Open Access

Journal of the Faculty of Medicine Baghdad
Print ISSN: 0041-9419, OnlineISSN: 2410-8057
DOI: https://doi.org/10.32007/jfacmedbaghdad3145

Research Article

Impact of Biologic and Conventical Synthetic DMARDs on Serum Coenzyme Q10 and Malondialdehyde in Rheumatoid Arthritis Patients

Nagham Y. Mohsin*[1], Basil O. Mohammed Saleh[1], Faiq I. Gorial[2]
[1]*Department of Clinical Biochemistry, College of Medicine, University of Baghdad, Baghdad, Iraq.*
[2]*Department of Internal Medicine, College of Medicine, University of Baghdad, Baghdad, Iraq.*

Received: April, 2025
Revised: May, 2025
Accepted: June, 2025
Published Online: June, 2025

Abstract

Background: Rheumatoid arthritis (RA) is a chronic inflammatory autoimmune illness. Oxidative stress such as Malondialdehyde plays a major role in the pathophysiology of RA while antioxidants such as Coenzyme Q10 play protect role against inflammation.

Objectives: The study aimed to compare serum concentrations of Coenzyme Q10 and Malondialdehyde in rheumatoid arthritis patients and healthy controls and to evaluate how different types of treatment (biologics and chemotherapy) influence serum levels of them in this disease.

Methods: The case-control study was conducted on 88 individuals (60 had been diagnosed with rheumatoid arthritis and 28 healthy as (Controls). The patients were recruited from the Rheumatology Unit of Baghdad Teaching Hospital in Baghdad, Iraq, during the period from November 2024 to March 2025. They were subdivided into four groups according to the type of treatment (biology, biology & chemotherapy, biology & other, chemotherapy & other). Serum investigations included measurements of anti-cyclic citrullinated peptide antibody, high-sensitivity C-reactive protein, Coenzyme Q10, and Malondialdehyde, measured by using Enzyme linked immunosorbent assay technique.

Results: The study found that the mean (±SD) values of Malondialdehyde were significantly highly increased (705.77 ± 168.83, p<0.0001), while that of Coenzyme Q10 was significantly lower (6.26 ± 1.08, p<0.0001) in RA patients. Also, the mean levels differed significantly among the treatment groups with the biological therapy with the other group having the highest malondialdehyde (and the lowest Coenzyme Q10.

Conclusion: Patients exhibited significantly elevated levels of Malondialdehyde and reduced levels of Coenzyme Q10, indicating oxidative stress. Treatment modalities significantly affect these biomarkers.

Keywords: Antioxidant; Coenzyme Q10; Malondialdehyde; Oxidative stress; Rheumatoid Arthritis.

Introduction:

Rheumatoid Arthritis (RA) is a symmetric polyarthritis that affects several joints bilaterally, causing discomfort and swelling in the hands and feet. The swelling is largely in the wrists and joints, and morning stiffness can continue for many hours. In contrast to psoriatic arthritis, finger swelling is concentrated around the joint. Both tiny and major joints may be implicated (1, 2). The exact cause of RA is unknown, but it is thought to develop in individuals with inherited genetic factors, after exposure to environmental triggers like cigarette smoking and exogenous hormones such as medications containing estrogen and progesterone/progestin, which are used for contraception. This is followed by immune activation, which results in the presence of inflammatory markers and autoantibodies. clinically evident articular disease occurs in the early rheumatoid arthritis phase (3).

*Corresponding author: nagham.shahl2309m@comed.uobaghdad.edu.iq

Coenzyme Q10 (CoQ10), also known as ubiquinone, is a lipid-soluble, vitamin-like compound that is essential for the synthesis of cellular energy and serves as a strong antioxidant shielding cells from oxidative damage brought on by free radicals, protecting cells from oxidative damage caused by free radicals (4). It is synthesized in the mitochondria and is crucial for the electron transport chain, facilitating ATP production, particularly in high-energy-demand organs, such as the heart and brain (5). Coenzyme Q10 (CoQ10) deficiency has been implicated in various disorders, including RA, due to its critical roles in mitochondrial function and antioxidant defense. CoQ10 is essential for ATP production in the electron transport chain and acts as a lipid-soluble antioxidant, mitigating oxidative stress caused by reactive oxygen species (ROS) (6). In RA, increased oxidative stress and inflammation are prominent, suggesting that CoQ10 deficiency may exacerbate these conditions by impairing cellular

energy metabolism and promoting apoptosis (7). Serum malondialdehyde (MDA), a common biomarker of oxidative stress, accumulates in human tissues and indicates the presence of elevated oxidative stress levels, which can exacerbate or cause various chronic diseases across multiple body systems (8,9). Malondialdehyde plays a significant role in the pathogenesis of RA through its association with oxidative stress and the immune response. Elevated serum levels of MDA are observed in RA patients, correlating with disease activity markers and inflammatory cytokines like CRP and IL-6 (10,11).

