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ANTIDYSLIPIDEMIC EFFECT OF *EUGENIA JAMBOLANA* EXTRACT ON HIGH FAT DIET INDUCED DYSLIPIDEMIA IN RATS

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Keywords:

Dyslipidemia, Coronary Artery Disease, Atorvastatin, *Eugenia jambolana*

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ABSTRACT: Antidyslipidemic activity of extract of *Eugenia jambolana* (EJ, Jamun fruit) in adult Charles Foster rats of both sexes was studied and compared with that of Atorvastatin (Atorva). 4 groups were made [Gp 1-Control group (no drug), only high fat diet (HFD); Gp 2- HFD +EJ (evaluation of role of EJ in the prevention of dyslipidemia); Gps 3 & 4-only HFD]. This was the phase of disease production, lasting for 7 weeks, in which we also evaluated the preventive role of EJ. Thereafter, therapy phase started, which lasted for 90 days [Gp 1-Control group (no drug). Only HFD; Gp 3- HFD+ Atorva; Gp 4- HFD+EJ]. Parameters tested were TC (total cholesterol), TG (triglycerides), LDL (low density lipoproteins) and HDL (high density lipoproteins). During the prevention/ disease production phase, EJ brought an improvement in TC level as compared to control group, which was statistically not significant; but improvements in TG, LDL & HDL levels were significant. Moreover, in the therapy phase, EJ brought significant improvement in all the parameters. It means to say, EJ takes some time in the initiation of a significant response- as we know that natural products take some time for the onset of their therapeutic effects. During therapy phase, Atorva was slightly more effective than EJ. Therefore, *Eugenia jambolana*, as a low-cost herbal therapy, offers an inexpensive, safe & effective measure to combat major public health problems *viz* dyslipidemia/ Coronary Artery Disease/ Atherosclerosis.

INTRODUCTION: Globally, the rate of premature deaths from cardiovascular diseases (CVD) is at an all-time high [1]. Over the past 30 years, the number of CVD fatalities in Asia has nearly doubled from 5.6 million to 10.8 million[2]. As dyslipidemia develops, the risk of CVD rises by two times [1]. Currently, in this world, dyslipidemia is to blame for about 4 million deaths from CVD.

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The highest increases in cholesterol levels over the past few years have been observed in Asian countries like India, Indonesia, Thailand, Malaysia and China, which have already overtaken other western countries/continents including Europe and the United States [2].

Dyslipidemia can be brought on by genetic predisposition alone, external causes alone, or a mix of both [3, 4]. While, secondary dyslipidemias are the outcome of the association of risk factors with external variables or other pathologies; primary dyslipidemias are a diverse range of disorders with hereditary, mono or polygenic aetiologies [4, 5]. Total cholesterol (TC), Triglycerides (TG), Low-density lipoprotein (LDL) cholesterol, and High-density

lipoprotein (HDL) cholesterol readings can all vary as a result of dyslipidemias [6, 7]. The Third Report of the Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III, or ATP III) constitutes the National Cholesterol Education Program's (NCEP's) updated clinical guidelines for cholesterol testing and management.

The NCEP periodically produces ATP clinical updates as warranted by advances in the science of cholesterol management. The third (latest) ATP report (NCEP ATP-III) updates the existing recommendations for clinical management of high blood cholesterol. Current criteria for normal and disturbed lipid levels as per NCEP ATP-III have been shown in **Table 1.**

TABLE 1: CHOLESTEROL GUIDELINES BY NCEP ATP-III

Plasma Lipids	Optimal [mg/dL]	Borderline high [mg/dl]	high risk [mg/dL]	very high risk for CAD [mg/dL]
Total	< 200	200- 239	≥ 240	
Cholesterol				
TGs	< 150	150- 199	200- 499	≥ 500
LDL	< 100 (optimal) 100- 129 (> optimal)	130- 159	160- 189	
HDL	≥ 60		< 40	

The prevalence of dyslipidemia in the community nowadays is extremely high. Lipid abnormalities work in concert with metabolic and environmental factors to increase the risk of diabetes, atherosclerosis, and thrombosis; among other metabolic illnesses [7, 8].

