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VALUE OF SERUM ASSAYS OF COPEPTIN VERSUS TROPONIN I AT ADMISSION FOR EARLY DIAGNOSIS OF ACUTE CORONARY SYNDROME

By

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ABSTRACT

Background: The major drawback of current troponin assays is sensitivity deficit at presentation due to delayed release of circulating levels. Therefore, acute myocardial infarction (AMI) exclusion requires prolonged period of monitoring for 6 to 9 hours and serial blood sampling, the grey zone for troponin elevation, which consequently leads to overcrowding in the emergency department and increases the associated cost every year. As a result, novel biomarkers are continuously in development. A particularly interesting observation is the response of circulating copeptin levels as a result of an acute myocardial infarction.

Objective: Comparing the value of serum copeptin assays to serum troponin I for early diagnosis of acute coronary syndrome.

Patients and methods: A prospective non- randomized study will be conducted over 45 patients in National heart institute (NHI). All of the patients were above 40 years old, presented to the emergency department (ED) within 12 hours of symptom onset of acute chest pain suggestive of acute coronary syndrome. After informed consent and permission from the local administrative authority, all patients were subjected to a thorough history taking, examination, 12- lead ECG monitoring, coronary angiography and echocardiographic assessment. In addition to the routine laboratory investigations. Cardiac troponin I and copeptin were assayed from venous blood samples obtained at admission, then 6 hours later. Based on clinical picture, ECG findings, serial troponin I assays, coronary angiography and echocardiographic findings all of the patients were divided into 3 groups:

1. ST-segment elevation of myocardial infarction (STEMI).
2. Non ST- segment elevation of myocardial infarction (NSTEMI) .
3. Unstable angina (UA).

Results: The admission values of copeptin was significantly different. At a cutoff value of 30.5 pmol/l, AMI can be diagnosed with 100 sensitivity, 100 specificity, 100 PPV, 100 NPV and accuracy 100%. The admission values for troponin I was significantly different. At cutoff value of 0.23 ng/ ml AMI can be diagnosed with 93.33 sensitivity, 100 specificity, 100 PPV, 88.2 NPV and accuracy 98%. The 6 hours-later copeptin values were also significantly different. AMI was diagnosed at cutoff value of 32.2 pmol/l with 100 sensitivity, 100 specificity, 100 PPV, 100 NPV and 100% accuracy. The 6 hours later values of troponin I were significantly different. At cutoff value of 0.23 ng/ml, AMI was diagnosed with 93.33 sensitivity, 100 specificity, 100 PPV, 88.2 NPV and accuracy 98%.

Conclusion: The current study provided evidence that measurement of serum copeptin was better than troponin I for early diagnosis of acute myocardial infarction, but could not evaluate the value of copeptin in UA diagnosis versus non -ischemic causes of chest pain.

Keywords: Copeptin, acute coronary syndrome, troponin I, NSTEMI.

INTRODUCTION

Chest pain was the second leading reason for emergency department (ED) visits in USA. A principal hospital discharge diagnosis of ischemic heart disease was assigned to 2.9 % of all ED visits. Even though not all chest pain patients will cause the treating physician to suspect AMI and even though AMI also needs to be suspected in many patients with symptoms other than chest pain. These numbers emphasize the clinical need for an early and fast rule-out of AMI. Under the current standard process of evaluating these patients, a minimum stay of between 3 and 9 hours is mandatory (**Smaradottir** et al., 2017).

Given that the vast number of these stays are ultimately unnecessary, personnel and space resources are wasted in the ED and patients are left to wait until the possible diagnosis of a “heart attack” has dissolved. In times of increasing ED crowding, which has been shown to negatively impact patient outcome, this process is basically unacceptable (**Mockel** et al., 2013)

The diagnosis of acute myocardial infarction is based on symptoms, signs and findings on the electrocardiogram, but in some patients these findings are non-diagnostic. In this patient population, the use of cardiac biomarker which indicates cardiac tissue necrosis - of which troponin and creatine kinase isoenzyme (CKMB) are preferred markers - play a pivotal role. These biomarkers allow for rule in of AMI within 3 hours after presentation in majority of patients and offer the opportunity to initiate an appropriate and evidence based treatment strategy (**Vargas** et al., 2016).

