CODEN [USA]: IAJPBB ISSN: 2349-7750

INDO AMERICAN JOURNAL OF
PHARMACEUTICAL SCIENCES
http://doi.org/10.5281/zenodo.3758119

Available online at: http://www.iajps.com *Research Article*

DYSLIPIDEMIA: PATHOPHYSIOLOGY, PLASMA ATHEROGENIC INDEX AND EFFECTCS OF GINGKO BILOBA IN EXPERIMENTAL RATS

Farzana Rahim Memon[1], Navaid Kazi[2], Salma Farrukh Memon[3], Ashfaque Rahim Memon[4], Salahuddin Shaik[5], Aftab Hussain Pathan[6]

[1]MBBS, M. Phil(Physiology), Senior Lecturer, Department of Physiology, Isra University, Hyderabad, Sindh, Pakistan

[2]MBBS, M. Phil, Ph. D (Physiology), Professor and Chairman, Department of Physiology, Isra University, Hyderabad, Sindh, Pakistan

[3]MBBS, M. Phil (Physiology), Associate Professor, Department of Physiology, Liaquat University of Medical and Health Sciences, Jamshoro

[4]MBBS, M. Phil (Pharmacology), Professor of Pharmacology, Isra University, Hyderabad, Sindh, Pakistan

[5]MBBS, M. Phil(Physiology), Senior Lecturer, Department of Physiology, Isra University, Hyderabad, Sindh, Pakistan

[6]BSN, Staff Nurse, NICVD NawabShah, Sindh, Pakistan

Article Received: February 2020 **Accepted:** March 2020 **Published:** April 2020

Abstract:

Hyperlipidemia is the root cause of the common cardiovascular diseases like IHD and atherosclerosis globally related to morbidity and mortality. Hyperlipidemia is a controllable disease both through pharmacological and non-pharmacological interventions. Large variety of drugs is available but every class has its advantages and disadvantages Omega-3 fatty acids are also among the currently used drugs with promising results. Ginkgo biloba, an old Chinese herb has drawn attention in clinical research with proven efficacy in multiple disorders. This study evaluated the lipid lowering potential of Ginkgo biloba (GkB) alone as well as combined with Omega-3 fatty acids. Atherogenic index of plasma and serum CRP levels were measured in experimental rats following interventions with high fat diet. 50 Albino male Wistar rats were grouped into 5 equal groups (A, B, C, D, E), group A being the normal control, group B was positive control (High fat diet400mg/kg), group C was administered Omega -3- Fatty Acids 571mg/kg and high fat diet while group D was on G. Biloba 50mg/kg along with high fat diet and Group E:combined mixed therapy of Omega-3-FAs GkB and high fat diet were combined in group E. Experiments were carried out over one month followed by sacrifice and collection of blood samples was done. Lipid profile and CRP were measured at Isra research lab. Atherogenic index was calculated dividing triglycerides with HDL .It was observed that GkB significantly reduced plasma atherogenic index and serum CRP level when compared on ANOVA using SPSS 22nd version difference between serum triglycerides, HDL and atherogenic index was significant with p-values 0.01,0.001 and 0.0001respectively along with CRP with p-value 0.001.

CONCLUSION: *Ginkgo biloba reduces the atherogenic index of plasma as well as serum C-reactive protein levels*

KEY WORDS: *Triglycerides, Gingko Biloba, HDL, Omega-3 Fatty acids, atherogenic index*

Corresponding author:

Dr. Farzana Rahim Memon,

Senior Lecturer, Department of Physiology

Isra University, Hyderabad.

Email: farss05@yahoo.com

QR code

Please cite this article in press Farzana Rahim Memon et al, ***Dyslipidemia: Pathophysiology, Plasma Atherogenic Index And Effectcs Of Gingko Biloba In Experimental Rats.,*** *Indo Am. J. P. Sci, 2020; 07(04).*

INTRODUCTION:

