TURKISH JOURNAL OF MEDICAL AND HEALT SCIENCES
https://journals.academicianstudies.com/TTSB
E-ISSN; 3062-1720
https://doi.org/10.71284/tjmhs.2025232

KALP YETMEZLİĞİ HASTALARINDA KOPEPTİN VE OKSİDATİF STRES BELİRTEÇLERİNİN BELİRLENMESİ

DETERMINATION OF COPEPTIN AND OXIDATIVE STRESS MARKERS IN HEART FAILURE PATIENTS

Sura Salim MOHAMMED[(1)] Fahad K. Y. Al-DULAIMI[(2)] Müslüm KUZU[(3)]

[(1)] Department of Chemistry, Institute of GraduatePrograms, Karabuk University, Karabuk, 78050, Türkiye, suraalsamaraia@gmail.com, ORCID 0000-0002-6117-6371

[(2)] Department of Plant Production Technical,Faculty of Technical Agricultural, Northern Technical University, Mosul, 41001, Iraq, fahadbiologymycology@ntu.edu.iq, ORCID: 0000-0002-5958-3496

[(3)] Department of Nutrition and Dietetics, Faculty of Health Sciences, Karabuk University, Karabuk, 78050, Türkiye, muslumkuzu@karabuk.edu.tr, ORCID: 0000-0002-1375-7673

Article Info

Article Type
Research Article

Dates
Received: 06.11.2025
Accepted: 09.12.2025
Published: 30.12.2025

Keywords
Copeptin
Oxidative Stress
Antioxidants
Liver Enzyme

Abstract

This study aimed to determine the increase in copeptin levels in patients with heart failure and to compare them with those in a control group. Additionally, liver enzyme levels, renal function markers, and antioxidant levels were compared with those in a control group. Finally, the relationship between copeptin and all biochemical variables studied was analyzed. Copeptin and troponin variables were measured using an ELISA device, while liver enzymes and renal variables (AST, ALT, Urea, and Creatinine) were assessed using an Otto device. Antioxidants were measured colorimetrically using a spectrophotometer. The study included 100 participants, divided into two groups: a healthy group (n = 50) and a heart failure patient group (n = 50). Copeptin and troponin values were found to be higher in patients with heart failure compared to the control group. Additionally, there were significant increases in renal markers and liver enzymes in the patient group compared to the control group. In patients with heart failure, levels of GSH, GPx, and SOD decreased compared to the control group, while MDA levels increased. Copeptin levels were significantly higher in heart failure patients than in the control group, making it a potential indicator of heart-related problems.

Keywords: Copeptin, Oxidative Stress, Antioxidants, Liver Enzyme

Anahtar Kelimeler
Kopeptin
Oksidatif Stres
Antioksidantlar
Karaciğer Enzimleri

Özet

Bu çalışmanın amacı kalp yetmezliği hastalarındaki kopeptin seviyelerindeki artışın tespiti, kalp yetmezliği ile ilişkili troponin seviyelerini değerlendirerek kontrol grubu ile karşılaştırmaktır. Ayrıca, karaciğer enzim seviyeleri, böbrek fonksiyon belirteçleri ve antioksidan seviyeleri kontrol grubu ile karşılaştırılmıştır. Son olarak, kopeptin ile çalışılan tüm biyokimyasal değişkenler arasındaki ilişki analiz edildi. Kopeptin ve troponin değişkenleri ELISA cihazı kullanılarak ölçüldü, karaciğer enzimleri ve böbrek değişkenleri (AST, ALT, Üre ve Kreatinin) Otto cihazı kullanılarak değerlendirildi, antioksidanlar ise spektrofotometre cihazı kullanılarak kolorimetrik yöntemle ölçüldü. Çalışmaya sağlıklı grup (n=50) ve kalp yetmezliği olan hasta grup (n=50) olmak üzere iki gruba ayrılmış 100 katılımcı dahil edildi. Kopeptin ve troponin değerlerinin kalp yetmezliği hastalarında kontrol grubuna kıyasla daha yüksek olduğu görüldü. Ayrıca, hasta grubunda kontrol grubuna kıyasla böbrek belirteçlerinde ve karaciğer enzimlerinde anlamlı bir artış bulunmaktadır. Kalp yetmezliği hastalarında GSH, GPx ve SOD değerleri kontrol grubuna göre azalırken, MDA düzeyleri arttı. Kalp yetmezliği hastalarında kopeptin düzeyleri kontrol grubuna göre anlamlı derecede yüksekti ve bu da onu kalp ile ilgili sorunlar için kritik bir gösterge haline getirdi.