The major drawback of current troponin assays is sensitivity deficit at presentation due to delayed release of circulating levels. The vast majority of patients presenting to the emergency department with chest pain turn out not to have AMI. One quarter to one third of patients with AMI present without significant ECG changes indicative of ischemia. Therefore, ECG is of little help to rule out AMI (**Deedwania** et al., 2013).

As a result, and due to the lack of a completely early and accurate biomarker that effectively rule in and/or out AMI, novel biomarkers are continuously in development. A particularly interesting observation is the response of circulating copeptin levels as a result of an acute myocardial infarction (**Keller** et al., 2009 and **Pentighini,** 2015)**.**

The incremental value of copeptin to troponin was first elucidated by **Reichilin** et al. (2009a). **Keller** et al. (2009) confirmed these findings in a chest pain unit population. They reported the results of serial blood sampling in a subgroup of patients who presented within 2 hours after symptom onset to illustrate the complementary kinetics of troponin, the concentration of which increased 6 hours after admission, and copeptin, the concentration of which decreased during

the first 6 hours after a peak at presentation, in patients with AMI (**Keller** et al., 2009 and **Reichlin** et al., 2009a).

(CHOPIN), the largest multi-center trial of this type, confirm that the combination of a negative troponin and negative copeptin on presentation allows the rule out of AMI for 58% patients with >99.2% negative predictive value. In addition, copeptin value (>14 pmol/l) was able to detect greater numbers of patient with acute myocardial infarction and non-ST-elevation myocardial infarction at presentation when cardiac troponin was undetectable (**Maisel** et al., 2013).

PATIENTS AND METHODS

A prospective, non- randomized study was conducted over 45 patients in National heart institute (NHI) from Oct. 2014 to Oct. 2016. All of the patients were above 40 years old, presented to the ED within 12 hours of symptom onset of acute chest pain suggestive of acute coronary syndrome. After informed consents and permission from the local administrative authority, all patients were subjected to a thorough history taking, examination, 12- lead ECG monitoring, coronary angiography and echocardiographic assessment. In addition to the routine laboratory investigations, cardiac troponin I and copeptin were assayed from venous blood samples obtained at admission, then 6 hours later.

Based on clinical picture, ECG findings, serial troponin I assays, coronary angiography and echocardiographic findings all of the patients will be divided into 3 groups:

1. ST-segment elevation of myocardial infarction.
2. Non-ST- segment elevation of myocardial infarction.
3. Unstable angina.

Exclusion criteria:

1-Patients with heart failure, myocarditis and pericarditis 2-Sepsis and septic shock 3-Lower respiratory tract infection 4-Diabetes insipidus 5- Recent cerebrovascular stroke 6-Hyponatremia 7-Malignancy 8-Chronic renal failure

The concentration of copeptin was measured by the BRAHMS copeptin-us immune-luminometric assay on the KRYPTOR Compact Plus system (Thermo Fisher Scientific). The detection limit, as described by the manufacturer was signified as being 0.9 pmol/L, and the lowest concentration measurable with a coefficient of variation (CV) <10% has been reported <4 pmol/L. The direct measuring range was 0.9–500 pmol/L. Blood samples for copeptin were centrifuged, and plasma was frozen at −80°C. Copeptin measurement was performed at the end of the study recruitment, blinded to the final diagnosis. The concentration of troponin I was measured directly after withdrawal using the second-generation AxSYM Troponin-I ADV assay on the Abbott AxSym System. The analytical sensitivity of the assay was 0.02 ng/ml with a 10% coefficient of variation at 0.16 ng/ml. Two samples were used, i.e. on admission (A) and 6 - hours later(B).

