RESEARCH ARTICLE

Evaluating the Activity of *Momordica charantia* (Bitter Gourd) Fruit and *Curcuma longa* (Turmeric) Rhizome Extract as Anti-Hyperlipidemic Agents in Experimental Animals

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ABSTRACT

Hyperlipidemia may lead to a defect of blood vessels that raises the risk of atherosclerotic disease. The heightened prevalence of atherosclerosis is often linked to higher blood cholesterol, triglycerides, and low-density lipoprotein levels.

Objectives: To study the effects of extended consumption of dietary bitter gourd and turmeric extract on Triton WR 1339-induced hyperlipidemia in rats.

Methods: Albino Wistar rats were divided into five groups (6 rats/group) and fed specific diets for 21 days: ad libitum control (A), atorvastatin (B; 10 mg/kg body weight), turmeric extract (C; 150 mg/kg body weight), bitter gourd extract (D; 150 mg/kg body weight), and paired-fed extract (E; 150 mg/kg body weight each extract).

Results: Turmeric and bitter gourd extracts significantly reduce alanine transferase, aspartate aminotransferase, LDL cholesterol, triglycerides, and total cholesterol ($p<0.05$) compared to atorvastatin, potentially improving liver enzyme levels through antioxidant activity.

Keywords: Bitter gourd; *Momordica charantia* ; Turmeric; *Curcuma longa*; Hyperlipidemia.

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INTRODUCTION

The increased incidence of atherosclerosis is often associated with elevated serum cholesterol, triglycerides, and LDL levels. The most successful therapeutic strategy towards these disorders is focused on the administration of statins, yet some patients, particularly those with metabolic syndrome, fail to achieve their prescribed statin treatment for LDL targets, moreover, it can cause significant other side effect[1].

Turmeric (*Curcuma longa*), belonging to the Zingiberaceae family, mostly resides in the tropical areas of South and Southwest Asia. It is essential to the culinary traditions of Iran, Malaysia, India, China, and Thailand, serving as a spice that influences the texture, color, and flavor of various dishes. Furthermore, it has been employed for millennia in India and China for the therapeutic treatment of conditions, including dermatological issues, illnesses, stress, and depression[2]. Turmeric's health effects are largely attributed to curcumin, an orange-yellow polyphenol extracted from its rhizomes[2]. Studies of risk evaluation proved that curcumin and turmeric are well tolerated without any toxic effects at a very high dose. Therefore, for the treatment of hyperlipidemia, both curcumin and turmeric can develop modern antihyperlipidemic medicines[3]. Turmeric powder has a significant effect on LDL and VLDL on total liver cholesterol levels and plasma with an increase in the levels of a-tocopherol in rat plasma, indicating an *in-vivo* association with turmeric and a-tocopherol which increase vitamin E bioavailability and decrease levels of cholesterol[4].

Curcumin engages with adipocytes, pancreatic cells, hepatic stellate cells, and macrophages, and has been investigated as a treatment for obesity and associated metabolic problems[5,6]. Furthermore, it affects gut microbiota, with reductions in *Spirochaetae, Tenericutes*, and *Elusimicrobia* at the phylum level observed through high-throughput sequencing, while an increase in Actinobacteria was noted following curcumin administration[7,8].

Bitter gourd (*Momordica charantia*) is an important vegetable in southern and eastern Asia, as well as tropical Africa. The indigenous bitter gourd species are on the verge of extinction due to the proliferation of commercially cultivated varieties. Nevertheless, they are sometimes harvested as vegetables or for medicinal

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Table 1: Ethanolic extract antioxidant activity: DPPH and β-carotene assays.

Extract	µg/ml IC_{50} (DPPH radical)	µg/ml IC_{50} (β-carotene bleaching assay)
Bitter gourd Extract	193.6 ± 2.2	69.55 ± 2.35
Turmeric extract	56.2 ± 1.6	30.50 ± 1.09
Ascorbic acid	2.89 ± 0.08	-
Rutin	-	3.89 ± 0.59

purposes[9]. *M. charantia* exceeds other cucurbits in nutritional value because of its elevated mineral content, especially iron, and vitamin content, particularly ascorbic acid. The bitterness is ascribed to the non-toxic alkaloid momordicin[9]. Unripe *M. charantia* fruits can be utilized by numerous methods, such as frying, boiling, pickling, drying, or canning (e.g., for curries)[10,11]. Bitter gourd extracts contain proteins with anticancer, antiviral, and immunotoxin characteristics. Charantin, vicine, and polypeptide-p are key hypoglycemic compounds, where polypeptide-p being an insulin-mimetic protein synthesized in the fruit, seeds, and tissues, and like bovine insulin[12]. The resulting reduction of plasma (LDL) without doubt is one of the statins benefit gold standard.