Anahtar Kelimeler: Kopeptin, Oksidatif Stres, Antioksidantlar, Karaciğer Enzimleri

TURKISH JOURNAL OF MEDICAL AND HEALT SCIENCES
https://journals.academicianstudies.com/TTSB
E-ISSN; 3062-1720
https://doi.org/10.71284/tjmhs.2025232

1. Introduction

Heart diseases are a significant global health concern, responsible for a staggering 16% of all deaths worldwide. Disturbingly, the number of deaths attributed to heart diseases has been on the rise since 2000, with an increase of over two million fatalities, reaching nearly nine million deaths in 2019 (Gladysheva & Sullivan, 2022). Chronic heart failure is a prevalent condition within the spectrum of heart diseases. It is primarily caused by coronary heart disease, with a particular emphasis on myocardial infarction and myocardial ischemia (Kleinman et al., 2018). Myocardial infarction poses a life-threatening situation that occurs when one of the coronary arteries becomes obstructed, resulting in damage or death to a portion of the heart muscle (Anderson & Morrow, 2017). Several risk factors contribute to the development of myocardial infarction, including high blood pressure, smoking, obesity, elevated blood cholesterol levels, and lack of exercise (Katz & Ness, 2015). Although determining arginine vasopressin hormone levels is essential in diagnosing and prognosing some diseases, accurate results cannot be obtained since it is mainly bound to platelets in the serum and has a short half-life and low molecular stability.

Since copeptin and this hormone are provided in equimolar amounts, copeptin level can also determine the arginine vasopressin hormone level (Abdelmageed & Güzelgül, 2023). Although the physiological function of copeptin is not fully understood, it has been reported that it is subject to the same changes as AVP in osmotic, hemodynamic, and stress situations, and may serve as a marker in the diagnosis and prognosis of diseases associated with AVP dysregulation (Balanescu et al., 2011). Some studies have reported that chronic heart failure patients have worse long-term prognoses compared to those with lower levels of copeptin (Morgenthaler et al., 2008). It has also been stated that copeptin levels may be used to predict mortality in patients with critical heart failure, acute stroke, coronary artery disease, and lower respiratory tract infection (Pozsonyi et al., 2015). This glycopeptide is derived from the same gene as vasopressin and is located on chromosome 20. Copeptin is formed in reverse order with the oxytocin gene and originates from the third exon of neurophysin-2 mRNA. It includes the last 17 amino acids at the carboxylate end.

Copeptin levels change in serum due to alterations in blood osmosis as well as ischemia or heart failure. It is released from the hypothalamus and increases in response to myocardial ischemia and heart failure (Fenske et al., 2018a). As an alternative biomarker for regulating blood vessels and maintaining blood volume, Copeptin exhibits a short half-life but high stability in circulation compared to vasopressin (Fenske et al., 2018b). Oxidative stress is an imbalance between reactive oxygen species (ROS) and antioxidants within the body. This imbalance manifests as an excessive amount of ROS compared to antioxidants and has been associated with various health conditions such as diabetes, cardiovascular disease, and other illnesses (Hayes et al., 2020). Disruption of calcium balance, defects in signal transmission, structural disorders in cells, increased cardiac fibrosis, and loss of cardiomyocytes indicate that oxidative stress is the primary mechanism underlying the development of heart failure (Kumar et al., 2023). Detection of renal function disorders in patients with heart failure is essential in predicting the adverse events that may occur later. Although the mechanism of renal failure caused by heart failure is not fully known, it is estimated that excessive neurohormonal activation may occur (Savarese et al., 2022). In this study, which focused on patients diagnosed with heart failure, copeptin levels and troponin were measured. Additionally, markers indicative of oxidative stress and damage to the kidneys and liver were assessed.

2. Material and Metods

2.1. Sample Collection

This study was conducted in a hospital in Salah al-Din, Iraq. Fifty of the people subject to the study had heart failure and were in the intensive care unit, and 50 healthy others were hospitalised during the period from February to April 2023. As the affected individuals underwent an ECG examination (electrocardiography), informed consent was obtained from each participant prior to the procedure. Information was collected about them regarding age (in years), gender, and medications for other diseases. Venous blood samples were collected from the participants into plain tubes without anticoagulants. The blood samples were allowed to clot at room temperature for a specified period. Serum was separated by centrifugation at 8000 rpm for 15 minutes. The obtained serum samples were stored at −20 °C until biochemical analyses were performed.

