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Original Article

Internal Medicine Section

Correlation of High-sensitivity C-reactive Protein and Fasting Lipid Profile for Assessing Cardiovascular Risk Factors in patients with Autoimmune Connective Tissue Disorders: A Cross-sectional Study

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ABSTRACT

Introduction: Patients with Autoimmune Connective Tissue Diseases (AICTDs) face an increased cardiovascular risk that traditional risk factors cannot fully explain. Chronic systemic inflammation in these disorders is thought to disrupt lipid metabolism, contributing to accelerated atherosclerosis. Understanding the interplay between inflammation and dyslipidaemia is crucial for improving cardiovascular risk assessment in this population.

Aim: To assess the prevalence of dyslipidaemia and elevated High-sensitivity C-Reactive Protein (hsCRP) in patients with AICTDs and evaluate their correlation as potential markers of cardiovascular risk.

Materials and Methods: This cross-sectional study was conducted at Dr. D. Y. Patil Medical College, Hospital and Research Centre, Pune, Maharashtra, India, from October 2022 to February 2025. A total of 100 patients aged above 18 years, already diagnosed with AICTDs, were included in the study. Comprehensive clinical assessment, serological testing, and measurement of fasting lipid parameters and hsCRP levels were performed. Relationships between lipid parameters, hsCRP, and various clinical variables including disease type, disease duration, Body Mass Index (BMI), and steroid use were analysed. Statistical analysis was performed using Pearson's correlation coefficient. A p-value < 0.05 was considered statistically significant.

Results: The study population consisted of 100 patients, with females comprising 75% of participants and males 25%, and the majority aged between 41 and 60 years. Rheumatoid Arthritis (RA) was the most common diagnosis (46%), followed by Systemic Lupus Erythematosus (SLE) (16%), Sjögren's syndrome (12%), and other AICTDs. Dyslipidaemia was observed in 92% of patients, with elevated triglycerides (64%), high total cholesterol (56%), and elevated Low Density Lipoprotein (LDL) cholesterol (50%) being most common. Elevated hsCRP levels (>3 mg/L) were present in 64% of patients. Significant correlations were observed between hsCRP and lipid parameters: positive correlations with total cholesterol (r-value=0.411, p-value<0.001), triglycerides (r-value=0.224, p-value=0.02), LDL (r-value=0.404, p-value<0.001), and the TC/HDL ratio (r-value=0.394, p-value<0.001), and a negative correlation with High Density Lipoprotein (HDL) (r-value=-0.227, p-value=0.02).

Conclusion: AICTD patients showed a high prevalence of dyslipidaemia and elevated hsCRP, with significant inflammation–lipid correlations. This highlights inflammation-driven dyslipidaemia as a key cardiovascular risk factor, warranting comprehensive risk assessment regardless of disease subtype or duration.

Keywords: Atherosclerosis, Autoimmunity, Biomarkers, Inflammation

INTRODUCTION

The AICTDs represent a complex group of diseases characterised by immune system dysfunction leading to multiorgan involvement and significant morbidity [1]. These disorders, including SLE, RA, and systemic sclerosis, share common pathogenic mechanisms involving chronic inflammation and immune dysregulation. In recent years, there has been growing recognition of the intricate relationship between autoimmunity, inflammation, and cardiovascular risk, with mounting evidence suggesting that patients with AICTDs face an increased risk of cardiovascular complications compared with the general population [2].

The role of lipid metabolism in AICTDs has emerged as an area of significant research interest. Studies have demonstrated that these patients often exhibit distinctive patterns of lipid abnormalities, which may contribute to their elevated cardiovascular risk [3,4]. Traditional risk factors alone do not fully explain the increased cardiovascular morbidity observed in this population, suggesting that disease-specific mechanisms, including chronic inflammation and immune-mediated vascular damage, play crucial roles [5]. hsCRP has become a significant indicator of inflammation and cardiovascular risk [2]. Recent research has shown that hsCRP levels correlate not only with disease activity in autoimmune disorders but also with cardiovascular outcomes [6].

