

Raised Lipid Profile in Rheumatoid Arthritis- A Risk for CVD

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Abstract: Rheumatoid arthritis (RA) is an autoimmune disorder, which causes chronic inflammation of the joints. Patients with newly diagnosed rheumatoid arthritis have adverse serum lipid profile. We sought to determine the effects of treating rheumatoid arthritis with NSAIDs on these abnormal lipid profile.

The study population selected was from the city of Allahabad (U.P.). They were further classified into: Control group consisting of healthy males and females, Group-I consisting of individual suffering from RA and Group II consisting of individual suffering from RA and taking anti-inflammatory drugs. In all subjects a blood sample was collected after an overnight fast, plain vials are used for the determination of lipid profile. Of the blood lipid profile studied i.e. total cholesterol (TC-inc-22.6%), triglyceride (TG-inc-47%), high-density lipoprotein (HDL-inc-37.8%), low density lipoprotein (LDL-inc-55.6%) and very low density lipoprotein (VLDL-inc-12.7%), raised serum values were found in untreated RA patients. On treatment with NSAIDs brought a significant reduction in blood lipid parameters, thereby suggesting a beneficial effect of NSAIDs on the blood lipid profile of RA patients. This improvement may reduce the risk of cardiovascular disease.

Key Words: Autoimmune, Cholesterol, Triglyceride, LDL, VLDL.

I. Introduction:

Rheumatoid arthritis (RA) is a chronic multi system disease of unknown aetiology. The characteristic feature of RA is persistent inflammatory synovitis, usually involving peripheral joints in a symmetric distribution. This synovial inflammation causes cartilage destruction and bone erosions and subsequent changes in joint integrity. The rheumatoid synovium is witness to a complex interplay between a wide variety of cellular, chemical, enzymatic, neutral and genetic elements and is characterized by the presence of a number of secreted products of activated lymphocytes, macrophages and fibroblasts. The local production of these cytokines and chemokines may account for many of the pathological and clinical manifestation of RA⁽¹⁾.

Patients with rheumatoid arthritis (RA) have higher rates of morbidity and mortality than the general population, which is highly attributed to an increased risk of cardiovascular disease (CVD) among RA patients^(2,3). The increased risk of CVD appears to be linked to coronary atherosclerosis^(4,5) and may be directly caused by chronic inflammation or secondarily caused by physical inactivity and drugs used to treat RA⁽⁶⁾.

We have described here a series of routine biochemical investigation i.e., lipid profile, designed to assess whether any changes occur in the serum of patients with rheumatoid arthritis (RA) as compared to normal healthy adults. We have also tried to study the effect anti-inflammatory drugs, have in the normalization of these profile in patients suffering from rheumatoid arthritis.

II. Material And Method :

The study population selected was from the city of Allahabad (U.P.). 150 volunteers, both male and female between 20-50 yrs. of age were taken up for the proposed study. They were further classified into control group, Consisting of healthy males and females, Group-I Consisting of individual suffering from RA and Group II Consisting of individual suffering from RA and taking anti-inflammatory drugs.

For the biochemical parameters to be analyzed, blood sample were drawn from the anticubital vein avoiding venostasis. In all subjects a blood sample was collected after an overnight fast plain vials are used for the determination of lipid profile, Total cholesterol and HDL Cholesterol were measured by Henly's⁽⁷⁾ method. Serum triglyceride was estimated by Rosenberg and Gottfrieds⁽⁸⁾.

III. Statical Analysis :

Values are expressed as mean \pm S.D. The significance of mean difference between groups was analysed by student 't' test and distribution of probability 'p'.

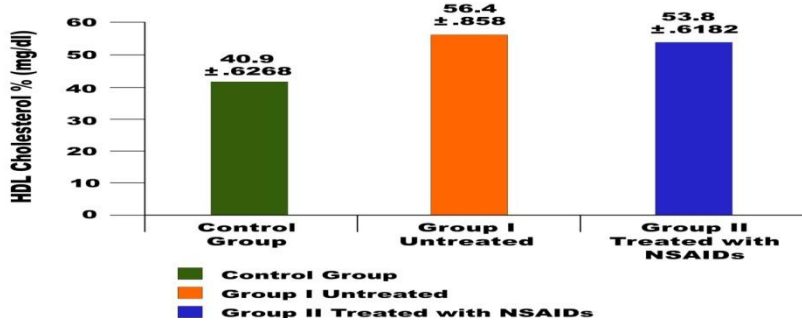
OBSERVATION TABLE :