The aim of the study was to investigate the oxidant-antioxidant imbalance in patients with rheumatoid arthritis by measuring the serum levels of CoQ10 and MDA and to evaluate the effects of treatment types on them.

Patients and Methods:

The case-control study was conducted in the Rheumatology unit /Baghdad Teaching Hospital, Medical City, Baghdad, Iraq, from November 2024 to March 2025. A total of 88 subjects were included in this study. Sixty of them were diagnosed by a consultant rheumatologist to have had rheumatoid arthritis using either the criteria established by the American College of Rheumatology (ACR) in 1987 or the criteria established by the European Alliance of Associations for Rheumatology (ACR- EULAR) in 2010. These patients were stratified into four main groups based on their therapeutic protocols: group 1 [biology only; biological Disease-Modifying Antirheumatic Drugs (bDMARDs), n=22), group 2 (Biology + chemotherapy; (bDMARDs) and Chemical Conventional synthetic DMARDs (csDMARDs) agents, n=11), group 3 [(biology (bDMARDs) and other (NSAIDs, simple analgesics, or corticosteroids), n=18)], and group 4 [chemotherapy (csDMARDs) and other, n=9)].

The inclusion criteria were adults over 18 years of age of both the sex with established rheumatoid arthritis.

The control group consisted of 28 healthy subjects selected from colleagues and relatives who were healthy and not suffering from any acute or chronic illness, like diabetes mellitus, thyroid disorders, autoimmune diseases, osteoarthritis and rheumatoid arthritis.

The exclusion criteria were pregnant women, diabetes mellitus, thyroid gland disorders, those with any other autoimmune disorders such as osteoarthritis, systemic lupus erythematosus (SLE), multiple sclerosis, and those on supplement of CoQ10, based on history, physical examination, and laboratory results including Thyroid stimulating hormone (TSH), antinuclear antibody (ANA) and hemoglobin A1c (HbA1c).

A Comprehensive medical history was collected for each participant, documenting basic demographic and clinical data, including age, sex, weight, height, and the duration of the disease. Body mass index (BMI) was calculated for all participants according to global equation: BMI= [weight (Kg) / height (m^2)].

Five milliliters (ml) of blood were aspirated from the peripheral vein of each patient and control group and permitted to coagulate for 15 minutes, subsequently centrifuged for 10 minutes at 2500 rpm. The isolated serum was preserved at -45°C until the day of laboratory analysis, which included measurements of Malondialdehyde, Coenzyme Q10, anti-cyclic citrullinated peptide (anti-CCP), and high sensitive- C reactive protein (hs-CRP). Malondialdehyde was measured using a semiautomatic ELISA Reader (Human Reader, by the Human Diagnostic USA company, Elabscience, the principle of the ELISA technique was a Competitive-ELISA principle and measurements of Coenzyme Q10 by the Human Diagnostic USA company ELK Biotechnology the principle was also Competitive-ELISA principle. The measurement of anti-CCP by Human Diagnostic China company, Fine Test, the principle was indirect-ELISA and hs-CRP was performed by the Human Diagnostic USA company, Elabscience using Sandwich-ELISA principle.

Statistical Analysis was done using the Med Calc Statistical Software version 20.110 and IBM SPSS version 26.0 which described the data using percentages, means, and standard deviation (± SD**).** Data distribution was found to be normal and the ANOVA test was used to assess the differences between means of numerical data when more than two means were tested. The only exception is that of disease duration which has non-normal distribution and a wide range. The correlation between the numerical data was evaluated using the Pearson correlation regression. A *P* value of < 0.05 was considered significant.

Results:

A total of 88 participants were enrolled in this study, comprising 60 patients with Rheumatoid Arthritis (RA) and 28 healthy controls. The sex distribution, age, body mass index, and duration of disease in studied groups are summarized in Table 1. The analysis of sex distribution between the RA and control groups showed no significant difference. Both groups showed a predominance of female participants. The RA group was significantly older than the control group ($P<0.0235$), while body mass index (BMI) was not significantly different between the groups. Disease duration data were only applicable to the RA group, with a median of 18.0 years (Table 1).