2.2. Measurements of Copeptin and Troponin

Copeptin and troponin levels were determined using enzyme-linked immunosorbent assay (ELISA) methods in accordance with the manufacturers' kit protocols. The copeptin and troponin kits were commercially obtained from Sunlong Biotech Co., China (catalog numbers SL1950Hu and QS0411Hu, respectively). The ELISA device used for these measurements was the Sunlong BioELISA from the U.S.A. This widely recognized and reliable method allowed for the accurate quantification of copeptin and troponin levels in the analysed samples.

2.3. Measurements of Oxidative Stress Parameter

The activity levels of antioxidants, including Glutathione (GSH), were estimated by the researchers using a modified method based on Elman's reagent, which contains 5,5-dithiobis-2-nitrobenzoic acid (DTNB). This reagent reacts with glutathione to produce a yellow compound with an absorbance reading at 412 nm (Katrukha et al., 1997). Malondialdehyde (MDA) Using the TBA reaction technique, measured the content of malondialdehyde (MDA), a Using the TBA reaction technique, we measured the content of malondialdehyde (MDA), a primary product of lipid peroxidation, by assessing its interaction with TBA The resulting colored product's absorbance intensity was measured at 532 nm (Al-Helaly & Ahmed, 2014), and Glutathione Peroxidase (GPx) as The mixture of Phenol, 4-Amino antipyrine, and 0.0017 M hydrogen peroxide was incubated at 37 °C for 3–4 hours. The reaction started with the addition of 0.1 ml of

TURKISH JOURNAL OF MEDICAL AND HEALT SCIENCES
https://journals.academicianstudies.com/TTSB
E-ISSN; 3062-1720
https://doi.org/10.71284/tjmhs.2025232

serum. The first absorbance was measured immediately at a wavelength of 510 nm, and the second absorbance was measured after 5 minutes, using the method provided in the examination kit (Zhang et al., 2016). The SOD activity was estimated using a modified method with photochemical NBT. Sodium cyanide inhibited the peroxidase enzyme. This method indirectly measures SOD activity by observing changes in optical density resulting from formazan formation during NBT reduction. A decrease in formazan's light density indicates increased SOD enzyme activity. These were determined through the optical absorption method. These measurements were conducted using a Spectrophotometer (Genesys, U.S.A.) following standardized measurement procedures.

2.4. Measurement of Liver and Kidney Function Markers

The automated chemistry analyzer (Smart 150 - U.S.A.) facilitated the quantification of Urea, Creatinine, Aspartate Aminotransferase (AST), and Alanine Aminotransferase (ALT). This was achieved through Auto Chemistry analysis, which involved the use of pre-prepared test kits to automatically determine the concentration levels of enzymes in a serum sample. The analyser's system ensured standardized and precise measurements for these specific tests.

2.5. Statistical Analysis

Analysis of variance was used to analyse the findings of this study. The findings of this study were analysed using analysis of variance (SPSS), and the T-test was used to analyse variance between two groups at the probability level (P≥0.05). The ANOVA test was used to analyse the variance between the two groups. The linear correlation coefficient was found (correlation coefficient) to find the correlation between the two cubes and the variables studied in this research. The graphs and tables were drawn using Excel 2010.