Variety of important functions are Cholesterol dependent in body from cell membrane to synthesis bile acids, steroid hormones and vitamin- D. Continuing provision of this is of high importance for cells to function properly however if its levels remained increased on persistent bases, pathology results that involves vessels. This Deposited fat may cause, Atherosclerosis, Hypertension, Angina, Myocardial infarction, Stroke, Fatty liver, Obesity (peripheral / central), Depression [1-3]. Sources of cholesterol are milk along with its byproducts, Meat, Egg **,** Cooking oils**.** Saturated fat utilization elevates TC (total cholesterol) and LDL-C that prone for developing cardiac illness [4]. Liver regulates the cholesterol in body through continuously synthesizing and managing supply and demand. Cholesterol is also produced by Adrenal glands, GIT, Testis, Ovaries and Placenta. Synthesis of HMG Co-A reductase is controlled by Transcription factor, SREBP-2 (sterol regulatory element binding protein-2) through gene expression. Any reduction in sterol level in cells stimulates gene expression (HMG Co-A reductase) moving SREBP-SCAP complex from ER to Golgi enhancing the synthesis of cholesterol and wise versa if level gets raised[4].AMP activated kinase ,2.phosphoprotein phosphatase both of these regulate the HMG Co-A activity by activating and deactivating on phosphorylation and dephosphorization respectively[4].Enhanced gene expression to synthesize HMG Co-A reductase by Insulin, Thyroxine, Reduction in gene expression by Glucagon and Glucocorticoids are also the regulatory mechanisms[4].Sterol ring present in cholesterol is not metabolized by human so body converts it to bile acids ,Bile salts Smaller proportion goes in feces. The normal total cholesterol serum level is <200mg/dl it needs to be transported with the lipoproteins due to its hydrophobic nature. Low density lipoproteins are responsible for transporting the cholesterol to the peripheral tissues taking away from the liver as 50% of its contents. Cells have surface receptors for LDL. LDL- receptors complex gets entered into cells through endocytosis mediated by Apo-B100 (Apo lipoprotein). LDL is utilized by intracellular lysosomes enzymes as well as chemically alteration of LDL is also observed by macrophage by oxidizing the Apo lipoprotein converting macrophages into foam cells a factor in atherosclerosis. Serum normal value is <130mg/dl but Lost or deficient LDL receptors r elevates the LDL-Cholesterol [4]. HDL-Cholesterol is famously called “good cholesterol” due to its transporting function from periphery (un-esterified cholesterol) back to liver for the synthesis of hormones, VLDL and bile acids .Intestine and liver are the sites of synthesis of HDL-C and Serum levels of HDL-cholesterol should be >35mg/dl under normal conditions in all individuals. Higher serum HDL levels are protective against atherosclerosis and IHD. Triglycerides are exogenous form of lipids being 90% of content of chylomicron which are circulated to body tissues ultimately divided into fatty acid and glycerol, liver utilizes the glycerol part and fatty acids content gets deposited in peripheral tissues. Normally they should exceed 200mg/dl above this is a proven risk for the ischemic cardiac pathologies. Elevated circulating fat in plasma are termed hyperlipidemia having further categorization on the bases of specific content involved [5]. Asia has 182-342/100000, prevalence figure for Ischemic stroke while deaths rate of 3.5/5.5million/Y with underlying cause of dyslipidemia [6]. This work describes the role of Gingko biloba and omega-3-fatty acids on serum CRP levels and the plasma atherogenic index.

METHADOLOGY:

Animals were divided as Group A. Controls, Group B was given high Cholesterol diet (400mg/kg), Group C was administered Ω-3 FA (571 mg/kg) and HFD, Group D was given GkB (50mg/kg) along with HFD and Group E was administered Ω-3 FA (571 mg/kg), GkB (50mg/kg) and HFD. All diet and drugs were given per oral during the experimental period. Animal were sacrificed and blood samples were collected for measuring serum lipids and CRP levels. Data analysis and comparison of variables was done on SPPS version 22 using ANOVA with 0.05 as level of significance. AIP was calculated as log triglycerides/HDL-c while the detailed methodology for lipid profile was taken from previously published literature [7].