Table 1: Demographic characteristics of study participants

Parameter	Rheumatoid arthritis Group (n=60)	Control Group (n=28)	*P* value
gender [NS]			
Female	51 (85.0%)	22 (78.6%)	
Male	9 (15.0%)	6 (21.4%)	0.4
Age (years)	48.20 ± 10.63	42.86 ± 8.91	0.0235•
Body mass index [NS] (kg/m²)	29.70 ± 6.01	30.5 ± 4.5	0.5208
Disease duration (years)	median 18	N/A	

Independent *t*-test revealed: NS: non-significant differences in sex distribution and BMI, ● significant difference in age.

Table 2 showed a comparative analysis of four studied biomarkers including anti-CCP, hs-CRP, CoQ10, and MDA between rheumatoid arthritis patients and healthy control subjects. The mean value of Anti-CCP and hs-CRP were significantly increased in RA patients in comparison with the control group (P <0.0001). In addition, the mean of MDA levels was significantly elevated in RA patients compared to control subjects (p <0.0001), representing a 210% increase in the RA group. While, the mean CoQ10 levels were significantly lower in RA patients compared to control subjects (P< 0.0001), representing a decrease of approximately 51% in the RA group.

Table 2: Mean (±SD) values of anti-cyclic citrullinated peptide antibody, high-sensitivity C-reactive protein, malondialdehyde, and Coenzyme Q10 in rheumatoid arthritis and control groups

Parameter	Control group n=28	RA patient n=60	*P* value
Anti-CCP (ng/ml)	10.43 ± 2.24	16.52 ± 4.17	<0.0001•
hs-CRP (ng/ml)	140.03 ± 21.48	238.37 ± 83.11	<0.0001•
MDA (ng/ml)	227.75 ± 26.15	705.77 ± 168.83	<0.0001•
CoQ10(ng/ml)	12.84 ± 1.33	6.26 ± 1.08	<0.0001•

***t*-test revealed: ● significant differences in studied parameters between two groups. Anti-CCP (anti-cyclic citrullinated peptide antibody), hs-CRP (high-sensitivity C-reactive protein), MDA (malondialdehyde) and CoQ10 (Coenzyme Q10)**

Table 3 and Figures (1 &2) presented a comparative analysis of age, BMI, anti-CCP, hs-CRP, MDA, and CoQ10 of the four subgroups (biology, biology, and chemotherapy, biology+ other, and Chemotherapy+ other) of RA patients according to the type of treatment. A significant difference was observed in mean MDA levels across treatment groups (P=0.0300). The biology + other group exhibited the highest MDA levels, differing significantly from certain other groups. Serum CoQ10 showed the highest levels in biology. only group and the lowest in the biology +other group. The mean (±SD) values of coenzyme Q10 levels of biology+ other and chemotherapy+ other were significantly lower than that of biology only (p < 0.001). Also, the mean value of coenzyme Q10 levels of biology+ other was significantly lower than that of biology+ chemotherapy (p=0.01). Also, the mean value of coenzyme Q10 of chemotherapy+ other was significantly lower than that of biology+ chemotherapy (p =0.03).

Table 3: Mean (± SD) values of Rheumatoid arthritis subgroups according to type of treatment (biology, biology+ chemotherapy, biology+ other, and chemotherapy+ other)

Parameter	Biology only (n = 22) (bDMARDs)	Biology (bDMARDs) + chemotherapy (csDMARDs) n = 11	Biology (bDMARDs) + other n = 18	Chemotherapy (csDMARDs) + other n = 9	*P* value
Age(years)[NS]	46.77 ± 13.01	49.82 ± 8.15	47.17 ± 9.94	51.78 ± 8.42	0.6172
BMI (kg/m²) [NS]	29.17 ± 7.20	29.56 ± 4.95	30.23 ± 5.88	30.12 ± 4.99	0.9516
Anti-CCP(ng/ml)	15.47 ± 4.01	17.30 ± 5.28	17.06 ± 4.06	17.04 ± 3.34	0.5377
hs-CRP (ng/ml)	244.56 ± 93.21	194.00 ± 35.42	263.91 ± 85.38	226.39 ± 81.49	0.1609
MDA (ng/ml)	643.26[a] ± 208.12	658.74 ± 146.45	783.69[a] ± 128.04	760.20 ± 63.95	0.0300•
CoQ10(ng/ml)	6.95[a,b] ± 0.92	6.56[c] ± 0.81	5.73[a] ± 0.92	5.25[b,c] ± 0.75	<0.0001•

ANOVA and independent *t*-tests were used for statistical analysis; ● significant differences, NS: non-significant differences.