Statistical analysis: Biomarkers were treated as categorical variables. Differences between the groups were examined with their means and SD in each group and, whenever we found a significant differences, ANOVA and

Tukey's tests were done as appropriate. Paired samples test was used to determine the paired differences between the first and second biomarker assays. The tests were done for samples (A) for troponin I and copeptin together, then for samples (B) together and then we compared the general copeptin and troponin I values. Receiver operating characteristic (ROC) curve was used to determine the sensitivity, specificity, negative predictive values (NPV) and positive predictive values (PPV) for the biomarkers, and optimal cutoff values for both troponin and copeptin. For all tests a p-value <0.05 was regarded as statistically significant.

RESULTS

- The admission values of copeptin were significantly different (P value < 0.001) in: STEMI versus UA, NSTEMI versus UA and in STEMI versus NSTEMI (Table 1).

Table (1): Ranges of copeptin (pmol/l) values in patient groups.

Copeptin \ Groups		STEMI	NSTEMI	UA
A	**Range**	43.2 - 55.8	31.4 - 38.1	21.7 - 30.5
	Mean ±SD	48.704 ± 3.303	35.071 ± 2.386	25.467 ± 2.276
B	**Range**	47.8 - 62.4	34.9 - 44	24.1 - 32.2
	Mean ±SD	53.361 ± 3.740	39.100 ± 3.272	27.180 ± 2.526
Paired Differences		-4.657 ± 1.766	-4.029 ± 1.444	-1.713 ± 1.558
P-value		<0.001*	<0.001*	0.001*

- Paired sample test showed significant increase in copeptin level after 6hs in all study groups. A=value at admission, B =value 6h later, C=copeptin, G=groups.

Tukey's test showed significant difference in copeptin in STEMI versus UA, in STEMI versus NSTEMI and in STEMI versus NSTEMI, either at admission or 6h later S=STEMI, N=NSTEMI, U=UA, A= copeptin at admission, B= copeptin 6h later (Table 2).

ANOVA test showed significant difference among the study groups in copeptin either at admission or 6h later.

Table(2): ANOVA and TUKEY's evaluating copeptin values in patient groups.

COPEPTIN	ANOVA		TUKEY'S Test		
	F	**P-value**	**S&N**	**S&U**	**N&U**
A	281.66	<0.001*	<0.001*	<0.001*	<0.001*
B	248.217	<0.001*	<0.001*	<0.001*	<0.001*

- At a cutoff value of 30.5 pmol/l, AMI was diagnosed with 100 sensitivity, 100 specificity, 100 PPV, 100 NPV and accuracy 100% (Table 3).

Table (3): Values of receiver operator curve (ROC) of copeptin at admission: cutoff, sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy.

ROC curve					
Cutoff	**Sens.**	**Spec.**	**PPV**	**NPV**	**Accuracy**
>30.5	100	100	100	100	100%

- At a copeptin cutoff value of 32.2 pmol/l, AMI was diagnosed 6 hs after admission with sensitivity 100, specificity 100, PPV 100, NPV 100 and accuracy of 100% (Table 4).

Table (4): Values of receiver operator characteristic (ROC) of copeptin 6 hours later: cutoff, sensitivity, specificity, (PPV), (NPV), and accuracy.

ROC curve					
Cutoff	**Sens.**	**Spec.**	**PPV**	**NPV**	**Accuracy**
>32.2	100	100	100	100	100%

- The admission values for troponin I were significantly different (P value < 0.001) in STEMI versus UA and also, in NSTEMI versus UA (Table 6).

Table (5): Ranges of troponin values (ng/ml) at admission (A) or 6h later (B).
Paired sample test showed significant increase in troponin I level after 6hs in STEMI and NSTEMI.

Groups / Troiponin		STEMI			NSTEMI			UA		
A	**Range**	0.21	-	1.2	0.1	-	0.7	0.1	-	0.23
	Mean ±SD	0.613	±	0.211	0.533	±	0.200	0.109	±	0.034
B	**Range**	0.21	-	1.5	0.1	-	0.92	0.1	-	0.23
	Mean ±SD	0.773	±	0.289	0.701	±	0.273	0.109	±	0.034
Paired Differences		-0.160	±	0.114	-0.169	±	0.087			
P-value		<0.001*			0.002*					

- ANOVA test showed significant difference among the study groups in troponin I level either at admission or 6h later. Tukey's test showed significant difference in troponin I level in STEMI versus UA and in NSTEMI versus UA, either at admission or 6h later, but no significant difference in STEMI versus NSTEMI (Table 6).