RESULTS:

Serum CRP level was 0.102± 0.115 mg/dl in group A, 0.702± 0.255 mg/dl in group-B, 0.659± 0.151 mg/dl in group-C while 0.572± 0.150 mg/dl in group-D and 0.428 ± 0.123 mg/dl in group-E level of significance calculated was 0.001 that is significant. Serum Triglycerides were 91.1+8.32 mg/dl in group-A, 109.45+23.52 mg/dl in group-B, 86.81+18.06 mg/dl in group-C, 100.98+15.18 mg/dl in group-D while 80.27+25.69 mg/dl in group-E with p-value 0.01 that statistically significant. Serum HDL was calculated as 9.54+2.33 mg/dl in group-A, 25.61+6.56 mg/dl in group-B, 25.47+4.78 mg/dl in group-C while 32.98+5.20 mg/dl in group-D and it was measured48.55+7.22 mg/dl in group-E the p-value was 0.001that is significant. AIP in group A was 0.051 ±0.034 while in group-B it was 0.267±0.013 mg/dl, in group-C it was 0.237± 0.024 mg/dl while in group-D as 0.223± 0.019 mg/dl where as in group E it was 0.117 ± 0.041 mg/dl p-value 0.0001[Table-I and Figure,I,II].

Table-1: Comparison of various variables on ANOVA

Parameters	Group-A	Group-B	Group-C	Group-D	Group-E	P-Value
Group Description	Control	High Fat Diet	Ω-3 FA+HFD	GkB+ HFD	GkB+Ω-3 FA+HFD	
Serum CRP	0.102±0.115	0.702±0.255	0.659±0.151	0.572±0.150	0.428±0.123	0.001
Serum Triglycerides	91.1±8.32	109.45±23.52	86.81±18.06	100.98±15.18	80.27±25.69	0.01
Serum HDL	9.54±2.33	25.61±6.56	25.47±4.78	32.98±5.20	48.55±7.22	0.001
Atherogenic Index	0.051±0.034	0.267±0.013	0.237±0.024	0.223±0.019	0.177±0.041	0.0001