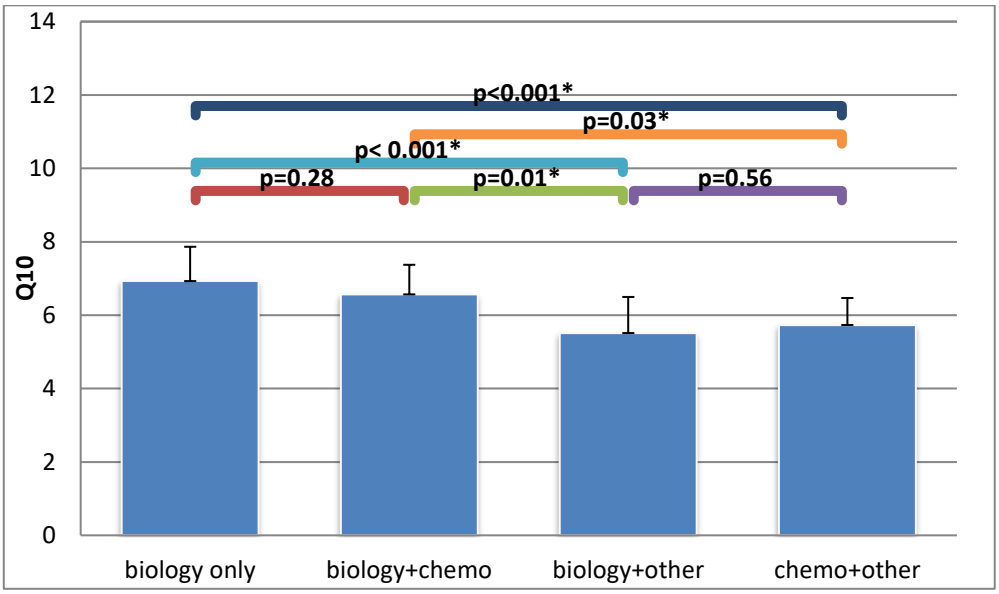

Figure (1): Mean serum level of Coenzyme Q10 in Rheumatoid Arthritis patients across treatment types.

The mean± SD of malondialdehyde levels of biology+ other was significantly higher than biology only (p=0.02; Figure 2).

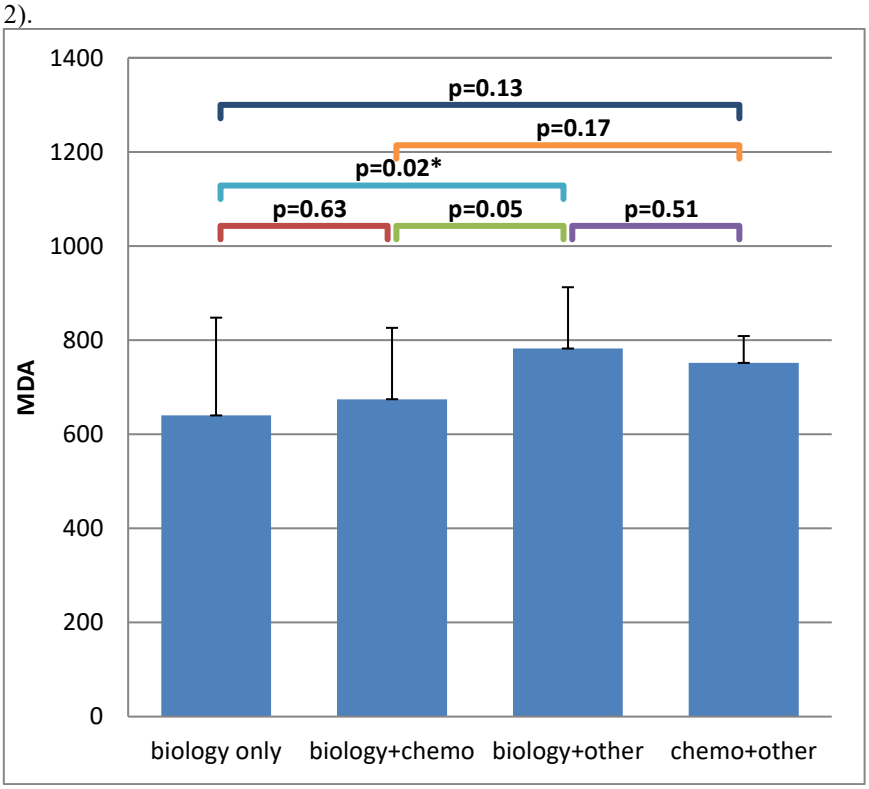

Figure (2): Mean serum level of Malondialdehyde in Rheumatoid Arthritis patients across treatment types.