Table (6): ANOVA and Tukey's tests evaluating troponin I values: (A)=at admission, (B)=6h later, (T)=Troponin I, (G)=Groups.

Troponin I	ANOVA		TUKEY'S Test		
	F	P-value	S&N	S&U	N&U
A	38.534	<0.001*	0.530	<0.001*	<0.001*
B	37.52	<0.001*	0.762	<0.001*	<0.001*

- At admission at a cutoff value of 0.23 ng/ml AMI was diagnosed with 93.33 sensitivity, 100 specificity, 100 PPV, 88.2 NPV and accuracy 98% (Table 7).

Table (7): ROC of troponin I value at admission: cutoff, sensitivity, specificity, (PPV), (NPV), and accuracy.

ROC curve					
Cutoff	**Sens.**	**Spec.**	**PPV**	**NPV**	**Accuracy**
>0.23	93.33	100	100	88.2	98%

- The 6 hours later values of troponin I values were significantly different (P <0.001) in STEMI versus UA and in NSTEMI versus UA (table 6). At cutoff value of 0.23 ng/ml AMI was diagnosed with 93.33 sensitivity, 100 specificity, 100 PPV, 88.2 NPV and accuracy 98% (Table 8).

Table (8): Cutoff value, PPV, NPV, sensitivity, specificity and accuracy of troponin I value 6 hours later.

ROC curve					
Cutoff	**Sens.**	**Spec.**	**PPV**	**NPV**	**Accuracy**
>0.23	93.33	100	100	88.2	98%

- The overall admission and 6 hours later values for copeptin were also significantly different, and AMI was diagnosed at cutoff value of 30.49 pmol/l with 100 sensitivity, 100 specificity, 100 PPV, 100 NPV and 100% accuracy.

- The overall admission and 6 hours later values for troponin I were also significantly different as before, and at a cutoff value of 0.22 ng/ml AMI was diagnosed with 93.33 sensitivity, 100 specificity, 100 PPV, 88.2 NPV and accuracy 98%.
- This study revealed that copeptin alone was better than troponin I for diagnosis of AMI, and this was in accordance with the studies done before. The optimal cutoff value of copeptin for AMI diagnosis in our study, being 30.49 pmol/l.

DISCUSSION

This study revealed that copeptin alone was better than troponin I for diagnosis of AMI, and this was in accordance with the studies done before. The optimal cutoff value of copeptin for AMI diagnosis in our study, being 30.49 pmol/l, is different from other studies that used cutoff values of (9 pmol/L, 14 pmol/L, 20 pmol/L, and 24 pmol/L). **Lipinski** et al. (2014) demonstrated that Copeptin shows only low specificity for myocardial damage. Accordingly, the positive predictive value (PPV) for AMI of copeptin alone is thought to be unacceptably low. The first studies investigating the diagnostic value of copeptin for AMI showed a very low PPV for AMI. For instance, **Reichlin** et al. (2009a) calculated the PPV of copeptin for AMI diagnosis for different cut-off concentrations. In their study, the PPV of copeptin ranged between 34.9% and 57.9%. (**Lipinski** et al., 2014 and **Reichlin** et al., 2009a).

Copeptin is a biomarker of AMI, not the acute coronary syndrome. **Reichlin** et al. (2009b) reported no significant difference in copeptin concentration between UA patients and patients with non- ischemic causes of chest pain and attributes this to that UA does not cause sufficient endogenous stress for vasopressin release. On the other hand, **Johan** et al. 2013 reported that copeptin values are higher in the UA patients compared to the non-ACS group, but this finding was statistically insignificant. The current study revealed that copeptin values in UA were significantly different from both STEMI and NSTEMI, but evaluation of copeptin values in UA compared to non- ACS was not possible in our study as all of our patients have ACS, and no patients of chest pain due to non- ischemic causes. Other studies should be warranted to highlight this issue (**Reichlin** et al., 2009b and **Johan** et al., 2013).