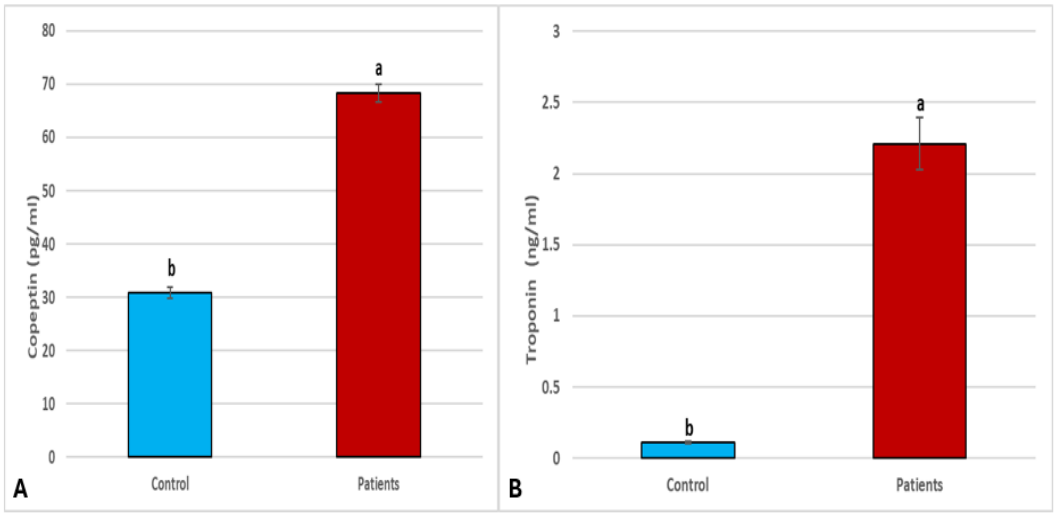
3. Bulgular

Çalışmaya katılan öğrencilerin %28,8'inin 20-23 yaş grubunda olduğu, %67,8'nin kadın, %32,2'sinin ise erkek olarak belirlendi. Katılımcıların %77,2'si çekirdek aile yapısına sahipti. Doğum yerlerine ilişkin analizde, öğrencilerin %68,3'ü il merkezi, 62'si %34,4'ü ise köy doğumlu idi. İkamet ettikleri bölgelere göre dağılımda; %41,7'si Karadeniz, %13,3'ü Akdeniz, %7,2'si Ege, 21'i %11,7'si Marmara, %10,6'sı Doğu Anadolu ve %4,4'ü Güneydoğu Anadolu Bölgesi'nde yaşadığı görüldü. Ebeveynlerin eğitim düzeyine bakıldığında, annelerin %35,6'sı ilk ve ortaöğretim mezunu, %33,9'u ise üniversite mezunu iken; babaların %28,3'ü ilk ve ortaöğretim, %48,3'ü üniversite mezunu olduğu sonucuna ulaşıldı. Ayrıca, öğrencilerin annelerinin %53,9'u herhangi bir işte çalışmadığı sonucuna varıldı (Tablo 1).

3.1. Copeptin and Troponin Results

The basic results for the two groups, presented for cardiac parameters copeptin and troponin, are shown in Table 1 and Figure 1. In the control group, the results for copeptin were (30.863±6.844), while in patients, the results were (68.258±12.164). For troponin, the control group had results of (0.110±0.030), while in patients, the results were (2.21±1.301). It appears that the value of these tests is higher in patients with heart failure compared to healthy individuals.

Figure 1. The Levels of Cardiac Parameters



Copeptin (A) and Troponin (B) levels in patients and the healthy group. The graphs (a and b) demonstrate statistically significant differences ($p \leq 0.05$) between each group.

TURKISH JOURNAL OF MEDICAL AND HEALT SCIENCES
https://journals.academicianstudies.com/TTSB
E-ISSN; 3062-1720
https://doi.org/10.71284/tjmhs.2025232

Table 1. Serum Copeptin and Troponin Parameters

Parameter/ Group	Copeptin (pg/mL)	Troponin (ng/mL)
Control	30.863±6.844[b]	0.110±0.030[b]
Patients	68.258±12.164[a]	2.21±1.301[a]

Values represent mean ± SD for each group. Different superscript letters (a, b) within the same column Indicate Significant differences between the patient groups and the control ($p < 0.01$), as shown in the table above.

3.2. The Results of Liver and Kidney Function Markers

The results show a significant increase in urea levels among the patient group compared to healthy individuals, with levels of (24.676±5.021) in the control group and (55.340±13.014) in patients. Similarly, creatinine levels were higher in patients (1.286 ± 0.371) compared to the control group (0.698 ± 0.123). Additionally, ALT levels were elevated in patients (52.72±10.19) compared to the control group (17.38±7.61), and AST levels were also higher in patients (51.88±8.53) compared to the control group (18.12±6.99) (Table 2).

Table 2. Serum AST, ALT, Urea, and Creatinine Parameters

Parameter/ Group	AST (U/L)	ALT (U/L)	Urea (mg/dL)	Creatinine (mg/dL
Control	18.12±6.997[b]	17.38±7.610[b]	24.676±5.021[b]	0.698±0.123[b]
Patients	51.88±8.532[a]	52.72±10.190[a]	55.340±13.014[a]	1.286±0.371[a]

Values represent mean ± SD for each group—different superscript letters (a, b) within the same column. There were statistically significant differences between the patient groups and the control ($p < 0.01$). AST: aspartate transaminase; ALT: alanine transaminase.