Discussion:

The current study revealed a higher prevalence of rheumatoid arthritis (RA) among females compared to males, which is consistent with the known higher prevalence of rheumatoid arthritis among women in the general population, aligning with the well-documented greater incidence of RA in women within the general population. This finding corroborates the results reported by Aldouri *et al.* (12) & Al-Jumaily *et al.* (13).

The mean body mass index BMI was in the overweight to obese, suggesting greater variability in body composition among RA patients. This finding is consistent with the result of Feng X *et al*. study which demonstrated that elevated BMI correlated with an increased risk for rheumatoid arthritis (14). and another study that was done in Egypt, found that 25.8% of the

RA patients had normal weight and the others had overweight or obesity (15).

In the present study, Coenzyme Q10 level was significantly lower in RA patients. This substantial reduction in Q10 levels among RA patients indicates a marked depletion of this antioxidant in the disease state. This result was in agreement with those reported by Gvozdjáková *et al.* (2021) study have shown that RA patients exhibited significantly reduced platelet CoQ10 levels compared to healthy controls, which may contribute to mitochondrial dysfunction and increased oxidative stress in these individuals. This substantial reduction in Q10 levels indicates a marked depletion of this antioxidant in the disease state. This supports its potential utility for RA and suggests possible therapeutic implications targeting antioxidant pathways (16). Another study that found CoQ10 supplementation in rheumatoid arthritis patients enhanced clinical results and reduced disease severity. CoQ10 may offer a novel adjunctive strategy for those with rheumatoid arthritis. (17). Malondialdehyde levels were significantly elevated in RA patients. This elevated indicates significantly increased oxidative stress in RA patients and may serve as a valuable marker for monitoring disease activity and oxidative burden.

This finding is consistent with the result of Saeed U *et al.* (2024) elevated levels of MDA in RA patients highlighted their significance as oxidative stress indicators, representing the continuous inflammatory process and probable joint damage in RA (18).

The malondialdehyde levels of biological therapy with other treatments were significantly higher than biology therapy only, while the opposite of the anti-oxidant substance, the CoQ10. These results reflect the high content of oxidant materials in NSAIDs, analgesics, glucocorticoids, and bypass products from their metabolism.

These results indicate the significant effects of supportive drugs like NSAID and glucocorticoid ones in potentiating reactive oxygen species and reducing the anti-oxidant substances. de Paz *et al.* (2024) found that only the consumption of NSAIDs shown a significant negative link with MDA, whereas DMARDs exhibited a non-significant association with MDA levels (19). Nonsteroidal anti-inflammatory drugs (NSAIDs), including ibuprofen and celecoxib, alleviate pain and inflammation by decreasing cyclooxygenase (COX) activity. While they offer symptomatic relief, they do not alter the disease trajectory or avert joint deterioration. The prolonged use of NSAIDs is linked to gastrointestinal toxicity, nephrotoxicity, and cardiovascular problems, particularly in patients with concomitant conditions. (20). Glucocorticoids are powerful anti-inflammatory and immunosuppressive compounds. Short-term use can yield considerable clinical relief; however, prolonged medication results in severe adverse effects, including osteoporosis, hypertension, diabetes, and an elevated risk of infection. (21). Antioxidants have demonstrated protective benefits against tissue damage in certain trials and may result in clinical improvement for these patients. (22). This is thought to result from antioxidant molecules mitigating inflammation by influencing the NF-κB transcription factor in rheumatoid arthritis patients. A recent study demonstrated that coenzyme Q10, a fat-soluble antioxidant, reduced serum MDA levels and the pro-inflammatory cytokine, TNF-α, in rheumatoid arthritis. (23). Bilski& Nuszkiewicz concluded that Oxidative stress is a critical factor in the etiology of rheumatoid arthritis, exacerbating inflammatory processes and leading to the degradation of joint structures. Antioxidant therapy, encompassing both pharmaceutical and nutritional methods, represents intriguing adjuncts to conventional rheumatoid arthritis treatment. Current pharmaceuticals mostly on immunological modulation and inflammation reduction, neglecting redox equilibrium. Consequently, they fail to adequately mitigate oxidative damage, potentially resulting in additional joint deterioration, despite managing inflammation (24,25).