The association between hsCRP levels and lipid profiles in AICTDs reflects the complex interaction between inflammation and lipid metabolism [7]. Treatment strategies for AICTDs can significantly impact both inflammation and lipid metabolism, with corticosteroids commonly used in management known to affect lipid profiles [8]. Recent evidence suggests that early recognition and management of cardiovascular risk factors, including dyslipidaemia, may improve outcomes in patients with AICTDs [9]. Understanding the patterns of lipid abnormalities and their relationship to inflammation in these patients could lead to more targeted therapeutic approaches and improved risk stratification strategies for this vulnerable population.

While previous studies have examined dyslipidaemia or inflammatory markers in AICTDs, most are limited to single diseases like RA or SLE and rarely explore both parameters together [5-9]. Few have evaluated the correlation between hsCRP and lipid profiles

as predictors of cardiovascular risk, especially in diverse AICTD cohorts [1,2]. Additionally, data from the Indian population on this subject remain sparse. Hence, this study was conducted to estimate the prevalence of dyslipidaemia and elevated hsCRP in a mixed AICTD cohort, and to assess correlations between hsCRP and individual lipid parameters (total cholesterol, triglycerides, LDL, HDL, and TC/HDL ratio), and to determine whether these associations are independent of confounding factors such as steroid use and BMI.

MATERIALS AND METHODS

This cross-sectional observational study was conducted at Dr. D.Y. Patil Medical College and Research Centre, Pimpri, Pune, Maharashtra, India from October 2022 to February 2025. The study was approved by the Institutional Ethics Committee (Approval Number: IESC/PGS/2023/17), and written informed consent was obtained from all participants. The study was conducted in compliance with the Declaration of Helsinki and Good Clinical Practice guidelines. Patient confidentiality was maintained throughout the study, and participants retained the right to withdraw at any point without compromising their ongoing medical care.

Inclusion criteria: Adult patients aged 18 years or older diagnosed with AICTDs based on clinical features and laboratory confirmation using Antinuclear Antibodies (ANAs) blot, ANA by immunofluorescence, rheumatoid factor, and anti-CCP antibody tests, who fulfilled the European League Against Rheumatism / American College of Rheumatology (EULAR)/ACR) criteria for diagnosis [10] were included in the study.

Exclusion criteria: Patients younger than 18 years; pregnant individuals; ongoing acute infections; active disease flares; history of smoking or alcohol consumption; history of co-morbidities such as type 2 diabetes mellitus, hypertension, chronic kidney disease, chronic liver disease, or thyroid disorders; and patients currently using lipid-lowering medications; BMI ≥ 30 kg/m_2 were excluded from the study.

Sample size calculation: Based on the proportion of patients with dyslipidaemia reported by Erum U et al., as 0.535, with an acceptable difference of 0.10 and 95% confidence, the minimum sample size was calculated to be 96 using WINPEPI 11.3, and was rounded to 100 patients [11].

Study Procedure

Eligible patients were recruited from the internal medicine and rheumatology outpatient and inpatient services. Each participant meeting the inclusion criteria was informed about the study purpose and procedures, and written informed consent was obtained. Blood samples of approximately 5 mL were collected after an overnight fast of 12 hours and processed within two hours. Investigations included hsCRP by immunoturbidimetry and fasting lipid profile (total cholesterol, triglycerides, HDL, LDL) via enzymatic colourimetric assay. The lipid profile parameters were categorised based on the NCEP ATP III guidelines (National Cholesterol Education Program-Adult Treatment Panel III) as follows [Table/Fig-1] [12].