Coronary artery disease may (CAD) occur from atherosclerosis. No boundaries related to geography, gender, or socioeconomic status exist for CAD. CAD may be a precursor of ischemic heart disease (IHD). IHD is one of the top causes of death worldwide.

One of the primary methods of atherosclerotic cardiovascular disease (ASCVD) prevention and treatment is lipid-lowering therapy- an area of great complexity and efficacy [9]. Alterations in lipid profile are one of the most common complications in diabetes mellitus and affects ~ 40% of all diabetic patients [10].

Statins have a great risk-to-benefit profile and are one of the most thoroughly researched medication classes in medical history. Notwithstanding, we can't ignore the emphasis on unfavourable side effects such as myalgia and, less frequently, rhabdomyolysis. According to the JUPITER experiment, rosuvastatin medication sped up the onset of type 2 diabetes by 5.5 weeks as compared to individuals receiving a placebo [11].

Eugenia jambolana (EJ. or, *Syzygium cumini*; commonly known as Malabar plum/ black plum or jamun), belonging to the family Myrtaceae is a tall evergreen tree grown in tropical and sub-tropical regions [12].

Scientific classification of EJis as follows [13]:

Class: Magnoliopsida

Family: Myrtales

Genus: *Eugenia*

Species: *Jambolana.*

It has been used for centuries in India to treat diabetes. Its antihyperglycemic effects have been demonstrated in many animal studies [22]. In diabetic rats, a considerable rise in plasma glucose, vitamin E, ceruloplasmin, and lipid peroxides was seen, along with a parallel fall in vitamin C and reduced glutathione levels.

In diabetic rats, the pancreatic antioxidant enzymes' activity was changed.

After the EJ seed kernel and glibenclamide treatment, these changes were nearly restored to normal. Histopathological studies also revealed the protective effect of EJ seed kernel on pancreatic beta-cells. This study showed that Eugenia jambolana seed kernel decreased oxidative stress in diabetic rats, which in turn might be due to its hypoglycemic property [18].

Various parts of the tree such as the bark and leaves have been reported to be used against chronic diarrhoea, dysentery, sore throat, as well as

antibacterial and antiviral infections [In a study, leaves and bark crude extracts showed good antiviral activity against HPAIV H5N1] [29]. Experiments with the flavonoid enriched extract from the seeds have also shown that it decreases the levels of LDL and triglycerides along with a concomitant increase in HDL levels over the untreated diabetic rats [22, 23]. Cumulatively all these observations suggest that usefulness of EJ as an antidyslipidemic agent. Arai *et al* lob served no correction of the lipid metabolism of diabetic rats by treatment with *Eugenia jambolana* fruit is in contrast with certain studies *in-vitro* that made use of extracts of the leaves of *Eugenia uniflora* (a related species of plant) in 70% ethanol and revealed an inhibitory effect on pancreatic lipase activity, which led to reduced hypertriglyceridemia in mice [23]. Another study carried out by Pepato *et al.* with a decoction of *Eugenia jambolana* leaves collected in south east Brazil showed negative results in relation to lipid metabolism [24]. In view of the previous results described above, we have done research on EJ seeds to evaluate its anti-dyslipidemic properties depending on appropriate investigations that employ well-chosen models with adequate controls and are designed to confirm or deny the existence of its effect on lipid profile.