Limitations:

Inability to incorporate newly diagnosed participants with RA due to the scarcity of cases observed throughout the study period. Also, the incomparable patients' number in different groups of treatment types, and the doses and duration of used drugs.

Conclusion:

Rheumatoid Arthritis patients exhibited significantly elevated levels of MDA and reduced levels of CoQ10indicating oxidative stress. Treatment modalities significantly affect these biomarkers. The group with biological therapy alone showed the most favorable profile (highest CoQ10 and lowest MDA levels). While the biological therapy with supportive treatment group exhibited the opposite pattern.

Authors' Declaration:

We confirm that all the Figures and Tables in the manuscript belong to the current study. Besides, the Figures and images, which do not belong to the current study, have been given permission for re-publication attached to the manuscript. Authors sign on ethical consideration's approval-Ethical Clearance: The project was approved by the local ethical committee in the place where their search was conducted or samples collected and treated) according to the code number (18) on (13/4/2025).

Conflict of interest: None

Funding: None

Authors' Contributions:
Study conception &design:(Basil O Saleh, Faiq I Gorial). Literature search: (Nagham Y Mohsin). Data acquisition: (Nagham Y Mohsin). Data analysis & interpretation: (Nagham Y Mohsin & Basil O Saleh). Manuscript preparation:(Basil O Saleh, Faiq I Gorial & Nagham Y Mohsin). Manuscript editing & review: (Basil O Saleh, Faiq I Gorial).

References:

1. Mohammed RH, Bhutta BS. Hand and wrist rheumatoid arthritis [Internet]. StatPearls - NCBI Bookshelf. 2023. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560890/

2.Díaz-González F, Hernández-Hernández MV. Rheumatoid arthritis. Medicina Clínica (English Edition) [Internet]. 2023 Nov 11;161(12):533–42. Available from: https://doi.org/10.1016/j.medcle.2023.07.008

3.Shah P, Trivedi J, Shah P. Pathophysiology of rheumatoid arthritis. International Clinical Case Reports and Reviews [Internet]. 2024 Jun 12;2(1):1–4. Available from: https://doi.org/10.59657/2993-0855.brs.24.010

4.Ebrahimi A, Kamyab A, Hosseini S, Ebrahimi S, Ashkani-Esfahani S. Involvement of coenzyme Q10 in various neurodegenerative and psychiatric diseases. Biochemistry Research International [Internet]. 2023 Nov 1;2023:1–11. Available from: https://doi.org/10.1155/2023/5510874

5.Maciejewska-Stupska K, Czarnecka K, Szymański P. Bioavailability enhancement of coenzyme Q10: An update of novel approaches. Archiv Der Pharmazie [Internet]. 2024 Apr 29; Available from: https://doi.org/10.1002/ardp.202300676

6.Mantle D, Millichap L, Castro-Marrero J, Hargreaves IP. Primary Coenzyme Q10 Deficiency: An Update. Antioxidants (internet). 2023;12(8):1652. https://doi.org/10.3390/antiox12081652

7.Manzar H, Abdulhussein D, Yap TE, Cordeiro MF, Cordeiro MF. Cellular Consequences of Coenzyme Q10 Deficiency in Neurodegeneration of the Retina and Brain. Int J Mol Sci. 2020;21(23):9299. https://doi.org/10.3390/ijms21239299 .

8.Alghafoor A. Total oxidative stress and disease. Ln: Elsevier eBooks; 2023. p. 195-210. https://doi.org/10.1016/B978-0-323-91890-9.00038-6

9.Asfandiyar N, Hadi N, Zaidi IA, Kamal Z, Ashraf N, Khan RU, et al. Estimation of serum malondialdehyde (a marker of oxidative stress) as a predictive biomarker for the severity of coronary artery disease (CAD) and cardiovascular outcomes. Cureus [Internet]. 2024 Sep 19; Available from: https://doi.org/10.7759/cureus.69756

10.Sahlström P, Joshua V, Valkovskaia V, Biese C, Stålesen R, Israelsson L, et al. Autoreactive B cells against malondialdehyde-induced protein cross-links are present in the joint, lung, and bone marrow of rheumatoid arthritis patients. Journal of Biological Chemistry [Internet]. 2023 Oct 4;299(11):105320. Available from: https://doi.org/10.1016/j.jbc.2023.105320