The current study showed a significant difference between copeptin values in STEMI and NSTEMI in comparison to troponin I that showed an insignificant difference between them. These results were in consistence with the other studies (**Vargas** et al. 2016).

Unlike the other studies, in the current study, copeptin was tending to have a statistically significant increase from admission to 6 hours later, and this occurred in all patient groups including UA. The other studies reported that maximal serum values were one hour after the onset of chest pain then decreased gradually to reach a plateau one to two days later. The pattern of release was more questionable in UA, since the increase in copeptin values in UA is an issue of controversy (**Ananth** et al., 2016).

In consistence with the other studies, troponin I was tending to have a statistically significant increase from admission to the 6 hours later values, and this occurred in both STEMI and NSTEMI patient groups **(Shortt** et al., 2017).

Our study showed that copeptin values increased directly with troponin I, which in turn increased with increasing mass of infarction, and this was in accordance with other studies (**Kavsac**, 2017).

Troponin I was significantly higher in late presenter (>4h) of chest pain than early presenters (<4h), either at admission or 6 hour later. Results were in consistence with the other studies (**Kavsac** , 2017).

Unlike the other studies, this study revealed no significant difference in copeptin levels between early and late presenters of chest pain neither at admission or at 6 hours later. This implied that copeptin was valuable in late as well as early presenters of chest pain. This results made the pattern of copeptin release more questionable, and more studies using more frequent sampling times were warranted to adequately describe its pattern of release. An important factor should be considered in our study that many patients were not accurately able to determine the onset of chest pain. Secondly, we had only a limited number of early presenters compared to late presenters (**Vargas** et al. 2016).

Limitations

1. The study was limited by the small sample size, which was further decreased by subgrouping, an effect that was more obvious in NSTEMI patient group. So, the results were only preliminary that need large sample size studies.
2. The troponin I assay used in this study was a second-generation troponin assay. So, evaluation of copeptin in comparison to highly sensitive troponin was not possible.
3. Only two samples were taken, i.e. on admission and 6 hours later, while frequent the sampling was needed to delineate the pattern of copeptin release after AMI.
4. The effect of thrombolytic therapy on copeptin and troponin I values was not examined, since most of our patient received thrombolysis before referral to NHI or before PCI was done.
5. Being a single center study, our research may be limited by the implausible sample size, lack of blinding, unequal allocation of resources or limited external validity.
6. Management studies were needed to evaluate the safety and effectiveness of this strategy under routine conditions and in a larger number of patients.
7. Due to ethical considerations, copeptin versus troponin values could not be examined neither in stable angina, non-ischemic chest pain nor control cases, as coronary angiography - an essential part of the study to confirm the diagnosis of AMI – would be declined by the aforementioned patient groups after explanation of benefit/ risk considerations. Such an effect is needed to be examined in further studies.

CONCLUSIONS

The current study provided an evidence that measurement of serum copeptin was better than troponin I for early diagnosis of AMI but could not evaluate the value of copeptin in UA diagnosis versus neither stable coronary artery disease nor non-ischemic causes of chest pain.