3.3. Oxidative Stress Parameter Results

The values of some antioxidant tests show different results between patients with heart failure and healthy individuals Figure 2. For example, the levels of GSH in the control group were 9.433±2.191 µmol/L, whereas in patients, they were 5.544±1.594 µmol/L. Similarly, GPx levels in the control group were (3.391±0.872 U/L), whereas in patients, it was (1.349±0.434 U/L). SOD levels in the control group were (9.571±1.377 U/L), compared to (5.679±1.891 U/L) in patients; this indicates a decrease in the values of these tests for patients when compared with the results of healthy individuals. Additionally, MDA levels increased in patients with heart failure (6.734±2.364 nmol/mL) compared to the control group (4.505±0.978 nmol/mL). It is important to note that many individuals with heart failure also had pre-existing conditions such as high blood pressure and kidney disease.

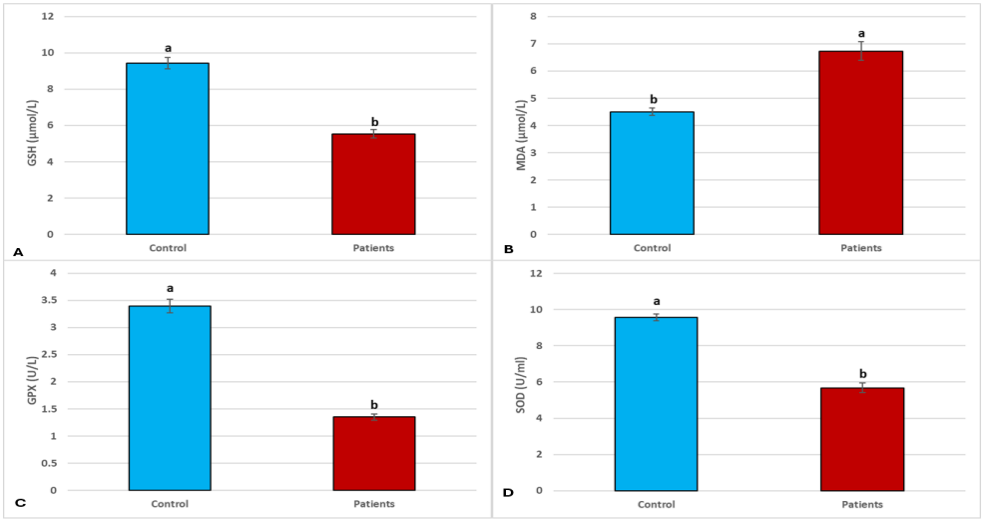
4. Tartışma

Heart failure is a disease generally characterized by a decrease in the heart's ability to pump/fill with blood or a structural or functional abnormality that causes insufficiency in cardiac functions (Giannopoulos et al., 2013). There is evidence that Copeptin may be used in the diagnosis, prognosis, and risk stratification of some cardiovascular diseases, including the prognosis of chronic stable or destabilized heart failure (Docherty et al., 2023). The heart failure patients in our study showed a significant increase in copeptin levels compared to the control group, which aligns with previous studies (AL-doorie, 2020). Elevated copeptin levels in heart failure primarily result from the activation of the neurohormonal system, specifically the release of arginine vasopressin (AVP) or antidiuretic hormone (ADH). Heart failure triggers a compensatory response involving various neurohormonal systems from the hypothalamic gland to maintain blood pressure and fluid balance. Copeptin levels decrease after five days of injury, as shown in a study conducted by Schill et al. (2021). Reveal an increase in human troponin I (cTnI), potentially causing damage to myocardial tissues. The cTnI isoform under study consists of 209 amino acids, weighing approximately 23-24 kilodaltons (Gaze & Collinson, 2008). In the aftermath of heart failure, three distinct cTnI isoforms, usually found at minimal levels in the bloodstream, undergo a substantial surge. These elevated cTnI levels become detectable within 4-6 hours, peak around 12-24 hours post-heart failure (Hamm et al., 1997), and remain detectable for an extended period. This heightened sensitivity and specificity render cTnI valuable in diagnosing heart failure, allowing detection within as little as 4 hours after the onset of ischemia (Nojiri et al., 2001). It was determined that ALT, AST, urea, and creatinine levels increased significantly in the patient group compared to the control group. Increased AST and ALT enzyme activity in heart failure patients is a result of heart muscle cell necrosis, which releases these enzymes into the bloodstream. Elevated AST levels indicate heart muscle damage, making them a useful diagnostic marker for cardiovascular diseases, as shown in studies conducted by Lin et al. (2010).