11.Asfandiyar N, Hadi N, Zaidi IA, Kamal Z, Ashraf N, Khan RU, et al. Estimation of serum malondialdehyde (a marker of oxidative stress) as a predictive biomarker for the severity of coronary artery disease (CAD) and cardiovascular outcomes. Cureus [Internet]. 2024 Sep 19; Available from: https://doi.org/10.7759/cureus.69756

12. Aldouri LY, Nader MI, Alosami MH. Evaluating TLR4 Gene Expression to Monitor Disease Progression in Iraqi Patients with Rheumatoid Arthritis. Al-Rafidain J Med Sci [Internet]. 2023 Nov 16 ;5(1S): S136-41. https://doi.org/10.54133/ajms.v5i1S.382

13.Al-Jumaily RMK, AL-Sheakli II, Muhammed HJ, Al-Rubaii BAL. Gene expression of Interleukin-10 and Foxp3 as critical biomarkers in rheumatoid arthritis patients. Biomedicine. 2023;43(4):1183-7. https://doi.org/10.51248/.v43i4.3107

14.Feng X, Xu X, Shi Y, Wang Y, Liu X, Yang F, et al. Body mass index and the risk of rheumatoid arthritis: an updated dose-response meta-analysis. BioMed Res Int. 2019 Jul 2; 2019:3579081. https://doi.org/10.1155/2019/3579081

15.Gamal RM, Mahran SA, Abo El Fetoh N, Janbi F. Quality of life assessment in Egyptian rheumatoid arthritis patients: Relation to clinical features and disease activity. Egypt Rheumatologist 2015;38(2):65-70. https://doi.org/10.1016/j.ejr.2015.04.002

16.Gvozdjáková A, Sumbalová Z, Kucharská J, Szamosová M, Čápová L, Rausová Z, et al. Platelet mitochondrial respiration and coenzyme Q10 could be used as new diagnostic strategy for mitochondrial dysfunction in rheumatoid diseases. PLoS ONE [Internet]. 2021 Sep 28;16(9):e0256135. Available from: https://doi.org/10.1371/journal.pone.0256135

17.Nachvak SM, Alipour B, Mahdavi AM, Aghdashi MA, Abdollahzad H, Pasdar Y, et al. Effects of coenzyme Q10 supplementation on matrix metalloproteinases and DAS-28 in patients with rheumatoid arthritis: a randomized, double-blind, placebo-controlled clinical trial. Clinical Rheumatology [Internet]. 2019 Aug 7;38(12):3367–74. Available from: https://doi.org/10.1007/s10067-019-04723-x

18.Saeed U, Ali M, Mahmood F, Saeed S, Mudassar S, Ahmed J. Lipid peroxidation and detection of Pro-Inflammatory variables in rheumatoid arthritis- A comprehensive analysis of malondialdehyde and isoprostanes in synovial fluids and SERA. Developmental Medico-life-sciences [Internet]. 2024 May 5;1(2):1–7. Available from: https://doi.org/10.69750/dmls.01.02.019

19.de Paz NM, Quevedo-Abeledo JC, Gómez-Bernal F, de Vera-González A, Abreu-González P, Martin-González C, et al. Malondialdehyde Serum Levels in a

Full Characterized Series of 430 Rheumatoid Arthritis Patients. J Clin Med. 2024;13(3):901. https://doi.org/10.3390/jcm13030901

20.Szekanecz Z, Szűcs G, Kerekes G. Antirheumatic Drugs and Cardiovascular Disease in Rheumatoid Arthritis. Rheumatol Forum. 2023;9:49-62. https://doi.org/10.5603/RF.2023.0010

21.Bobek D. Use of Non-Steroidal Anti-Inflammatory Drugs in Patients with Advanced Active Rheumatoid Arthritis. Acta Clin Croat. 2022;61:588. https://doi.org/10.20471/acc.2022.61.04.04

22.Pattison DJ, Winyard PG. Dietary Antioxidants in Inflammatory Arthritis: Do They Have Any Role in Etiology or Therapy? Nat Clin Pract Rheumatol. 2008;4(11):590-6. https://doi.org/10.1038/ncprheum0920

23.Abdollahzad H, Aghdashi MA, Asghari Jafarabadi M, Alipour B. Effects of Coenzyme Q10 Supplementation on Inflammatory Cytokines (TNF-α, IL-6) and Oxidative Stress in Rheumatoid Arthritis Patients: A Randomized Controlled Trial. Arch Med Res. 2015;46(7):527-533. https://doi.org/10.1016/j.arcmed.2015.08.00624.Bilski R, Nuszkiewicz J. Antioxidant therapies as emerging adjuncts in rheumatoid arthritis: Targeting oxidative stress to enhance treatment outcomes. International Journal of Molecular Sciences [Internet]. 2025 Mar 21;26(7):2873. Available from: https://doi.org/10.3390/ijms26072873