MATERIALS AND METHODS:

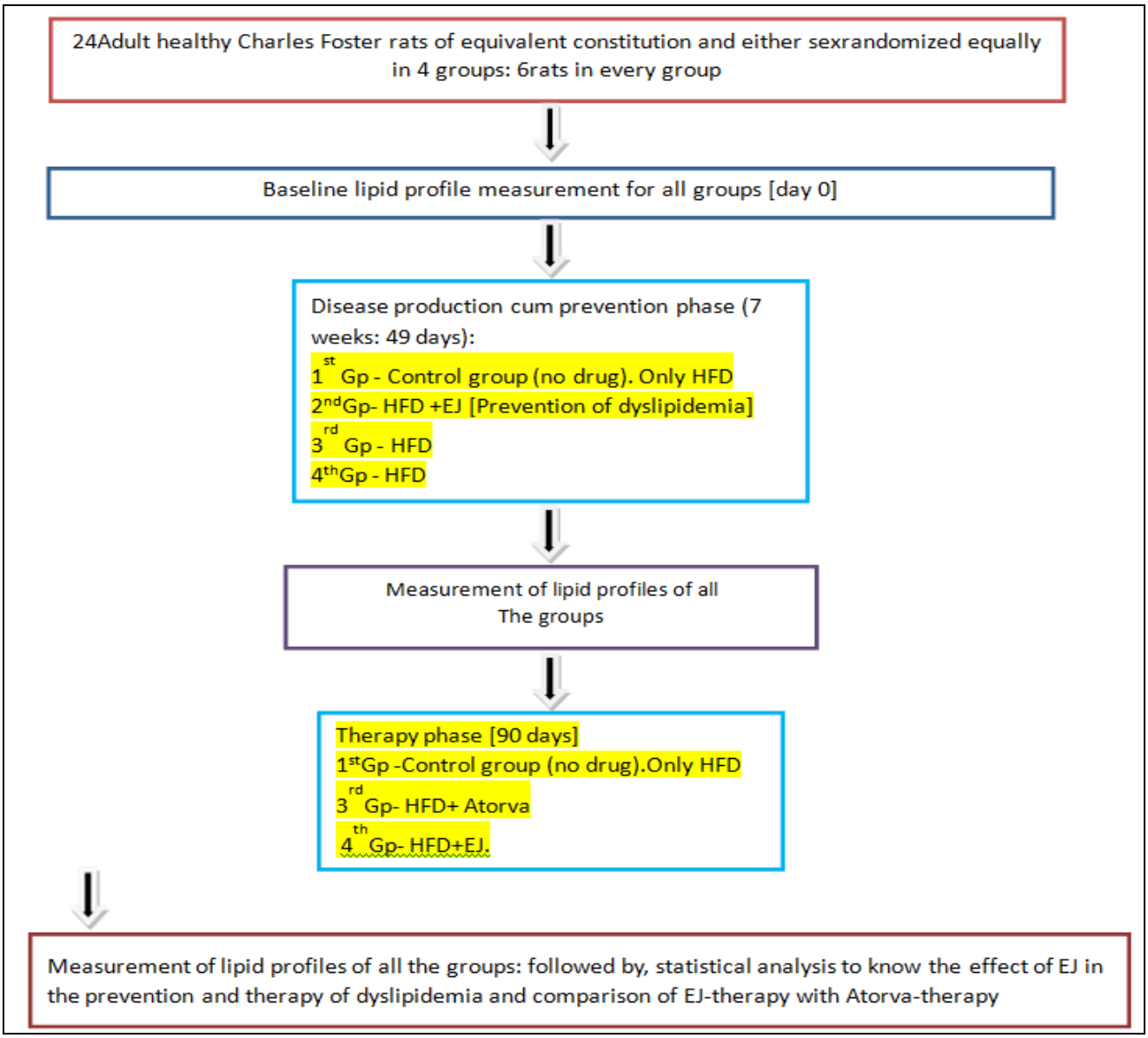

FIG. 1: LAYOUT OF EXPERIMENTAL PROTOCOL. GP: GROUP, HFD: HIGH FAT DIET, ATORVA: ATORVASTATIN, EJ: EUGENIA JAMBOLANA

The Approval for the study was taken from institutional Animal Ethical Committee (IAEC), Banaras Hindu University, Varanasi, Uttar Pradesh (542/GO/ReBi/S/02/CPCSEA dated 26.5.2017) vide letter No. Dean/2022/IAEC/3250. The study procedures were performed as per CCSEA [erstwhile CPCSEA] guidelines. Tablets of Atorvastatin [Atorva] were pulverized and powder thus formed was dissolved in distilled water for oral administration. Extract of EJ was provided by Tansukh herbals Pvt Ltd, Lucknow, UP. Extract was dissolved in distilled water just before oral administration. Drugs were given orally with the help of feeding cannula (oral gavage procedure). High fat diet [HFD] was given for the first 7 weeks [49 days] to produce the required dyslipidemia[25]. Group 2 received EJ also, besides HFD, as we were intended to observe the effect of EJ in the prevention of dyslipidemia. Groups 3 and 4 also received HFD during this phase. Thereafter, Groups 3 and 4 received Atorva and EJ respectively, for next 90 days, i.e. the therapy phase. HFD was continued like before in all the groupstill the completion of the study. Thus, span of the study was 139 [49+90] days.

Drug Doses: Atorva = 80 mg/ kg; EJ= 200 mg/ kg [26, 27, 28].