Lipid parameter	Category	Cut-off range
Total cholesterol	Optimal	< 200 mg/dL
	Borderline high	200-239 mg/dL
	High	≥ 240 mg/dL
Triglycerides	Optimal	< 150 mg/dL
	Borderline high	150-199 mg/dL
	High	200-499 mg/dL
HDL cholesterol	Low	< 40 mg/dL
	Borderline	40-60 mg/dL
	Optimal	> 60 mg/dL
LDL cholesterol	Optimal	< 100 mg/dL
	Near optimal	100-129 mg/dL
	Borderline high	130-159 mg/dL
	High	160-189 mg/dL
	Very high	≥ 190 mg/dL
TC/HDL ratio	Optimal	< 3.5
	Moderate risk	3.5-5
	High risk	>5

[Table/Fig-1]: Lipid profile classification based on NCEP-ATP III guidelines.
NCEP-ATP III: National Cholesterol Education Program - Adult Treatment Panel III

The hsCRP levels were interpreted according to American Heart Association (AHA) / Centres for Disease Control (CDC) guidelines [Table/Fig-2] [13].

hsCRP level (mg/L)	Cardiovascular risk category
< 1	Low risk
1-3	Moderate risk
> 3	High risk

[Table/Fig-2]: hsCRP risk stratification based on AHA/CDC Guidelines.
hsCRP: High sensitivity C-Reactive protein; AHA: American heart association; CDC: Centers for disease control

Data were recorded in structured case report forms covering demographic details, clinical features, laboratory and imaging findings, and treatment history.

STATISTICAL ANALYSIS

Statistical analysis was performed using Statistical Package for the Social Sciences (SPSS) version 16.0. Descriptive statistics were presented as means ± standard deviation for normally distributed variables and medians with interquartile ranges for non normally distributed variables. Categorical variables were expressed as frequencies and percentages. Correlations between lipid profile parameters, hsCRP levels, and disease activity markers were analysed using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 100 AICTD patients were included in the study. The demographic profile showed a female predominance (75%), with the majority of patients aged between 41-60 years (32%), followed by 21-40 years (31%). The mean age of the participants was 46.56±17.41 years. The mean duration of disease was 104.3±59.8 months, and 46% were on corticosteroids.

In the present study, RA was the most prevalent AICTD (46%), followed by SLE (16%), Sjögren's syndrome (12%), and systemic sclerosis (10%) [Table/Fig-3]. Regarding serological markers, 53% of patients were ANA positive, with speckled pattern (33%) being the most common immunofluorescence pattern. Among the specific antinuclear antibodies, anti-dsDNA (28.3%) and anti-Ro/SSA (22.6%) were most prevalent among the 53 positive cases. Rheumatoid factor was positive in 36% of patients, while Anti-cyclic citrullinated peptide (anti-CCP) was positive in only 10% [Table/Fig-4].

A remarkably high prevalence of dyslipidaemia (92%) was observed, with elevated triglycerides (>150 mg/dL) being the most common abnormality (64%), followed by high total cholesterol (>200 mg/dL) in 56% and elevated LDL cholesterol (>130 mg/dL) in 50% of patients. Low HDL cholesterol (<40 mg/dL) was found in 37% of patients, while an elevated TC/HDL ratio (>5) was present in 48%. Regarding inflammation, 64% of patients had hsCRP levels >3 mg/L, indicating significant inflammation, while 19% had intermediate levels (1-3 mg/L) [Table/Fig-5].

No statistically significant differences were observed between steroid users and non users, or between normal BMI and overweight patients, for any of the lipid parameters or hsCRP (all p-values >0.05).

AICTD type	Frequency N (%)	Female N (%)	Male N (%)
Rheumatoid Arthritis (RA)	46 (46)	36 (36)	10 (10)
Systemic Lupus Erythematosus (SLE)	16 (16)	12 (12)	4 (4)
Sjögren's syndrome	12 (12)	9 (9)	3 (3)
Systemic sclerosis	10 (10)	6 (6)	4 (4)
Polymyositis	5 (5)	4 (4)	1 (1)
Dermatomyositis	5 (5)	4 (4)	1 (1)
Mixed connective tissue disease	6 (6)	4 (4)	2 (2)
Total	100	75	25

[Table/Fig-3]: Distribution of AICTD type and gender in study participants (N=100)
AICTD: Autoimmune connective tissue disease.