REFERENCES

1. ***Ananth V, Beig JR, Tramboo NA, Rasool R, Choh NA, Bashir S, Rather HA and Lone AA (2016)****: Does plasma copeptin level at admission predict final infarct size in ST-elevation myocardial infarction. Int J Cardiol., 15:219:326-30.*
2. ***Deedwania PC (2013):*** *The need for a cost-effective strategy to detect ambulatory silent ischemia. Am J Cardiol, 74:1061-1070.*
3. ***Folli C, Consonni D, Spessot M and Salvini L (2013):*** *Diagnostic role of copeptin in patients presenting with chest pain in the emergency room. Eur J Intern Med., 24(2):189-93.*
4. ***Johan T, Catharina B, David E and Bertil O (2013)****: The combination of high sensitivity troponin T and copeptin facilitates early rule-out of ACS: a prospective observational study, BMC Cardiovascular Disorders, 13:42*
5. ***Kavsak PA (2017):*** *Rule-In and Rule-Out of Myocardial Infarction Using Cardiac Troponin and Glycemic Biomarkers in Patients with Symptoms Suggestive of Acute Coronary Syndrome. Clin Chem., 63(1):403-414.*
6. ***Keller T, Zeller T, Peetz D and Tzikas S (2009):*** *Sensitive troponin I assay in early diagnosis of acute myocardial infarction. N Engl J Med., 361(9):868–77.*
7. ***Lipinski MJ, Esc´arcega RO and D'Ascenzo F (2014)****: A systematic review and collaborative meta-analysis to determine the incremental value of copeptin for rapid rule-out of acute myocardial infarction. The American Journal of Cardiology, 113, 9: 1581–1591.*
8. ***Maisel A1, Mueller C, Neath SX, Christenson RH and Morgenthaler NG (2013):*** *Copeptin helps in the early detection of patients with acute myocardial infarction: primary results of the CHOPIN trial (Copeptin helps in the early detection Of Patients with acute myocardial infarction). J Am Coll Cardiol., 62: 150-60*
9. ***Mockel M, Searle J, Muller R, Slagman A, Storchmann H and Oestereich P (2013):*** *Chief complaints in medical emergencies: do they relate to underlying disease and outcome? The Charité Emergency Medicine Study (CHARITEM). Eur J Emerg Med., 20(2):103–8.*
10. ***Pentighini M (2015):*** *Recommendations on the use of biochemical markers in Acute Coronary Syndrome: IFCC proposals, The Journal of International Federation of Clinical Chemistry and Laboratory Medicine. 108(20):2543-9.*
11. ***Reichlin T, Hochholzer W and Bassetti S (2009a):*** *Early diagnosis of myocardial infarction with sensitive cardiac troponin assays. N Engl J Med., 361(9):858–867.*
12. ***Reichlin T, Hochholzer W and Stelzig C (2009b):*** *Incremental value of copeptin for rapid rule out of acute myocardial infarction, Journal of the American College of Cardiology, 54, 1:60–68.*
13. ***Shortt C, Clayton N, Sherbino J, Whitlock R, Pare G, Hill SA, McQueen M, Mehta SR, Devereaux PJ Worster A and Kavsak PA (2017):*** *Rule-In and Rule-Out of Myocardial Infarction Using Cardiac Troponin and Glycemic Biomarkers in Patients with Symptoms Suggestive of Acute Coronary Syndrome. Clin Chem., 63(1):403-414.*
14. ***Smaradottir MI, Ritsinger V, Gyberg V, Norhammar A, N?sman P and Mellbin LG (2017):*** *Copeptin in patients with acute myocardial infarction and newly detected glucose abnormalities - A marker of increased stress susceptibility? A report from the Glucose in Acute Myocardial Infarction cohort. Diab Vasc Dis Res., 40(3):200-5.*
15. ***Vargas KG, Kassem M, Mueller C, Wojta J and Huber K (2016):*** *Copeptin for the early rule-out of non-ST-elevation myocardia infarction. Int J Cardiol., 15;223:797-804.*

أهمية قياس كوببتن بالمصل مقارنة بتروبونين آي للتشخيص المبكر لمتلازمة الشريان التاجي الحاد