High Fat Diet (HFD): It was prepared by Dayal Industries Pvt. Ltd., Barabanki Road, Lucknow, Uttar Pradesh, India **Table 2.**

TABLE 2: COMPOSITION OF HIGH FAT DIET

Ingredients	Approximate amount
Crude Fat (Prepared from Rice Bran)	15%
Crude Protein	16%
Acid Insoluble Ash	2.30%
Moisture	8%
Vitamins and Minerals	Appropriate quantity

Measurement of Lipid Profile: Blood samples of 1 ml volume each were taken from retro-orbital plexuses of all the rats at day 0, 49 and 139; for chemical analysis. Serum was separated with the help of proper centrifugation (2000 r.p.m. for 15 minutes). Lipid profile was measured with the help of *semi-autoanalyzer*.

Statistical Analysis: Data were summarized as mean ± SD. Groups were compared by 'two factors (groups × days) analysis of variance (ANOVA)' and the significance of mean difference within and among the groups was done by Tukey post hoctest; and, $p<0.05$ was considered statistically significant. Analyses were performed on SPSS 26.0 (software).

RESULTS & DISCUSSION:

Prevention Phase:

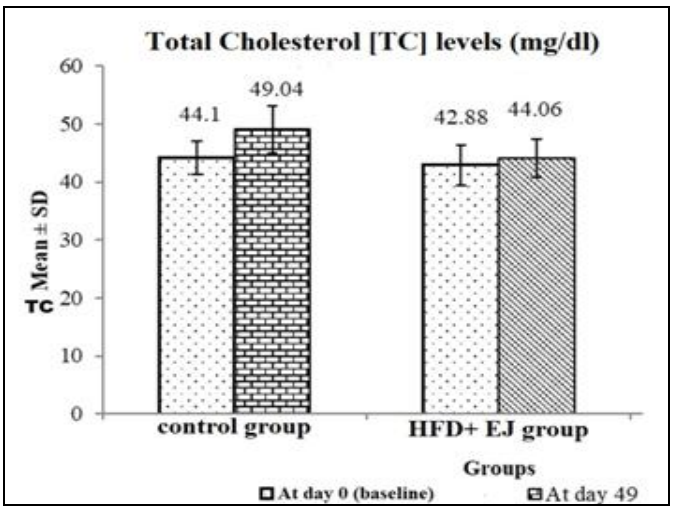

FIG. 2: BAR DIAGRAM SHOWING PRE AND POST TREATMENT MEAN (±SD) TC-LEVELS IN GROUPS 1 & 2 [CONTROL GP & EA+HFD GP] AT DAY 0 AND DAY 49 [PREVENTION PHASE]

Thus, as compared to control group, EJ has caused 10.16% decrease [statistically insignificant, yet a good response] in the TC level after 7 weeks of feeding with HFD.