Serological marker	Positive cases (N)	Negative cases (N)	Total
Anti-nuclear antibody -Anti ds-DNA (28.3%) - Anti Ro/SSA (22.6%) -Others (49.1%)	53	47	100
Rheumatoid factor	36	64	100
Anti-CCP	10	90	100

[Table/Fig-4]: Distribution of serological marker among AICTD patients in the study (N = 100)

Parameter	Category	n (%)
Total cholesterol	Optimal (<200 mg/dL)	44 (44%)
	Borderline high (200-239 mg/dL)	21 (21%)
	High (>240 mg/dL)	35 (35%)
Triglycerides	Optimal (<150 mg/dL)	36 (36%)
	Borderline High (150-199 mg/dL)	24 (24%)
	High (200-499 mg/dL)	40 (40%)
HDL cholesterol	Low (<40 mg/dL)	37 (37%)
	Borderline (40-60 mg/dL)	57 (57%)
	Optimal (>60 mg/dL)	6 (6%)
LDL cholesterol	Optimal (<100 mg/dL)	24 (24%)
	Near Optimal (100-129 mg/dL)	26 (26%)
	Borderline high (130-159 mg/dL)	18 (18%)
	High (160-189 mg/dL)	26 (26%)
	Very high (>190 mg/dL)	6 (6%)
TC/HDL ratio	Optimal (<3.5)	20 (20%)
	Moderate risk (3.5-5)	32 (32%)
	High risk (>5)	48 (48%)
hsCRP	Low (<1 mg/L)	17 (17%)
	Moderate (1-3 mg/L)	19 (19%)
	High (>3 mg/L)	64 (64%)

[Table/Fig-5]: Distribution of lipid parameters and hsCRP Levels among study participants (N=100).
HDL: High density lipoprotein; LDL: Low density lipoprotein; TC/HDL: Total cholesterol/High density lipoprotein; hsCRP: High sensitivity C-reactive protein

This suggests that these factors did not significantly influence lipid metabolism or inflammation in this AICTD cohort. The observations are represented in [Table/Fig-6].

No significant associations were found between disease duration and dyslipidemia (p-value=0.66) or between AICTD type and dyslipidaemia (p-value=0.42). These findings suggest that dyslipidaemia and inflammation in AICTD patients may be independent of specific diagnosis or disease chronicity. There was a strong positive association between hsCRP levels and dyslipidaemia (p-value=0.00013). The [Table/Fig-7] examines the relationship between disease duration, AICTD type, and hsCRP levels with dyslipidaemia status.

Variable	Category	Dyslipidaemia absent (n=8)	Dyslipidaemia present (n=92)	p-value
Disease duration	Short (<60 months)	2 (25%)	25 (27.2%)	0.66
	Medium (60-119 months)	4 (50%)	32 (34.8%)	
	Long (≥120 months)	2 (25%)	35 (38%)	
AICTD Type	Dermatomyositis	0	5 (5.4%)	0.42
	Mixed connective tissue disease	0	6 (6.5%)	
	Polymyositis	1 (12.5%)	4 (4.3%)	
	Rheumatoid Arthritis (RA)	6 (75%)	40 (43.5%)	
	Sjögren's	0	12 (13%)	
	Systemic Lupus Erythematosus (SLE)	0	16 (17.4%)	
	Systemic sclerosis	1 (12.5%)	9 (9.8%)	
hsCRP	Low (<1 mg/L)	0	17	< 0.00013
	Moderate (1-3 mg/L)	6	13	
	High (>3 mg/L)	2	62	

Table/Fig-7]: Relationship between disease duration, AICTD type and hsCRP levels with dyslipidaemia, (N=100).

Statistically significant positive correlations were found between hsCRP and total cholesterol (r-value=0.411, p-value<0.001), triglycerides (r-value=0.224, p-value=0.02), LDL (r-value=0.404, p-value<0.001), and the TC/HDL ratio (r-value=0.394, p-value<0.001). A significant negative correlation was observed with HDL (r-value=-0.227, p-value=0.02). These findings suggest that higher inflammation is associated with a more atherogenic lipid profile in AICTD patients [Table/Fig-8].