25.Hosi SS, Hammo ZSY, Hussein YK, Almahdawi ZMM. Investigating the Role of Oxidative Stress and Immune Response in Rheumatoid Arthritis. Int J Med Toxicol Legal Med [Internet]. 2024;27(3S):443. Available from: https://ijmtlm.org

How to Cite this Article

Mohsin NY, Saleh BO, Gorial FI. Effect of type of treatment (biology and chemotherapy) on serum levels of Coenzyme Q10 and Malondialdehyde in patients with Rheumatoid Arthritis. J Fac Med Baghdad [Internet]. 2025 Jun. 19 [Available from: https://iqjmc.uobaghdad.edu.iq/index.php/19JFacMedBaghdad36/article/view/3145

تأثير نوع العلاج (البيولوجي والعلاج الكيميائي) على مستويات الانزيم المساعد (Q10) والمالونديالديهيد في مصل الدم لدى مرضى التهاب المفاصل الروماتويدي

نغم ياسين محسن[1]، باسل عويد محمد صالح[1]، فائق ايشو كوريال[2]
[1]فرع الكيمياء الحياتية السريرية، كلية الطب، جامعة بغداد، بغداد، العراق.
[2]فرع الطب الباطني، كلية الطب، جامعة بغداد، بغداد، العراق.

الخلاصة:

خلفية البحث: التهاب المفاصل الروماتويدي (RA) هو مرض التهابي مناعي ذاتي مزمن، يلعب الاجهاد التأكسدي، مثل مالونديالديهايد (MDA) دورا حاسما في الفيزيولوجيا المرضية لالتهاب المفاصل الروماتويدي، بينما يلعب مضاد الاكسدة، مثل الانزيم المساعد (CoQ10) دورا وقائيا ضد الالتهاب.

الأهداف: تهدف الدراسة الى مقارنة مستويات (CoQ10) و (MDA) في مصل الدم لدى مرضى التهاب المفاصل الروماتويدي والاصحاء وتقييم كيفية تأثير أنواع مختلفة من العلاج (العلاجات البيولوجية والعلاج الكيميائي) على مستويات (CoQ10) و(MDA) في مصل الدم لدى مرضى التهاب المفاصل الروماتويدي.

المرضى وطرق العمل: أجريت هذه الدراسة المقارنة على 88 فردا (60 منهم تم تشخيص اصابتهم بالتهاب المفاصل الروماتويدي و28 سليما كمجموعة ضابطة).تم سحب العينات من قسم امراض الروماتيزم في مستشفى بغداد التعليمي في بغداد، العراق، خلال الفترة من نوفمبر 2024 الى مارس 2025. تم تقسيم المرضى وفقا لنوع العلاج (البيولوجي، البيولوجي والكيميائي، البيولوجي وغيره، العلاج الكيميائي وغيره). تضمنت فحوصات المصل قياسات الاجسام المضادة للببتيد السيتريليني الحلقي (anti-CCP)، والبروتين التفاعلي سي عالي الحساسية (hs-CRP) و(CoQ10) و(MDA)، باستخدام تقنية (ELISA).

النتائج: وجدت الدراسة ان متوسط (±الانحراف المعياري) لقيم (MDA) قد زاد بشكل ملحوظ (P<0.0001) بينما كان متوسط (CoQ10) اقل بشكل ملحوظ (P<0.0001) في مرضى التهاب المفاصل الروماتويدي، كما كان هناك فرق كبير في مستويات (MDA) و (CoQ10) بين مجموعات العلاج، حيث سجلت المجموعة العلاجية البيولوجية مع المجموعة الأخرى اعلى مستويات (MDA) واقل مستويات (CoQ10).

الاستنتاجات: يظهر مرضى التهاب المفاصل الروماتويدي مستويات مرتفعة بشكل ملحوظ من (MDA) وانخفاضا في مستويات CoQ10، مما يشير الى الاجهاد التأكسدي.تؤثر طرق العلاج بشكل كبير على هذه المؤشرات الحيوية.

الكلمات المفتاحية مضاد للأكسدة، انزيم (Q10) المساعد، التهاب المفاصل الروماتويدي، مالونديالديهيد، الاجهاد التأكسدي.