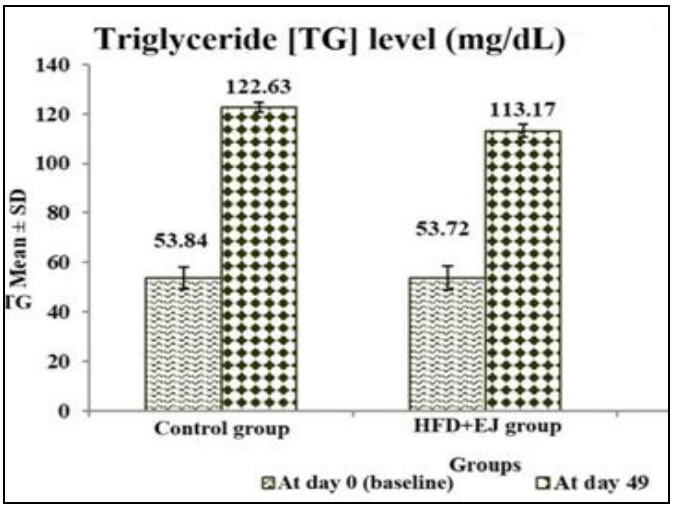

FIG. 3: BAR DIAGRAM SHOWING PRE AND POST TREATMENT MEAN (±SD) TG-LEVELS IN GROUPS 1 & 2 [CONTROL GP& EA+HFD GP] AT DAY 0 AND DAY 49 [PREVENTION PHASE].

Thus, as compared to control group, EJ has caused 7.01% decrease [statistically significant] in the TG level after 7 weeks of feeding with HFD.

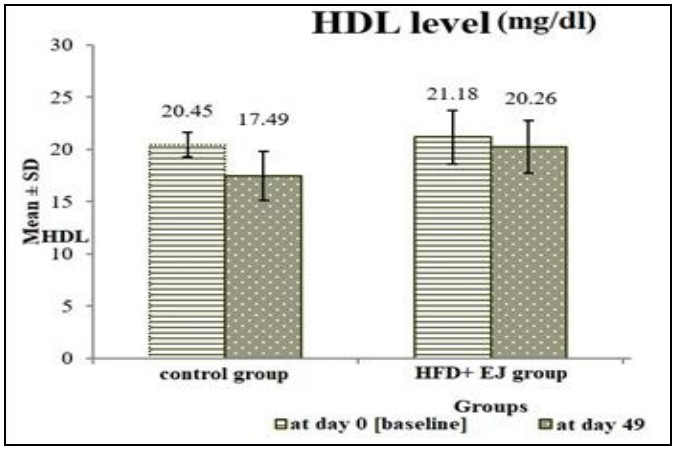

FIG. 4: BAR DIAGRAM SHOWING PRE AND POST TREATMENT MEAN (±SD) HDL-LEVELS IN GROUPS 1 & 2 [CONTROL GP& EA+HFD GP] AT DAY 0 AND DAY 49 [PREVENTION PHASE]

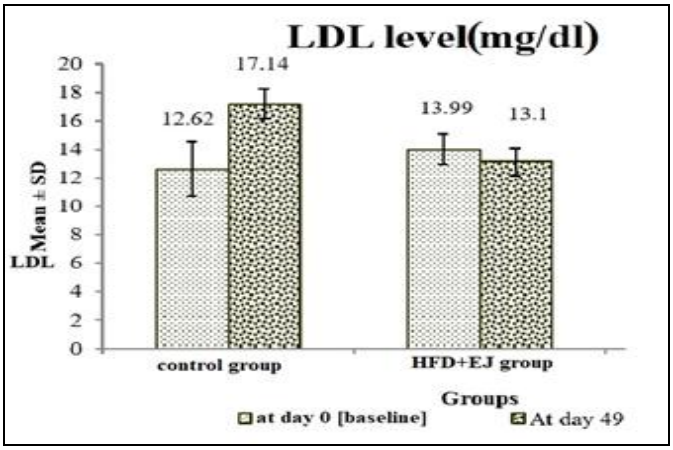

FIG. 5: BAR DIAGRAM SHOWING PRE AND POST TREATMENT MEAN (±SD) LDL-LEVELS IN GROUPS 1 & 2 [CONTROL GP& EA+HFD GP] AT DAY 0 AND DAY 49 [PREVENTION PHASE]