SN.	PARTICULARS	CONTROL GR.	GROUP-I UNTREATED	GROUP-II-TREATED
	SAMPLE SIZE	50	50	50
	PARAMETERS	Mean \pm SD	Mean \pm SD	Mean \pm SD
1	TOTAL CHOLESTEROL(mg/dl)	178.4 \pm 5.05	218.8 \pm 3.72 (22.6% \uparrow)	183 \pm 3.46 (2.5% \uparrow)

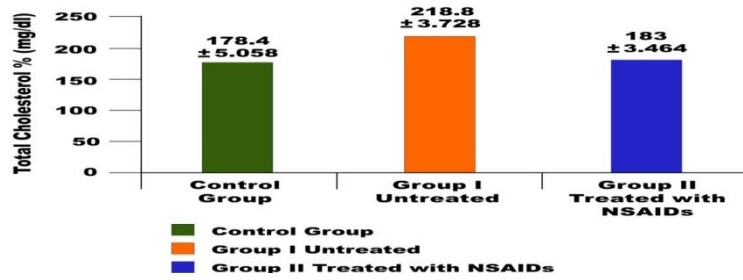
2	HDL-CHOLESTEROL(mg/dl)	40.9±4.99	56.4 ± 1.36 (37.8%↑)	53.8±1.76 (31.5%↑)
3	LDL-CHOLESTEROL(mg/dl)	63.4±0.29	98 ±0.76 (55.6%↑)	85 ±1.41 (34%↑)
4	VLDL-CHOLESTEROL(mg/dl)	27.16±0.29	30.62 ± 0.76(12.7%↑)	29.4±1.41 (12.5%↑)
5	TRIGLYCERIDE(mg/dl)	66±1.96	97.3± 2.56 (47%↑)	89.8±2.62 (36%↑)

GRAPHS:

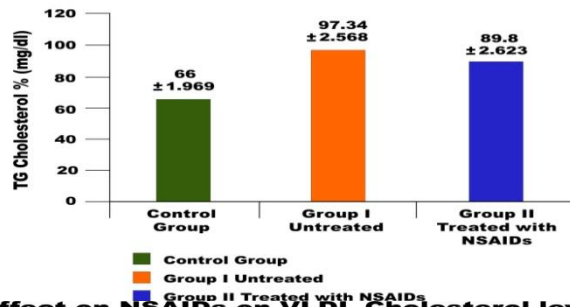
Effect on NSAIDs on HDL Cholesterol level in RA Patients



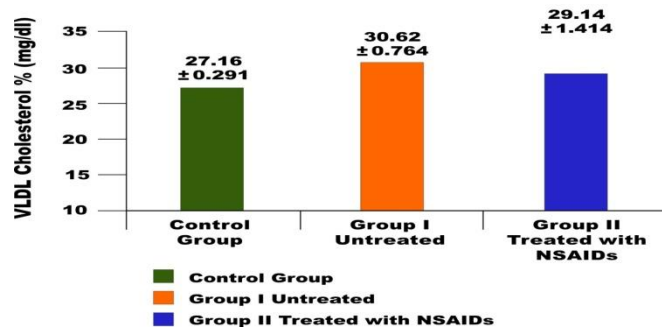
Effect on NSAIDs on Cholesterol level in RA Patients



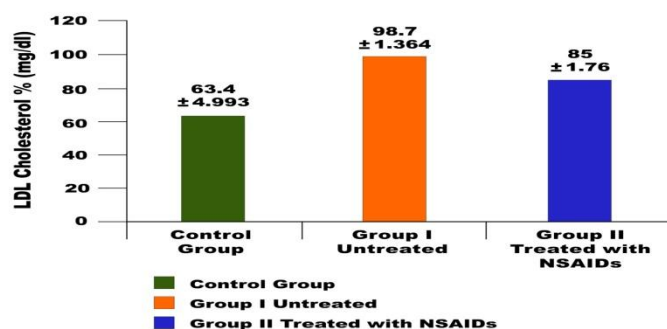
Effect on NSAIDs on Triglyceride Cholesterol level in RA Patients