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خلفية البحث: من أهم عيوب استخدام تروبونين آى في تشخيص الاحتشاء القلبي هو تأخر ظهوره أحيانا بعد ظهور أعراض الاحتشاء، مما يؤدي إلى عدم القدرة علي التشخيص في الساعات الأولي من ظهور الاعراض مما يؤدي إلي ضرورة متابعة المريض بأقسام الطوارئ من ٦ - ٩ ساعات يحتاج المريض خلالها إلي أخذ عينات من الدم بشكل متكرر، مما يؤدي إلي ازدحام تلك الاقسام و يزيد من التكلفة السنوية، لذلك فالبحث مستمر عن مواد جديدة و من بين هذه المواد كوببتن.
الهدف من البحث: مقارنة أهمية كوببتن المصل بتروبونين آى للتشخيص المبكر لمتلازمة الشريان التاجي الحاد
المرضي و طرق البحث: أجريت دراسة استباقية غير عشوائية علي 45 مريض بمعهد القلب القومي من أكتوبر ٢٠١٤ إلي أكتوبر ٢٠١٦ ، و قد حظيت هذه الدراسة بالموافقة عليها من قبل لجنة أخلاق البحث العلمي بكلية الطب جامعة الازهر ، وقد أجريت الدراسة علي المرضي الذين تتجاوز أعمارهم ٤٠ سنة ، و يعانون من ألم صدري حاد يوحي بنقص التروية الدموية القلبية ، خلال ١٢ ساعة من ظهور الالم ، وبعد موافقة المريض وموافقة الادارة المسئولة يؤخذ التاريخ المرضي للحالات متبوعا بالفحص الإكلينيكي وتخطيط القلب الكهربائي و تصوير القلب بالموجات الفوق صوتية و تصوير الشرايين التاجية ، و تم سحب عينات دم وريدي لعمل التحاليل الاساسية بالإضافة إلي تروبونين آى و كوببتن علي مرتين ؛ مرة عند وصول المريض و أخري بعدها بست ساعات.
وقد قسم المرضي إلى 3 مجموعات
١- مرضي الإحتشاء القلبي المصحوب بارتفاع في القطعة إس تي
٢- مرضي الإحتشاء القلبي الغير مصحوب بارتفاع في القطعة إس تي
٣- مرضي الذبحة الصدرية الغير مستقرة
النتائج: في عينات المصل الأولي: اختلفت نسبة كوببتن بين مجموعات المرضي بشكل ملحوظ إحصائيا، وعندما وصلت النسبة بالمصل إلى ٣٠,٥ بيكومول/لتر تم تشخيص الاحتشاء بحساسية ١٠٠ واختصاصية ١٠٠ ودلالة إيجابية ١٠٠ ودلالة سلبية ١٠٠ و دقة ١٠٠%، و كانت نسبة تروبونين في عينات المصل الأولي مختلفة أيضا بين مجموعات المرضي بشكل ملحوظ إحصائيا، و عندما كانت نسبة الأخير بالمصل ٠,٢٣ نانوجرام/مليلتر تم تشخيص الإحتشاء بحساسية ٩٣,٣٣ و اختصاصية ١٠٠ و دلالة إيجابية ١٠٠ ودلالة سلبية ٨٨,٢ و دقة ٩٨%. **أما في عينات المصل الثانية:** فقد اختلفت أيضا نسبة كوببتن بين مجموعات المرضي بشكل ملحوظ إحصائيا، وعندما وصلت النسبة بالمصل إلى ٣٢,٢ بيكومول/لتر تم تشخيص الإحتشاء بحساسية ١٠٠ واختصاصية ١٠٠ ودلالة إيجابية ١٠٠ ودلالة سلبية ١٠٠ ودقة ١٠٠%، وكانت نسبة تروبونين في عينات المصل الثانية مختلفة أيضا بين مجموعات المرضي بشكل ملحوظ إحصائيا، وعندما كانت نسبة الأخير بالمصل ٠,٢٣ نانوجرام/مليلتر تم تشخيص الإحتشاء بحساسية ٩٣,٣٣ واختصاصية ١٠٠ ودلالة إيجابية ١٠٠ ودلالة سلبية ٨٨,٢ ودقة ٩٨%.
الاستنتاج: قياس كوببتن المصل أفضل من قياس تروبونين آى للتشخيص المبكر للإحتشاء ولكن لم نتمكن من دراسة قيمة كوببتن لتشخيص الذبحة الصدرية الغير مستقرة وتمييزها عن أسباب الألم الصدري الغير ناجم عن نقص التروية الدموية للقلب.