Thus, as compared to control group, EJ has caused 15.83% increase [statistically significant] in the HDL level after 7 weeks of feeding with HFD.

Thus, as compared to control group, EJ has caused 23.57% decrease [statistically significant] in the LDL level after 7 weeks of feeding with HFD.

Therapy Phase:

Effect on TC Levels:

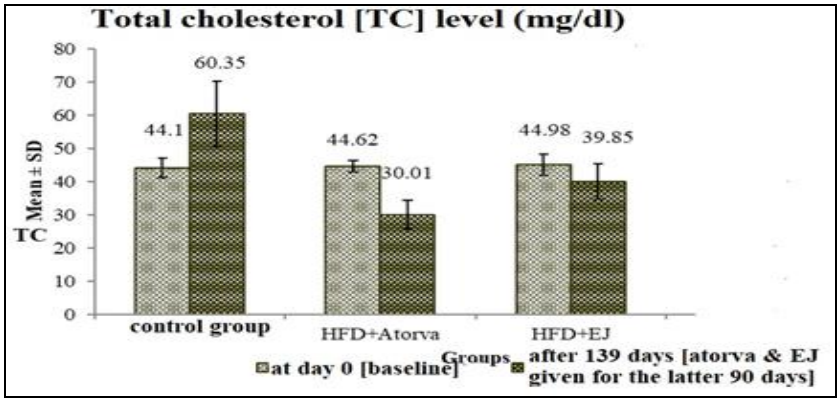

FIG. 6: BAR DIAGRAM SHOWING PRE AND POST TREATMENT MEAN (±SD) TC-LEVELS IN GROUPS 1, 3 & 4 [CONTROL GP, HFD+ ATORVA & HFD+ EJ] AT DAY 0 AND DAY 139 [LATTER 90 DAYS BELONG TO THERAPY PHASE]

Thus, after completion of the therapy phase; as compared to control group, Atorva caused 50.27% and EJ caused 33.96% decrease [both statistically significant] in the TC level.

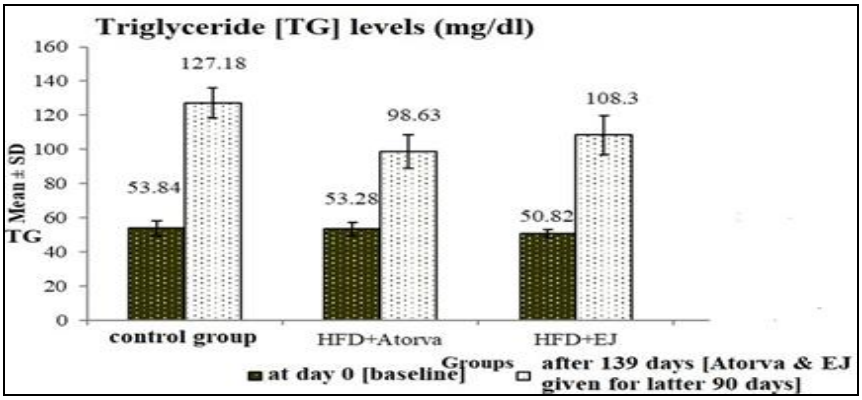

FIG. 7: BAR DIAGRAM SHOWING PRE AND POST TREATMENT MEAN (±SD) TG-LEVELS IN GROUPS 1, 3 & 4 [CONTROL GP, HFD+ ATORVA & HFD+ EJ] AT DAY 0 AND DAY 139 [LATTER 90 DAYS BELONG TO THERAPY PHASE]

Thus, after completion of the therapy phase; as compared to control group, Atorva caused 22.50% and EJ caused 14.84% decrease [both statistically significant] in the TG level.