TURKISH JOURNAL OF MEDICAL AND HEALT SCIENCES
https://journals.academicianstudies.com/TTSB
E-ISSN; 3062-1720
https://doi.org/10.71284/tjmhs.2025232

Elevated urea levels can occur due to damage to cardiac muscle cells, which leads to the release of proteins and amino acids into the bloodstream that eventually convert into urea. Additionally, high blood pressure can increase filtration from cardiac muscle cells, resulting in higher levels of substances, including urea, in the blood. These findings are consistent with a study conducted by Laville et al. (2023). Elevated creatinine levels often occur in people who have had a heart attack, as increased cell membrane permeability releases enzymes into the bloodstream. Similarly, in patients with heart failure, cell membrane damage leads to the release of enzymes, which raises creatinine concentrations in the blood. These observations are supported by Horjus (2019). GSH (A), MDA (B), GPX (C), and SOD (D) levels in patients and the healthy group. The graphs (a and b) demonstrate statistically significant differences ($p \leq 0.05$) between each group.

Figure 2. The Antioxidant Levels Between the Patient and Healthy Group



This study reveals elevated oxidative stress and high peroxide levels in the fat of heart patients. These conditions lead to the release of malondialdehyde as a byproduct of lipid peroxidation, increasing its blood concentration. These findings align with a study conducted by O'Brien et al. (2005). Reduced glutathione (GSH) levels in heart failure patients align with a study conducted in 2014 by Yang and co-workers. GSH levels decrease due to oxidative stress, inflammation, and impaired antioxidant defenses. GSH acts as a reducing agent, protecting cellular membranes from oxidative stress and free radical damage. Our studies revealed a decrease in blood serum glutathione levels in the patient group, likely due to increased oxidative stress impacting the activity of glutathione peroxidase (GPx). This decrease aligns with the role of GPx in protecting cardiac tissues from oxidative damage in pathological conditions (Yang et al., 2014). The enzyme's activity relies on glutathione, and our findings support the association between its reduced concentration and the observed changes. Patients with heart failure exhibited decreased levels of superoxide dismutase (SOD). Reduced SOD levels in coronary arteries may stem from endothelial cell dysfunction resulting from ROS formation. Elevated SOD levels in outer endothelial cells could counteract declining vascular function due to increased oxygen-free radical formation (Harrison et al., 2003). Results obtained from the measurement of oxidative stress markers suggest a potential link between antioxidant levels and heart failure, particularly in individuals with co-existing health issues.

5. Conclusion

Patients with heart failure exhibited significantly elevated copeptin levels compared to the control group, making it a potential indicator for heart-related issues. Copeptin's role in regulating fluid balance involves controlling the elimination of excess water via the kidneys. This process is triggered when cardiac muscle cells undergo necrosis due to oxygen deprivation from coronary artery blockage, leading to copeptin release into the bloodstream and subsequently increased levels. Copeptin levels increased in heart failure patients, influenced by gender and age, differing from the healthy group. Patients with heart failure exhibited notably higher levels of urea, creatinine, and troponin compared to the healthy group. GSH, AST, ALT, and MDA levels significantly escalated in comparison to the healthy group among heart failure patients. Heart failure patients exhibited a significant decrease in GPx and SOD levels compared to the healthy group.

Kaynaklar

Abdelmageed, M, Güzelgül, F. (2023) Copeptin: Up-to-date diagnostic and prognostic role highlight. *Analytical Biochemistry, 673*, s. 115181.

TURKISH JOURNAL OF MEDICAL AND HEALT SCIENCES
https://journals.academicianstudies.com/TTSB
E-ISSN; 3062-1720
https://doi.org/10.71284/tjmhs.2025232

AL-doorie, H. T. (2020) Evaluation of some physidogical and biochemical parameters by using new biomarkers in the Diagnosis of Heart disease in tikrit city. Tikrit: University of Tikrit, PhD Thesis.

Al-Helaly, L. A., Ahmed, T. Y. (2014) Antioxidants and some biochemical parameters in workers exposed to petroleum station pollutants in Mosul City, Iraq. *International Research Journal of Environment Sciences, 3*(1), s. 1-7.

Anderson, J. L., Morrow, D. A. (2017) Acute heart failure. *New England Journal of Medicine, 376*(21), s. 2053-2064.

Balanescu, S., Kopp, P., Gaskill, M. B., Morgenthaler, N. G., Schindler, C., Rutishauser, J. (2011) Correlation of plasma copeptin and vasopressin concentrations in hypo-, iso-, and hyperosmolar States. *The Journal of Clinical Endocrinology & Metabolism, 96*(4), s. 1046-1052.

