sizes may be associated with higher copeptin levels due to both direct cardiac release as well as neurohypophysis release in response to augmented hemodynamic stress [5,14]. This study was conducted with an aim of investigating the relationship between circulating plasma copeptin values at admission and final infarct size in patients with STEMI.

2. Patients and methods

2.1. Study design

This was a single centre observational study conducted in Sher-i-Kashmir Institute of Medical sciences, Soura, Srinagar, Jammu and Kashmir, for a period of 1 year.

2.2. Study population

This study included 60 patients with STEMI who were admitted in the Department of Cardiology, Sher-i-Kashmir Institute of Medical Sciences (SKIMS) between 2014 and 2015 and fulfilled the eligibility criteria (described below). After obtaining an informed consent, blood samples were drawn at the time of admission in serum tubes and centrifuged for 5–10 min at 2000 RPM within 0.5 h. Plasma was stored at −70 °C until analysis. Plasma copeptin concentrations were determined by Human copeptin (CPP) ELISA Kit (Bioassay Technology Laboratory, Shanghai, China), as per manufacturer protocol. Demographic and clinico-pathological characteristics of each patient were recorded in a specifically predesigned questionnaire. Patients were subsequently reperfused with either primary percutaneous coronary intervention (primary PCI) or pharmacoinvasive strategy. All patients who had successful reperfusion underwent CMR after 5 days to 2 weeks of admission for the evaluation of final infarct size.

Table 1
Demographic and clinical profile of the study population.

Variable	Subgroup	Frequency (N = 60)	Percentage (%)
Age (years)	<50	9	15.00
	51–60	21	35.00
	61–70	16	26.67
	>70	14	23.33
Sex	Male	49	81.67
	Female	11	18.33
Body mass index (kg/m^2 BSA)	<18	7	11.67
	18–25	26	43.33
	26–30	15	25.00
	>30	12	20.00
Smoking	Present	31	51.67
	Absent	29	48.33
Hypertension	Present	30	50.00
	Absent	30	50.00
Diabetes mellitus	Present	22	36.67
	Absent	38	63.33
Dyslipidemia	Present	16	26.67
	Absent	44	73.33
Killip class	I	32	53.33
	II	13	21.67
	III	15	25.00

Note: BSA – Body surface area; N – Number; % - Percentage.

2.3. Inclusion criteria

Patients with the diagnosis of STEMI (according to the third universal definition of myocardial infarction) [25] admitted in the Department of Cardiology, Sher-i-Kashmir Institute Of Medical Sciences (SKIMS), and who gave informed consent were included in the study.

2.4. Exclusion criteria

i. Patients with conditions other than STEMI that could cause increased copeptin levels; like renal dysfunction (eGFR < 30 ml/min), respiratory tract infection, stroke, sepsis or malignancy.
ii. Patients with contraindications to perform CMR.
iii. Patients with cardiogenic shock (Killip class IV).
iv. Patients in whom reperfusion therapy failed.
v. Patients who were referred for emergency coronary artery bypass grafting (CABG) in view of unfavourable coronary anatomy.

2.5. CMR protocol

CMR was performed on a 1.5 Tesla MRI scanner (Avanto, Siemens, Germany). Cine true FISP (Fast imaging with steady-state precession) images were obtained in standard short axis, four chamber and two chamber planes. Cine images were utilised for assessment of wall motion abnormality and ventricular function analysis Following intravenous gadolinium given in dose of 0.1 mmol/kg, dynamic images were obtained using FLASH (Fast low angle shot) sequence for seeing resting perfusion; and delayed enhancement images were obtained using phase sensitive inversion recovery images (PSIR FLASH), breathhold sequence and true FISP inversion recovery images. The single shot true FISP sequence was useful in patients, who were unable to hold their breath. For patients who had arrhythmias, prospective ECG gated sequences were obtained. The total area of infarction was obtained by semi-automated method using Argus software available on the dedicated MR work station.

2.6. Consent and ethical issues

An informed consent was obtained from each patient after explaining the study in detail. The study was cleared by the Institutional Ethics Committee.

2.7. Statistical analysis

Statistical analysis was performed by SPSS software package (version 20.0, SPSS Inc, Chicago, Illinois, USA). All continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were reported as frequency and percentages. Data was analysed using Pearson's correlation coefficient, stepwise multiple regression analysis, two-sample independent t-test and one way analysis of variance. A p value of <0.05 was considered to be statistically significant.

3. Results

3.1. Patient characteristics

The demographic and clinical profile of the study population is presented in Table 1. This study included 60 patients with mean age of 61.32 ± 11.33 years (range 38 to 91 years). Males and females constituted 81.67% and 18.33% of the study group respectively. 11.67% patients were underweight [Body Mass Index (BMI) < 18 kg/m^2 body surface area), and 20% patients were obese (BMI > 30 kg/m^2 body surface area). Risk factors included smoking (52%), hypertension (50%),

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