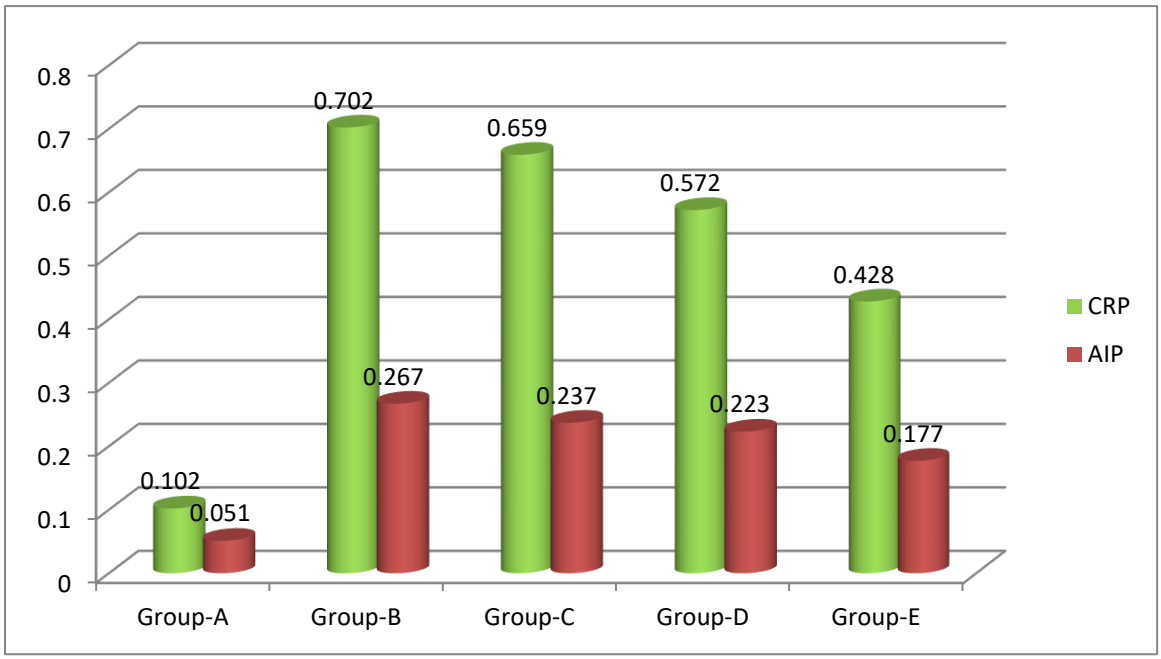

Fig.-1: Representation of serum CRP and Atherogenic index among various groups

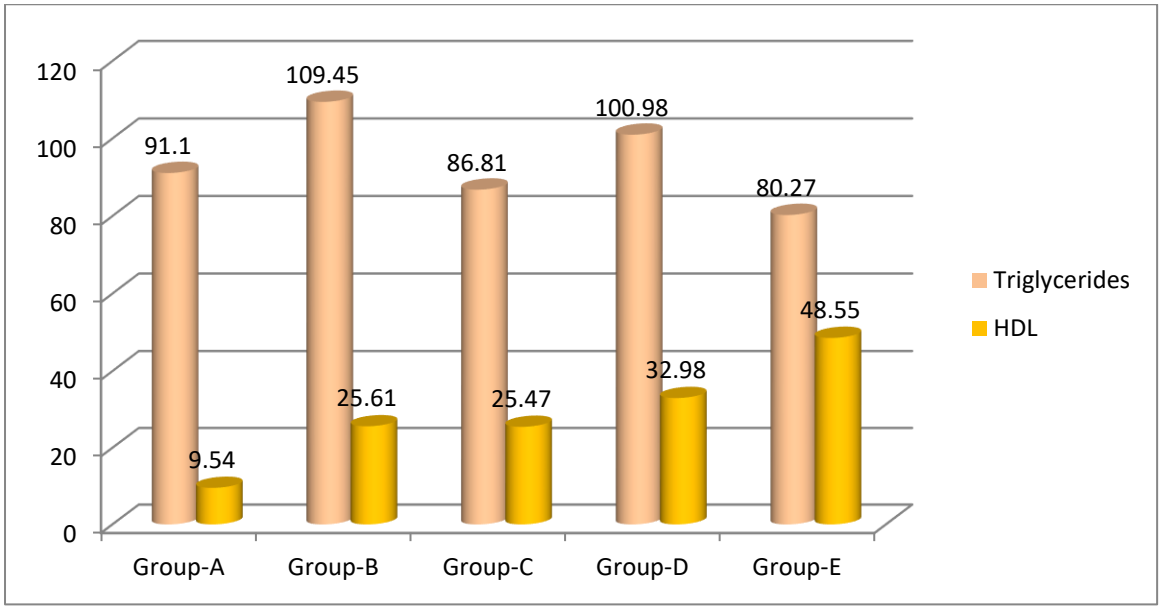

Fig.-II: Serum Triglycerides and serum HDL in different study groups

DISCUSSION:

Lipoprotein lipase deficiency being the core cause results into elevation of TGs, no remedy except reduction in fat intake Type I dyslipidemia: In type IIA hyperlipidemia there is abnormality in synthesis of LDL receptor cause inhibition or reduction in its cellular uptake ↑LDL-C consequently however TGs remain normal ,treatment option are statins Cholestyramine and Niacin in Type IIB dyslipidemia over VLDL production occurs by liver marked by ↑ TGs and VLDL. In Type III dyslipidemia ↑ IDL-C along TGs occurs caused by either IDL-C excessive production or decreased utilization due to apolipoprotein E mutations. Niacin, statins and Fenofibrates are therapeutic answer. Type IV Familial hypertriglyceridemia is associated with ↑VLDL and TGs while ↓LDL or normal possible fault is in VLDL and TGs over production or decreased removal while type V hyperlipidemia manifests with ↑ synthesis or↓ degradation of Chylomicron and VLDL so ultimately chylomicrons, TGs and VLDL get elevated .Drugs used in the Treatment of dyslipidemia are statins, niacin, fibrates, bile acid sequestrants, cholesterol absorption inhibitors, omega-3-fatty acids[8]. Ginkgo biloba (Gb) is among very ancient plants on earth. It has been growing up in forests since 150-200 million years ago the reason for being called as the living fossil. The word ginkgo biloba came from the China meaning a silver fruit. The tree belongs to class Ginkgoatae of the family Ginkgoaceae having strong nerve cells protection potential on deprival of oxygen [9]**.** GB extract is among few Chinese herbs recognized in the international communities for medical use[10]. Its extract is derived using leaves of surviving Ginkgoaceae (Ginkgo biloba family). Perhaps it is enlisted in most famous OTC (over-the-counter) herbal supplements. An estimated glycosides content is 22%–27% while terpenes part accounts for 5%–7% in standard GB extract[11].Studies clue patients of diabetes (Type II) may have beneficial outcomes following Gingko biloba ingestion due to improvement in functions of platelet , interactions with vessel wall as well as reduction in platelet levels of malondialdehyde [12]. Active ingredients of Ginkgo also provide improvement in circulation, formation of clot is also reduced, capillaries walls reinforcement, Dementia, Stroke, Improvement in memory, Alzheimer's disease, Anti-aging, Cognitive speed Free radical injuries scavenger Lipid-regulation in hyperlipidemia [9,13].