This is an unquestionable therapeutic value. Furthermore, the potentially beneficial pleiotropic effects of statins are of great interest[13,14]. Although statins were promoted by their pharmaceutical producers as entirely safe wonder medications, there are use concerns that are insufficiently disclosed in promotional materials and some clinical research. Several specialists and pharmaceutical businesses want to enhance the quality of statins via over the counter (OTC) medications[3,15]. Numerous studies reported the occurrence of statin related myopathy[16]. In 2002, it has been reported that the death incidence of rhabdomyolysis was 0.15 per 1.0 million statin prescriptions associated with severe future rhabdomyolysis.

This rhabdomyolysis may also advance considerably within weeks or fewer[17]. Availability of OTC statins for patients at risk of adherence issues and without regular medical supervision can cause substantial harm, especially from rhabdomyolysis, which may go unnoticed until effective therapy is no longer feasible[18,19]. Neurological and neurocognitive consequences, hepatotoxicity, renal failure, and other complications are among the numerous substantial adverse effects of statin medication[20]. As shown by statin-associated muscle symptoms deficiency or sensitivity to statin most commonly occurs[21]. Statin toxicity presumably results from the inhibition of HMG-CoA reductase, direct cellular effects, subcellular effects, or a combination of these factors[22]. Likely causes including genetic susceptibility, medication interactions, and metabolic or immunological variables, including vitamin D levels[23]. So, one of our goals is to identify safer, fewer toxic herbals that demonstrate the same effect as statin and may be taken naturally with little adverse effect. This study investigated the hypolipidemic and *in vitro* antioxidant properties of bitter gourd and curcumin extracts in rats, utilising atorvastatin as a positive control, and evaluated their influence on biochemical indicators of liver and kidney function.

MATERIALS AND METHODS

Bitter gourd Fruits were purchased from local markets, which were brought from India, while turmeric (*Curcuma longa* L.) was procured from local markets. The identification of fruits and dried rhizomes was carried out. Triton WR1339, diphenyl-1-picryl-hydrazyl (DPPH), β-carotene, rutin, ascorbic acid, chloroform, ethanol, methanol, acetonitrile, and other chemicals of analytical grades were purchased from Sigma-Aldrich USA. Calibrator 18011 for chemistry analyzer, Calibrator HDL/LDL (11693), HDL direct kit, LDL direct kit, and TG direct were procured from Biosystem, Spain.

Animal Studies

Male New Zealand animals were treated humanely according to established guidelines to minimise pain and suffering. Housed in a cross-ventilated shelter under controlled conditions (25±2°C, 44-56% relative humidity, 12:12 light-dark cycle), they received daily food and water. Localised ocular inflammation was noted during the trial; however, no animals were sacrificed.

Experimental Groups

Animals were categorized according to their average biochemical profiles and classified into five groups (n=6 male Wistar albino rats per group). Over a duration of 21 days, mice were subjected to oral administration by

Table 2: Baseline biochemical parameters values of different groups before Triton injection

Parameters	*Group A* Negative control	*Group B* Atorvastatin, (10mg/kg b. wt.)	*Group C* Turmeric extract (150 mg/kg b. wt.)	*Group D* Bitter guard extract (150 mg/kg b. wt.)	*Group E* Turmeric and Bitter guard extract (150 mg/kg b. wt.)
ALT	65.80 ± 5.47	65.43 ± 4.28	66.80 ± 2.91	56.90 ± 3.96	50.90 ± 1.57*
AST	148.63 ± 3.26	153.03 ± 2.65	150.33 ± 5.08	154.43 ± 6.41	147.63 ± 5.81
HDL	23.63 ± 2.45	29.00 ± 0.87	33.10 ± 2.70	24.43 ± 2.93	27.27 ± 3.70
LDL	26.00 ± 1.25	20.2 ± 0.87	23.47 ± 0.67	23.83 ± 0.65	20.00 ± 0.30*
TC	62.67 ± 5.51	50.33 ± 5.03	53.00 ± 4.36	56.00 ± 4.36	49.00 ± 5.00*
TG	92.67 ± 5.03	86.33 ± 2.52	84.00 ± 5.00	87.33 ± 3.51	81.00 ± 3.61
Cr	0.37 ± 0.09	0.26 ± 0.04	0.18 ± 0.04	0.20 ± 0.05	0.42 ± 0.12
Glucose	79.19 ± 4.34	76.68 ± 1.79	83.07 ± 3.05	84.57 ± 4.66	90.33 ± 4.24

The significant difference between Group A (negative control) and the other is denoted by *($p<0.05$)

Table 3: Biochemical parameters value of different groups after the exposure to Triton WR1339 challenge