DISCUSSION

Present study demonstrated a strikingly high prevalence of dyslipidaemia (92%) among patients with various AICTDs, significantly higher than the reported prevalence in the general adult population (30–40%). This finding aligns with previous research by Tselios K et al., who reported dyslipidaemia in 56.5% of SLE patients; however, present study higher prevalence may reflect differences in study populations, genetic factors, and lifestyle patterns [14].

Parameter	Steroid non users (n=54)	Steroid users (n=46)	p-value	Normal BMI (n=73)	Overweight (n=27)	p-value
Total cholesterol (mg/dL)	218.3±38.7	211.2±38.4	0.36	213.3±38.4	219.6±39.3	0.47
Triglycerides (mg/dL)	191.1±63.1	176.3±64.4	0.25	186.4±65.5	178.4±59.6	0.58
HDL (mg/dL)	43.7±9.7	45.5±8.8	0.35	45.2±9.4	42.7±9.04	0.23
LDL (mg/dL)	135.5±38.01	129.8±39.6	0.46	130.4±38.5	139.6±39.1	0.29
TC/HDL Ratio	5.28±1.7	4.86±1.49	0.19	4.97±1.5	5.41±1.67	0.23
HsCRP (mg/dL)	5.82±4.39	5.90±4.12	0.923	5.79±4.13	6.05±4.56	0.789

Table/Fig-6]: Mean values of lipid parameters by steroid use and BMI category (N=100).
HDL: High density lipoprotein; LDL: Low density lipoprotein; TC/HDL: Total cholesterol/High density lipoprotein

Lipid parameter	Pearson's correlation with hsCRP	p-value
Total cholesterol	0.411	<0.001
Triglycerides	0.224	0.02
HDL	-0.227	0.02
LDL	0.404	<0.001
TC/HDL ratio	0.394	<0.001

[Table/Fig-8]: Correlation of hsCRP with lipid parameters.
HDL: High density lipoprotein; LDL: Low density lipoprotein; TC/HDL: Total cholesterol/High density lipoprotein; hsCRP: High sensitivity C-reactive protein

Similarly, present study found elevated hsCRP levels (>3 mg/L) in 64% of patients, confirming the substantial inflammatory burden in these conditions. The significant correlations between hsCRP and lipid parameters (positive with TC, TG, LDL, and TC/HDL ratio; negative with HDL) underscore the intimate relationship between inflammation and lipid metabolism in AICTDs, supporting the concept of inflammation-driven dyslipidaemia described by Choy E and Sattar N [15]. This inflammation–lipid interplay may contribute significantly to the elevated cardiovascular risk observed in AICTD patients, as proposed by Shoenfeld Y et al., [16].

Interestingly, present study analysis found no significant differences in lipid parameters between steroid users and non users, contrary to the conventional understanding that glucocorticoids adversely affect lipid metabolism. This aligns with findings by Tselios K, et al., who found no significant differences in lipid profiles between SLE patients receiving low-dose prednisone and those not on steroids [14]. Ruiz-Arruza I et al., suggested that the anti-inflammatory effects of low-dose corticosteroids might partially offset their adverse metabolic effects by suppressing inflammation-driven lipid abnormalities [17]. Similarly, present study found no significant associations between disease duration and dyslipidaemia or between AICTD type and lipid parameters, suggesting that lipid abnormalities may develop early in the disease course and persist throughout, regardless of specific diagnosis or disease chronicity. Turesson C and Matteson EL proposed that, despite differences in clinical manifestations, various AICTDs share fundamental pathophysiological mechanisms leading to accelerated atherosclerosis, including endothelial dysfunction, oxidative stress, and autoantibody-mediated vascular damage [18].