Docherty, K. F., Lam, C. S., Rakisheva, A., Coats, A. J., Greenhalgh, T., Metra, M., et al. Heart failure diagnosis in the general community–Who, how and when? A clinical consensus statement of the Heart Failure Association (HFA) of the European Society of Cardiology (ESC). *European Journal of Heart Failure, 25*(8), s. 1185-98.

Fenske, W. K., Schnyder, I., Koch, G., Walti, C., Pfister, M., Kopp, P., Fassnacht, M., Strauss, K., Christ-Crain, M. (2018a) Release and decay kinetics of copeptin vs AVP in response to osmotic alterations in healthy volunteers. *The Journal of Clinical Endocrinology & Metabolism, 103*(2), s. 505-13.

Fenske, W., Refardt, J., Chifu, I., Schnyder, I., Winzeler, B., Drummond, J., et al. (2018b) A copeptin-based approach in the diagnosis of diabetes insipidus. *New England Journal of Medicine, 379*(5), s. 428-39.

Gaze, D. C., Collinson, P. O. (2008) Multiple molecular forms of circulating cardiac troponin: analytical and clinical significance. *Annals of Clinical Biochemistry, 45*(4), s.349-55.

Giannopoulos, G., Deftereos, S., Panagopoulou, V., Kossyvakis, C., Kaoukis, A., Bouras, G., et al. (2013) Copeptin as a biomarker in cardiac disease. *Current topics in medicinal chemistry, 13*(2), s. 231-40.

Gladysheva, I. P., Sullivan, R. D. (2022). *Diagnosis and Management of Heart Failure*. Basel: MDPI.

Hamm, C. W., Goldmann, B. U., Heeschen, C., Kreymann, G., Berger, J., Meinertz, T. (1997) Emergency room triage of patients with acute chest pain by means of rapid testing for cardiac troponin T or troponin I. *New England Journal of Medicine, 337*(23), s. 1648-53.

Harrison, D., Griendling, K. K., Landmesser, U., Hornig, B., Drexler, H. (2003) Role of oxidative stress in atherosclerosis. *The American Journal of Cardiology, 91*(3), s. 7-11.

Hayes, J. D., Dinkova-Kostova, A. T., Tew, K. D. (2020) Oxidative stress in cancer. *Cancer cell, 38*(2), s. 167-97.

Horjus, D. J. (2019) Creatine kinase and cardiovascular disease. Amsterdam: University of Amsterdam, PhD Thesis.

Katrukha, A. G., Bereznikova, A. V., Esakova, T. V., Pettersson, K., Lovgren, T., Severina, M. E., et al. (1997) Troponin I is released in bloodstream of patients with acute myocardial infarction not in free form but as complex. *Clinical chemistry, 43*(8), s. 1379-85.

Katz, M. J., Ness, S. M. (2015) Coronary artery disease (CAD). *Journal of Graduate Medical Education. 1*(1), s. 2-89.

Kleinman, M. E., Goldberger, Z. D., Rea, T., Swor, R. A., Bobrow, B. J., Brennan, E. E., et al. (2018). American Heart Association focused update on adult basic life support and cardiopulmonary resuscitation quality: an update to the American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation, 137*(1), s. 7-13.

Kumar, R., Chhillar, N., Gupta, D. S., Kaur, G., Singhal, S., Chauhan, T. (2023) Cholesterol homeostasis, mechanisms of molecular pathways, and cardiac health: a current outlook. *Current Problems in Cardiology, 14*, s.102081.

Laville, S. M., Couturier, A., Lambert, O., Metzger, M., Mansencal, N., Jacquelinet, C., et al. (2023) Urea levels and cardiovascular disease in patients with chronic kidney disease. *Nephrology Dialysis Transplantation, 38*(1), s. 184-92.

Lin, J. D., Lin, P. Y., Chen, L. M., Fang, W. H., Lin, L. P., Loh, C. H. (2010) Serum glutamic-oxaloacetic transaminase (GOT) and glutamic-pyruvic transaminase (GPT) levels in children and adolescents with intellectual disabilities. *Research in Developmental Disabilities, 31*(1), s. 172-7.

Morgenthaler, N. G., Struck, J., Jochberger, S., Dünser, M. W. (2008) Copeptin: clinical use of a new biomarker. *Trends in endocrinology & metabolism, 19*(2), s. 43-49.

Nojiri, S., Daida, H., Mokuno, H., Iwama, Y., Mae, K., Ushio F,et al. (2001) Association of serum antioxidant capacity with coronary artery disease in middle-aged men. *Japanese Heart Journal, 42*(6), s. 677-90.