Parameters	Group A Negative control	Group B Atorvastatin, (10mg/kg b. wt.)	Group C Turmeric extract (150 mg/kg b. wt.)	Group D Bitter guard extract (150 mg/kg b. wt.)	Group E Turmeric and Bitter guard extract (150 mg/kg b. wt.)
ALT	114.53 ± 3.17	123.3 ± 2.96*	75.63 ± 3.32*#	75.57 ± 4.47*#	68.68 ± 6.88*#
AST	267.65 ± 2.25	291.47 ± 4.06	162.85 ± 4.89*#	170.42 ± 2.05*	150.62 ± 2.57*
HDL	28.65 ± 3.64	38.22 ± 3.70*	37.5 ± 6.9*	39.12 ± 5.28*	31.15 ± 4.5
LDL	152.17 ± 4.07	32.17 ± 2.93*	31.67 ± 4.27*	32.00 ± 3.03*	26.17 ± 1.72*
TC	85.5 ± 6.25	51.83 ± 2.79*	54.67 ± 3.67*	46.50 ± 4.51*	42.5 ± 5.36*
TG	171.50 ± 5.32	99.67 ± 6.5*	113.83 ± 5.98*#	115.83 ± 5.71*#	91.67 ± 3.33*
Cr	0.45 ± 0.07	0.50 ± 0.08	0.49 ± 0.12	0.43 ± 0.06	0.32 ± 0.03*
Glucose	143.67 ± 7.86	135.06 ± 1.79	128.99 ± 7.39*	135.92 ± 9.13	101.04 ± 6.97*

The significant difference between Group A (negative control) and the other is denoted by *($p<0.05$)
The significant difference between group B and the other treatment group is denoted by #($p<0.05$).

gavage of either normal saline (Group A, 1 mL/kg), atorvastatin (Group B, 10 mg/kg), turmeric extract (Group C, 150 mg/kg), bitter gourd extract (Group D, 150 mg/kg), or a mixture of both extracts (Group E, 150 mg/kg each). Extracts were solubilized in 0.02% w/v Tween 80 in normal saline using ultrasonication. Baseline values of HDL, LDL, TG, TC, glucose, creatinine, ALT, and AST were evaluated using a chemical analyzer after first blood extraction from the retro-orbital plexusRats labeled with yellow dye to identify them as head (H), tail (T), back (B), head-back (HB), head-tail (HT), and upper right (RU).

Extraction of Bitter Gourd (Momordica charantia)

Bitter gourd fruits (8 kg) were sliced, air-dried for 24 hours, and further dried in a vacuum oven for 8 hours. Seeds were then removed, and the dried material was ground into a fine powder. The powder was sieved and extracted with chloroform: ethanol (30:70) in a conical flask to obtain both lipophilic and hydrophilic compounds. Each extraction used 50 g of powder with 400 ml of solvent, followed by sonication for 1 hour, then filtered. The pooled extracts concentrated via rotary evaporator at 50°C, with vacuum decreasing from 350 to 200 mbar. The residual material was re-extracted three times with 400 ml of solvent, followed by filtration and rotary evaporation. The final extract was dried and stored at (2–8°C) in a stoppered conical flask.

Extraction of Turmeric (Curcuma longa L.)

Extract of Turmeric is performed using 100 g of powder in three different batches. Ethanol (70%) was used as a solvent to extract the different compounds from the turmeric powder. A rotary evaporator was used to evaporate the solvent (70% ethanol). The final extract was dried via rotary evaporator. The vacuum was initially set to 350 mbar, then decreased to 200 mbar to evaporate ethanol and water. The residual material from the extracted powder was re-extracted with the same solvents three times and processed as mentioned aboveThe extract was then placed in a vacuum oven for 24 hours at 50°C and 200 mbar, then kept in a stoppered conical flask in the refrigerator at 2-8°C for future use.

Induction of Hyperlipidaemia

The experiment started with thirty Wistar male albino rats (average weight 250 ±20 g). Their weights are measured to calculate the Triton WR1339 dose needed to induce hyperlipidemia. According to the reported methods, the optimal dose of Triton WR1339 (300 mg/kg) was selected. A stock solution of Triton WR1339 at 300 mg/mL was created by dissolving 3 g in 10 mL of warm saline. Rats had a 24-hour fasting period before receiving an intraperitoneal injection of Triton WR1339, with the dosage calibrated according to body weight (kg)[24].

Preparation of Extract and Atorvastatin Solution

The required quantity of the extract was prepared for administration to animals (for 28 days). Bitter guard extract and turmeric extract (10 g for each) were suspended separately with the help of tween 80 (0.02%) and HPMC (0.05%) in 50 ml of normal saline. These extracts were stored in the refrigerator under nitrogen till further use. A solution of atorvastatin (10 mg/ml) was produced by dissolving 500 mg of atorvastatin in 50 ml of normal saline and thereafter kept under nitrogen in a refrigerated environment.