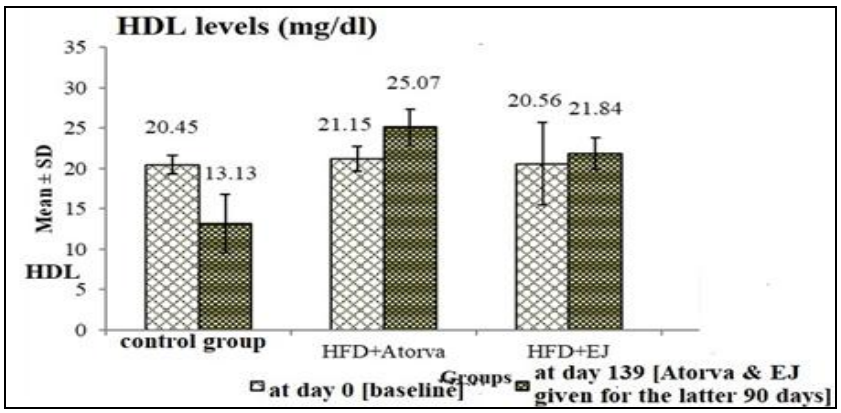

FIG. 8: BAR DIAGRAM SHOWING PRE AND POST TREATMENT MEAN (±SD) HDL-LEVELS IN GROUPS 1, 3 & 4 [CONTROL GP, HFD+ ATORVA & HFD+ EJ] AT DAY 0 AND DAY 139 [LATTER 90 DAYS BELONG TO THERAPY PHASE]

Thus, after completion of the therapy phase; as compared to control group, Atorva caused 90.93% and EJ caused 69.33% increase [both statistically significant] in the TG level.

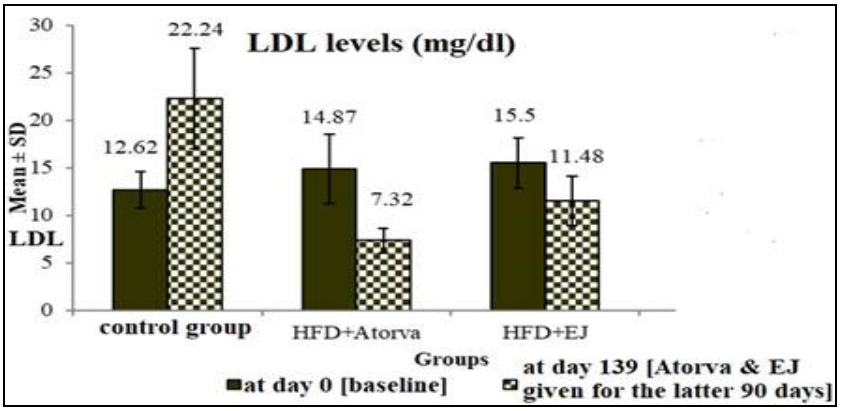

FIG. 9: BAR DIAGRAM SHOWING PRE AND POST TREATMENT MEAN (±SD) LDL-LEVELS IN GROUPS 1, 3 & 4 [CONTROL GP, HFD+ ATORVA& HFD+ EJ] AT DAY 0 AND DAY 139 [LATTER 90 DAYS BELONG TO THERAPY PHASE]