CONLUSION:

Ginkgo Biloba reduces the serum CRP level and plasma atherogenic index.

REFERENCES:

1. Alice T.C.R., Kibba Koumare et al (2015). Plasma lipid profile including the high density lipoprotein (HDL), subclasses in hypertensive patients in Ougadougou, Burkina Faso. African Journal of Biochemistry Research 9(3):47-54.
2. Bichitra .N. Patra, Sudhir K(2014). Khandelwal Rakish K. Chadda et al. A controlled study of serum lipid profile in indian patients with depressive episodes. Indian J Psychol Med 36 (2):129-133.
3. Liang Y, Yan Z, Cai et al(2014). Association between lipid profile and depressive symptoms among Chinese older people:mediation by cardiovascular disease. Int J Behav Med 21(4):590-596.
4. Denise R. Ferrier(2014). Nutrition. Lippincott's Biochemistry Wolters Kluwer USA. 6th edition 357-372.
5. Siddiqui NA, Asadullah M(2016). Primary hyperlipidemic patients; differences in lipid profile with original product of simvastatin and herbal product of allium sativum .Prof. Med J 23(5): 564-570.
6. Khalid Sher, Shahnaz Shah and Suneel Kumar(2013). Etiologic Patterns of Ischaemic Stroke in Young Adults.Journal of the College of Physicians and Surgeons Pakistan 23 (7): 472-475.
7. Ashique A A, Abdul R M, Humayion K, Barkat A M (2017). Reduction of Serum Lipid Profile by Escitalopram in Depressive Patients: A Cardio Protective Aspect of SSRI Use. J Cardiol & Cardiovasc Ther 4(4): 555642. DOI: 10.19080/JOCCT.2017.04.555642.
8. Karen Sando(2015). Drugs for Hyperlipidemia. In, Lippincott Illustrated Reviews Pharmacology. Karen Whalen, Wolters Kluwer USA, 6th edition 311-323.
9. Bushra S. Aljadaani, Abrar A. Bakr, Amal H. Hamza(2016). Effect of Ginkgo Biloba and Commiphora Opobalsamum Extracts on Liver Fibrosis and Kidney Injury Induced By Carbon Tetra Chloride in Experimental Models. World J Pharm Sci 4(2): 148-152.
10. .H. Y. Li, L. Cui, and M. Cui(2009). "Hot topics in chinese herbal drugs research documented in pubmed/medline by authors inside china and outside of china in the past 10 years: based on coword cluster analysis," Journal of Alternative and Complementary Medicine, vol. 15, no. 7, pp. 779–785.
11. P.-C. Chan, Q. Xia, and P. P. Fu(2007) "Ginkgo biloba leave extract: biological, medicinal, and toxicological effects," Journal of Environmental Science and Health Part C, vol. 25, no. 3, pp. 211–244.
12. Lei Zhang, Wei Mao, Xinfeng Guo, Yifan Wu, Chuang Li, Zhaoyu Lu,et al(2013).Ginkgo

biloba Extract for Patients with Early Diabetic Nephropathy: A Systematic Review. Evidence-Based Complementary and Alternative Medicine http://dx.doi.org/10.1155/2013/689142.

13. Yao P, Song F, Li K, Zhou S, Liu S, Sun X, et al(2007). Ginkgo biloba extract prevents ethanol induced dyslipidemia. Am J Chin Med 35: 643−52.