Effect on NSAIDs on VLDL Cholesterol level in RA Patients



Effect on NSAIDs on LDL Cholesterol level in RA Patients



IV. Result:

Of the Blood lipid profile studied i.e. total cholesterol (TC), triglyceride (TG), high-density lipoprotein (HDL), low density lipoprotein (LDL) and very low density lipoprotein (VLDL), raised serum values were found in untreated RA patients (Group-I). The percentage increase in the lipid profile of these untreated RA patients are as follows; TC=22.6%, TG= 47%, HDL 37-8% LDL= 55.6% and VLDL = 12.7% as compared to healthy controls. On treatment with NSAIDs brought a significant reduction in blood lipid parameters, thereby suggesting a beneficial effect of NSAIDs on the blood lipid profile of RA patients.

V. Discussion:

Lipids consumed in the diet, including fatty acids, cholesterol and fat-soluble vitamins, exert their effects upon the immune system. This might suggest a nutritionally based therapeutic route for disease characterised by inappropriate immune responses (Calder PC, 1998).

Patients with Rheumatoid Arthritis (RA), who by definition manifest persistent high levels of inflammation are at greater risk of developing cardiovascular disease.⁽⁹⁾

Several pieces of evidence indicate that rheumatoid arthritis (RA) is a pro-atherogenic disease associated with increased cardiovascular (CV) mortality^(10,11). Beside genetic and traditional CV risk factors^(12,13), chronic inflammation has emerged as a pivotal component implicated in the development of this process.

Despite some similarities, there are also some differences between patients with chronic inflammatory diseases and the general population. According to Perk J et al (2012)⁽¹⁴⁾, the risk to develop atherosclerosis increases progressively with increasing low density lipoprotein (LDL) cholesterol levels, this is in accordance with our findings which showed marked increase of 37.8% LDL-C, in patient suffering from RA.

Fatty acids are organic acids and form the fundamental constituents of many important lipids, including triglycerides. Their oxidation also brings about raised LDL cholesterol level. Some fatty acids can be synthesized by the body, others the essential fatty acids, must be obtained from the diet. Linoleic acid and α -Linolenic acid are examples of essential fatty acids and much of the work on rheumatoid arthritis and lipids focuses on them and their derivatives. They are the precursors of the two main classes of polyunsaturated fatty acids (PUFA), the omega-3 (or n-3) and omega-6 (or n-6) families. It is the role of these essential fatty acids in inflammation and immunoregulation that has led to the idea that they may be the key to a new approach in treating rheumatoid arthritis (RA). Fatty acids consumed in the diet are metabolised to arachidonic acid. Arachidonic acid is the precursor of eicosanoids (prostaglandins, thromboxanes and prostacyclins) of the 2 series and leukotrienes of the 4 series, which have potent pro-inflammatory and immunoregulatory properties. The arachidonic acid is converted by cyclo-oxygenase enzyme, and series-4 leukotrienes by lipoxygenase enzymes.

The prostaglandins, particularly prostaglandin E₂, produced by arachidonic acid metabolism are inflammatory mediators, contributing significantly to inflammations characterizing symptoms like pain, swelling, heat and redness.

On treating these RA patients with NSAIDs, decreased values were found in general lipid profiles which were statistically significant, the low values of lipid profiles obtained on treatment with NSAID's may be due to a decrease activity of cyclo-oxygenase enzyme which is inhibited by NSAID's, thereby causing less production of prostaglandins.

VI. Conclusion

From our studies on "Raised lipid profile in Rheumatoid Arthritis-A risk for CVD", it can be concluded that rheumatoid arthritis (RA) is accompanied with raised blood lipid profiles. This also showed that in clinical practice RA patients were tested for dyslipidemia less frequently. Due to the increased risk of CVD and mortality among RA patients who have elevated lipid levels. Additional prospective, long term studies are needed to comprehensively determine the role of inflammation and the impact of biologics on lipid levels and CV outcomes in patients with RA.

Supplementation studies with omega-3 and omega -6 fatty acid or Cod liver oil could further be done to help relieve the symptoms of rheumatoid arthritis. As they would inhibit the production of arachidonic acid.

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