O'Brien, P. J., Siraki, A. G., Shangari, N. (2005) Aldehyde sources, metabolism, molecular toxicity mechanisms, and possible effects on human health. *Critical Reviews in Toxicology, 35*(7), s. 609-62.

Pozsonyi, Z., Förhécz, Z., Gombos, T., Karádi, I., Jánoskuti, L., Prohászka, Z. (2015) Copeptin (C-terminal pro arginine-vasopressin) is an independent long-term prognostic marker in heart failure with reduced ejection fraction. *Heart, Lung and Circulation, 24*(4), s. 359-367.

Savarese, G., Becher, P. M., Lund, L. H., Seferovic, P., Rosano, G. M., Coats, A. J. (2022) Global burden of heart failure: a comprehensive and updated review of epidemiology. *Cardiovascular research, 118*(17), s. 3272-87.

Schill, F., Timpka, S., Nilsson, P. M., Melander, O., Enhörning, S. (2021) Copeptin as a predictive marker of incident heart failure. *ESC heart failure, 8*(4), s. 3180-8.

Yang, S., Jensen, M. K., Rimm, E. B., Willett, W., Wu, T. (2014) Erythrocyte superoxide dismutase, glutathione peroxidase, and catalase activities and risk of coronary heart disease in generally healthy women: a prospective study. *American Journal of Epidemiology, 180*(9), s. 901-8.

TURKISH JOURNAL OF MEDICAL AND HEALT SCIENCES
https://journals.academicianstudies.com/TTSB
E-ISSN; 3062-1720
https://doi.org/10.71284/tjmhs.2025232

Zhang, C., Bruins, M. E., Yang, Z. Q., Liu, S. T., Rao, P. F. (2016) A new formula to calculate activity of superoxide dismutase in indirect assays. *Analytical biochemistry*, *503*, s. 65-7.

Yalçın, E. K. (2019). *Türkiye'deki etnik grupların toplumsal cinsiyet rollerine ilişkin tutumlarının karşılaştırılması* (Publication Number 657443) Hasan Kalyoncu Üniversitesi].

TURKISH JOURNAL OF MEDICAL AND HEALT SCIENCES
https://journals.academicianstudies.com/TTSB
E-ISSN; 3062-1720
https://doi.org/10.71284/tjmhs.2025232

Article Information

Name and surname Sura Salim MOHAMMED,
Fahad K. Y. Al-DULAIMI,
Müslüm KUZU

Adress Department of Chemistry, Institute of Graduate Programs, Karabuk University, Karabuk/Türkiye
Department of Plant Production Technical,Faculty of Technical Agricultural, Northern Technical University, Mosul, 41001, Iraq
Department of Nutrition and Dietetics, Faculty of Health Sciences, Karabuk University, Karabuk, 78050, Türkiye

Email address suraalsamaraia@gmail.com,
fahadbiologymycology@ntu.edu.iq,
muslumkuzu@karabuk.edu.tr

Orcid 0000-0002-6117-6371, 0000-0002-5958-3496, 0000-0002-1375-7673

Article type Research Artical

Article Arrival date 06.11.2025

Revision date 09.12.2025

Accepted Date 10.12.2025

Published date 30.12.2015

DOI **https://doi.org/10.71284/tjmhs.2025232**

Peer review Externally and internally peer-reviewed.

Ethical Statement This study was conducted with the approval of the Non-Interventional Clinical Research Ethics Committee of Karabük University (Approval No: 2023/1232) and with the permission of the Research and Information Management Department of the Education and Human Development Center, Salah din Health Directorate, Ministry of Health, Iraq (Permit No: 2038, dated 22.11.2022).

Artificial intelligence usage statement In this study,generative artificial intelligence tools were used in a limited manner solely for writing support, in accordance with the ethical guidelines published by the Council of Higher Education (YÖK). The entire content was produced by the author(s), who bear full responsibility for the final manuscript.

Author Contribution Rate All authors contributed equally.

Plagiarism Checks Yes;Ithenticate/Turnitin.

Conflict of interest No conict of interest was declared by the authors.

Grant Support or funding Authors declared that this tudy received no financial support

ROR code https://ror.org/04wy7gp54

JEL code I12

Citation Mohammed, S.S., Al-Dulami, F.K.Y., Kuzu, M. (2025). Determination Of Copeptin and Oxidative Stress Markers In Heart Failure Patients. Turkish Journal of Medicine and Health Sciences, 2(3):9-16.