Thus, after completion of the therapy phase; as compared to control group, Atorva caused 67.10% and EJ caused 48.38% decrease [both statistically significant] in the TG level. Thus, Atorva was found to be more effective than EJ. However, both drug options produced significant improvement. During the prevention/ disease production phase, EJ brought an improvement in TC level as compared to control group, which was statistically not significant, but improvements in TG, LDL & HDL levels were significant. Even if the prevention in the rise in TC level has not been statistically significant, overall prevention [in all the 4 parameters] is commendable. Further studies are definitely needed in this regard. Moreover, after the completion of the study, EJ brought significant improvement in all parameters. It means to say, EJ takes some time in the initiation of a significant response- as we know that natural products take some time for the onset of their therapeutic effects. All parts of the EJ can be used medicinally and it has a long tradition in alternative medicine. Different parts of the EJ; especially fruits, seeds and stem bark possess promising activity against various diseases [14]. Jambosine, Gallic acid, Ellagic acid, Corilagin, 3,6-hexahydroxydiphenoylglucose, 1-galloylglucose, 3-galloylglucose, Quercetin, β-sitoterol and 4,6 hexa hydroxy diphenoyl glucose are the important chemical constituents of EJ [15]. By correcting lipid levels in the body, its seeds are used to treat dyslipidemia and diabetic mellitus. This activity is thought to be caused by 3-hydroxyl3-methyl-glutaryl co enzyme A reductase (HMG CoA reductase: an enzyme found in our

body) being blocked [17]. The scavenging of free radicals and protective effect on antioxidant enzymes are attributed to the seeds' high flavonoid content, a well-known antioxidant [18]. In an experiment, Allaxon-induced diabetic rats were given an ethanolic extract of EJ seeds, and this had a significant hypolipidemic effect; lowering the ratio of total serum cholesterol to HDL, serum levels of LDL, and the activity of HMGCoA reductase [21].

Future works are undoubtedly needed to elucidate the concerned mechanisms. Studies have also demonstrated that giving ethanolic extract of the seed kernel to streptozotocin induced diabetic rats resulted in a drop in the levels of cholesterol, phospholipids, triglycerides, and free fatty acids to levels that were close to normal in both plasma and tissues. After providing the extract, the plasma lipoproteins (HDL, LDL and VLDL) and fatty acid content, which were also deranged in diabetic rats, returned to normal levels[22].

Preclinical Safety Profile of EJ: In a toxicity study using in Swiss albino mice, no mortality was recorded in the acute toxicity evaluation up to a dose of 5000 mg/kg bodyweight [BW] of EJ. Median lethal dose [LD_{50}] was assumed to be >5000 mg/kg BW. In the sub chronic toxicity evaluation, no adverse observations were recorded in mice administered with 2000 mg/kg BW of EJ; however at 3000 mg/kg BW dose, moderately significant increase in the plasma levels of urea and creatinine was observed. Hence, the 'Lowest observable adverse effect level (LOAEL)' for EJ was found to be 3000 mg/kg BW and the 'No observable adverse effect level (NOAEL)' was adjudged as 2000 mg/kg BW. Thus, it can be concluded front this study that, orally administered EJ is safe up to a10 fold higher dose than its reported therapeutic dose [30]. In the present study, Atorva was found to be more efficacious than herbal drug, *i.e.* EJ. This difference could be due to use of less-potent doses of EJ (i.e. 200mg/kg of extract), secondly due to short duration of treatment. Though, we started our test drug EJ with lower doses, but it is not so difficult to take this herbal drug in some greater amount, because it is quite free from adverse or toxic effects even at much higher doses. Longer duration is also not a major problem, because if the patient wants to compare, modern drugs are also taken for long period, sometimes lifelong; i.e. more studies with higher doses & longer durations are needed to be performed to evaluate our conclusions.

CONCLUSION: Study reveals that Eugenia Jambolana [Jamun] is significantly effective remedy against dyslipidemia. Extract of this fruit didn'texpress any detrimental effects on experimental animals. Therefore, Eugenia Jambolana, as a low-cost herbal therapy, offers an inexpensive, safe & effective measure to combat major public health problems *viz* dyslipidemia/ CAD/Atherosclerosis. The results of this study support the utilization of Jamun fruit as a dietary intervention strategy and functional food with potential beneficial antidyslipidemic effects.

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CONFLICTS OF INTEREST: Nil

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