Research Article

"Role of Serum Relaxin Hormone in Early Diagnosis of Acute Myocardial Infarction."

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Abstract

Introduction: Acute myocardial infarction (AMI) is a major health problem worldwide with serious consequences in morbidity and mortality. For the diagnosis of AMI serum cardiac markers are used to support the diagnosis and among the many available biomarkers for the detection of AMI, cardiac troponin I is the preferred biomarker. **Aim of the work:** To measure the levels of serum relaxin hoinione in patients with recent onset of acute myocardial infarction and evaluate its sensitivity and specificity as a marker for early diagnosis of AMI. **Patients and Methods:** a prospective case-contol study at the department of clinical pathology on 55 subjects divided into 40 patients with AMI and 15 controls. Blood samples were collected from all patients and controls at time of admission to emergency room. Routine laboratory analysis including CBC, liver and kidney functions, lipid profile and random blood sugar were checked. Troponin I was measured at time of admission. A commercially available .LISA Kit was used to check serum relaxin levels at time of admission and after 6 hours. **Results:** We found that elevated serum Relaxin hormone may be a good biomarker for detection of early myocardial damage with great sensitivity and specificity.
Keywords: M': Acute Myocardial Infarction cTn: Cardiac Troponin RX: Relaxin hormone

Introduction

Acute myocardial infarction (AMI) is the world's leading cause of morbidity and mortality. An early and correct diagnosis may warrant immediate initiation of reperfasion therapy to potentially reduce the mortality rate (Philip A Tideman, et al., 2014).

Biomarkers, used to establish a diagnosis in patients with AMI, have emerged largely from targeted analyses of known myocardial proteins and become more and more important for diagnosis of AMI (Thygesen, et al., 2007). Current biomarkers such as creatine kinase-MB isoenzymes, cardiac myoglobin, and troponins have been widely applied in clinical. diagnosis. Among these, cardiac troponins are currently considered as the 'gold standard' for AMI diagnosis. However, the exploration of new biomarkers with high sensitivity and specificity in early diagnosis of AMI never stop. (Arang Samim, John A. Ambrose, 2015).

Relaxin is mainly known as a reproductive hormone which is produced by the corpus luteum and/or placenta in many species. There are varying effects of relaxin on the cervix, mammary glands, nipples, pubic symphysis and uterus of different species. (Sherwood, 2004). Relaxin is produced by the heart and as the specific heart receptors. It has been validated as a cardiotropic hormone. The significant function of relaxin in heart is to increase coronary blood flow and positive chrono-tropism and inotropism. It could also be able to counteract the pathophysiological mechanisms of ischemic heart disease. (Samuel, et al, 2003). Furthermore, relaxin could result in systemic vasodilation and extracellular body fluid expansion, and convert the cardio-circulatory apparatus to the needs of pregnancy. (Mohsin Sarwar, et al., 2016).

Aim of the work

The aim of our study was to measure the levels of serum relaxin hormone in patients with recent onset of acute myocardial infarction and to correlate the results with other laboratory markers as Troponin I.

Patients and Methods

We conducted a prospective case-contol study at the department of clinical pathology, faculty of medicine, Minia university. The study was conducted on 55 subjects divided into 40 patients and 15 controls recruited through the period from October 2016 to May 2017. The only criterion for eligibility in the patient group was the presence of acute ischaemic-type chest pain of recent onset. Patients with concomitant liver or kidney disease, infectious diseases and pregnant patients were excluded, because it is well known that these conditions may affect the level of relaxin hormone. We wanted to assume a baseline level within the adult range in all our patients and controls. For the same reason, we chose our controls of healthy volunteers to be in the same adult age range as the patients.

Blood samples were collected from all patients and controls at time of admission to emergency room. Routine laboratory analysis including CBC, liver and kidney functions, lipid profile and random blood sugar were checked. Troponin I was measured at time of admission by enzyme linked fluorescent assay using minividas analyser, Italy. A commercially available ELISA Kit (Glory science Co, Ltd) was used to check serum relaxin levels at time of admission and after 6 hours.

Results

Our study demonstrated the following : Serum relaxin levels after 6 hours (RX2) were significantly high in patients (ranged from 39 to 120 pg/ ml with a mean value±SD 77.8±20.3) compared to controls (ranged from 18 to 66 pg/ml with a mean value ± SD 38.4±16.1). This finding was consistent with Zhang study (2015).

- Serum relaxin levels after 6 hours were significantly high in patients (with a mean value ± SD 77.8±20.3) compared to relaxin level in patients at time of admission (mean value± SD 48.1±19.6). There was no statistically significant difference between cases and controls in relaxin levels at time of admission.

- It was found that serum relaxin levels at admission and after 6 hours were significantly higher in patients with STEMI than those with NSTEMI together with Troponin I levels with a P- value <0.001.

Table (I): Relaxin hormone levels in studied groups:

	Group I (n=40)	**Group II (n=15)**	**P Value**
RX1 (pg/m1)			
Range	(20-85)	(18-66)	0.094
Mean ± SD	48.1±19.6	38.4±16.1	
RX2(pg/m1)			
Range	(39-120)	(18-66)	< 0.001*
Mean ± SD	77.8±20.3	38.4±16.1	
P2	< 0.001*		

Table (II) : Correlation study of ST se2, mnt elevation to troponin I, RX1 and RX2 :

Group I	**ST segment elevation**	
	R	**P value**
Troponin I	0.857	<0.001*
RX1	0.571	<0.001*
RX2	0.540	<0.001*

Sensitivity and predictability:

Table (III): ROC curve analysis of RX1 and RX2 for prediction of MI in Group I

Variable	Optimal cutoff	AUC	P value	Sensitivity	Specificity	PPV	NPV	Accuracy
RX1	>38	0.647	0.078	67.5	73.33	87.1	45.8	69.1
RX2	>39	0.934	<0.001*	99	73.33	90.9	100	92.73

Discussion

In 2015, Zhang et al., designed a study to investigate the potential association between serum relaxin levels and the risk of AMI. They measured circulating relaxin levels in 80 patients (median age 62.3 years) who presented with first-time AMI 8 hours after the incident. The circulating relaxin levels in 80 healthy volunteers (median age 61.5 years) was also measured. Relaxin-2 was detected using enzyme immunoassay kits (Guangzhou, China) in both groups. Our study demonstrated that Serum relaxin levels after 6 hours (RX2) were significantly high in patients than controls. This finding was consistent with Zhang study (2015). There was no significant relation between relaxin and other biochemical parameters. This agreed with study of Zhang, et al., 2015. Similar to Zhang study (2015), we also failed to detect a causal relationship between increased serum relaxin levels and AlVII propably due to limited number of cases and the case-control design. There were minor differences in results of our study from Zhang study and it might be due to limited number of patients involved, different assays (kits), different timing of sample withdrawal, racial and genetic differences between patients study and Chinese people and different guidelines for MI diagnosis have been used.

Conclusion and future recommendations

From the study we concluded that Elevated serum Relaxin hormone may be a good biomarker for detection of early myocardial damage with great sensitivity and specificity. The elevation is more significant in patients with STEMI than those with non STEMI. we recommend Large scale cohort studies on a larger sample size to improve accuracy of results and studies comparing the sensitivity and specificity of relaxin with other new markers as: heart type fatty acid binding protein and glycogen phosphorylase BB.

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