The high prevalence of dyslipidaemia and elevated hsCRP in present study cohort highlights the significant cardiovascular risk burden in AICTD patients. Traditional cardiovascular risk assessment tools may underestimate risk in these populations, as suggested by Crowson CS et al., who found that the Framingham Risk Score underestimated the actual risk in RA patients by approximately 100% [19]. Present study findings support the European League Against Rheumatism (EULAR) recommendation of applying a multiplication factor to standard risk scores for inflammatory rheumatic diseases.

The strong correlation between hsCRP and lipid parameters observed in present study study suggests that controlling inflammation might have favourable effects on lipid profiles in AICTD patients, showing improvements in lipid parameters with effective disease-modifying therapies [20]. Additionally, statin therapy may have particular benefits in AICTD patients due to both lipid-lowering and anti-inflammatory effects, as demonstrated in the Trial of Atorvastatin in RA [21]. The table below tabulates and compares the various studies mentioned with the present study [Table/Fig-9] [7,11,15,16].

Limitation(s)

This study had a cross-sectional design rather than a longitudinal one, which limits its ability to infer causality between inflammation and dyslipidaemia in AICTD patients. Additionally, being a single-centre study, the findings may not be generalisable to broader and more diverse populations. Lastly, while the sample size was adequate for overall analyses, subgroup assessments based on specific AICTD subtypes or treatment modalities were limited due to smaller numbers within each category.

CONCLUSION(S)

This study highlights a high prevalence of dyslipidaemia and elevated hsCRP levels among AICTD patients, indicating a substantial cardiovascular risk burden. The lack of significant differences based on steroid use suggests that lipid abnormalities may be intrinsic to the disease process. Strong correlations between hsCRP and lipid parameters reinforce the role of inflammation in dyslipidaemia. These findings support integrating inflammatory markers like hsCRP into routine cardiovascular risk assessment in AICTDs. Effective control of both inflammation and lipid levels may be crucial for reducing long-term cardiovascular complications in this population. Future studies could focus on the development of AICTD-specific cardiovascular risk prediction models incorporating inflammatory markers.

REFERENCES

[1] Mormile I, Osman M, Rossi FW. Prognostic and predictive factors in autoimmune connective tissue disorders. Front Immunol. 2024;15.
[2] Mason JC, Libby P. Cardiovascular disease in patients with chronic inflammation: Mechanisms underlying premature cardiovascular events in rheumatologic conditions. Eur Heart J. 2015;36:482-9.
[3] Giacaglia MB, Pires V, Santana MFM, Passarelli M. Unraveling the pleiotropic role of high-density lipoproteins (HDLs) in autoimmune rheumatic diseases. Int J Rheumatol. 2024;2024:1896817.
[4] Mandel A, Schwarting A, Cavagna L, Triantafyllias K. Novel surrogate markers of cardiovascular risk in the setting of autoimmune rheumatic diseases: Current data and implications for the future. Front Med (Lausanne). 2022;9:820263.
[5] McMahon M, Hahn BH, Skaggs BJ. Systemic lupus erythematosus and cardiovascular disease: Prediction and potential for therapeutic intervention. Expert Rev Clin Immunol. 2011;7:227-41.
[6] Ridker PM, Danielson E, Fonseca FAH, Genest J, Gotto AM, Kastelein JJP, et al. Reduction in C-reactive protein and LDL cholesterol and cardiovascular event rates after initiation of rosuvastatin: A prospective study of the JUPITER trial. Lancet. 2009;373(9670):1175-82.
[7] Tselios K, Sheane BJ, Gladman DD, Urowitz MB. Optimal monitoring for coronary heart disease risk in patients with systemic lupus erythematosus: A systematic review. J Rheumatol. 2016;43:54-65.

Study	Place/Publication year	Population	Dyslipidaemia prevalence	Elevated hsCRP	hsCRP–Lipid correlation	Remarks
Tselios K et al., [7]	Canada/2016	106 SLE patients	56.5%	Not specified	Not clearly assessed	Moderate prevalence of dyslipidaemia, limited data on hsCRP correlation
Erum U et al., [11]	Pakistan/2017	75 RA patients	53.5%	Not assessed	Not assessed	Focused on lipid patterns; no inflammatory marker analysis
Shoenfeld Y et al., [16]	Israel/2005	Multiple AICTDs (review-based)	High	High	Strong link suggested	Review article highlighting immune-driven atherosclerosis
Choy E and Sattar N [15]	United Kingdom/2009	RA and inflammatory states	Variable	Elevated	Inflammation-linked lipid shifts	Conceptual link between cytokine activity and atherogenic lipid profile
Present study	India/2025	100 AICTD patients (RA, SLE, etc.)	92%	64%	Significant correlations (TC, TG, LDL, HDL, TC/HDL)	Supports inflammation-driven dyslipidaemia; independent of steroids or BMI

[Table/Fig-9]: Comparison table of various mentioned studies with the present study [7,11,15,16].
HDL: High density lipoprotein; LDL: Low density lipoprotein; TC/HDL: Total cholesterol/ High density lipoprotein; hsCRP: High sensitivity C-reactive protein; AICTD: Autoimmune connective tissue disease; SLE: Systemic lupus erythematosus; RA: Rheumatoid arthritis; BMI: Body mass index

[8] Bruce IN. Cardiovascular disease in lupus patients: Should all patients be treated with statins and aspirin? Best Pract Res Clin Rheumatol. 2005;19:823-38.
[9] Symmons DPM, Gabriel SE. Epidemiology of CVD in rheumatic disease, with a focus on RA and SLE. Nat Rev Rheumatol. 2011;7:399-408.
[10] Mallawaarachchi K. Autoimmune connective tissue diseases. Atlas Dermatoses Pigment Skin. 2021:563-84.
[11] Erum U, Ahsan T, Khowaja D. Lipid abnormalities in patients with rheumatoid arthritis. Pak J Med Sci. 2017;33:227-30.
[12] Cleeman JI. Executive summary of the third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III). JAMA. 2001;285:2486-97.
[13] Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO, Criqui M, et al. Markers of inflammation and cardiovascular disease: Application to clinical and public health practice. Circulation. 2003;107:499-511.
[14] Tselios K, Koumaras C, Gladman DD, Urowitz MB. Dyslipidemia in systemic lupus erythematosus: Just another comorbidity? Semin Arthritis Rheum. 2016;45:604-10.
[15] Choy E, Sattar N. Interpreting lipid levels in the context of high-grade inflammatory states with a focus on rheumatoid arthritis: A challenge to conventional cardiovascular risk actions. Ann Rheum Dis. 2009;68:460-69.
[16] Shoenfeld Y, Gerli R, Doria A, Matsuura E, Cerinic MM, Ronda N, et al. Accelerated atherosclerosis in autoimmune rheumatic diseases. Circulation. 2005;112:3337-47.
[17] Ruiz-Arruza I, Ugarte A, Cabezas-Rodriguez I, Medina JA, Moran MA, Ruiz-Irastorza G. Glucocorticoids and irreversible damage in patients with systemic lupus erythematosus. Rheumatology (Oxford). 2014;53:1470-76.
[18] Turesson C, Matteson EL. Cardiovascular risk factors, fitness and physical activity in rheumatic diseases. Curr Opin Rheumatol. 2007;19:190-96.
[19] Crowson CS, Matteson EL, Roger VL, Therneau TM, Gabriel SE. Usefulness of risk scores to estimate the risk of cardiovascular disease in patients with rheumatoid arthritis. Am J Cardiol. 2012;110:420-24.
[20] Popa C, Van Den Hoogen FHJ, Radstake TRDJ, Netea MG, Eijsbouts AE, Den Heijer M, et al. Modulation of lipoprotein plasma concentrations during long-term anti-TNF therapy in patients with active rheumatoid arthritis. Ann Rheum Dis. 2007;66:1503-07.
[21] McCarey DW, McInnes IB, Madhok R, Hampson R, Scherbakov O, Ford I, et al. Trial of Atorvastatin in Rheumatoid Arthritis (TARA): Double-blind, randomised placebo-controlled trial. Lancet. 2004;363:2015-21.

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