Blood Sampling

Four hundred µl of blood samples were obtained from the retro-orbital plexus after a 24 hrs. Serum was isolated by centrifugation and evaluated for total cholesterol, LDL, HDL, triglycerides, creatinine, glucose, AST, and ALT using a chemical analyzer. Statistical analysis was conducted with ANOVA (GraphPad Instat, USA).

In-vitro Antioxidant Studies

Ethanolic and methanolic extracts were evaluated for antioxidant activity using two methods. Samples of bitter gourd and turmeric extract at different concentrations were assessed for absorbance using a multi-mode ELISA reader (Synergy HTX).

DPPH Radical Scavenging Activity

IC_{50} estimates antioxidant activity as the concentration necessary to decrease DPPH concentration by 50%[25]. A 0.002% (w/v) DPPH stock solution in methanol was prepared. Extracted samples serially diluted in methanol with concentrations between 4-500 µg/ml. DPPH solution (150 µl) was mixed with 150 µl extract (various concentrations) in an ELISA plate and incubated in the dark (30 min). Absorbance was read at 517 nm, and antioxidant activity was compared to ascorbic acid (0.2–25 µg/ml). This test evaluates hydrogen atom donation, electron donation, and the compound's antioxidant mechanism[26].

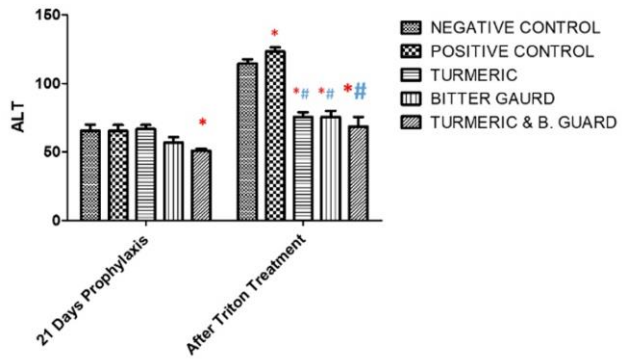

Figure 1: Medication effects on ALT levels before and after Triton 1339 WR

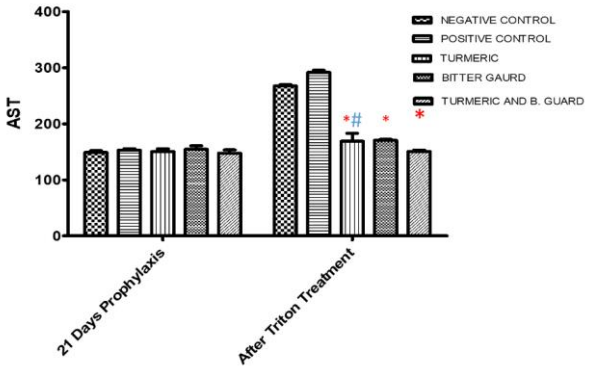

Figure 2: Medication effects on the AST level, during prophylaxis and after Triton 1339 WR

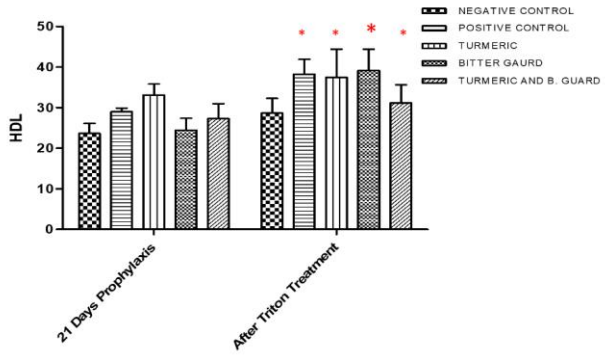

Figure 3*:* Medication effects on the HDL level, during prophylaxis and after Triton 1339 WR

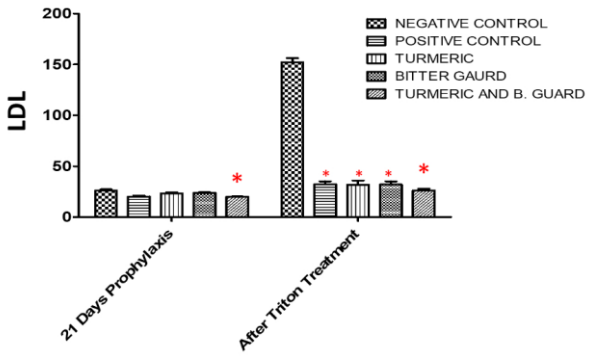

Figure 4: Medication effects on the LDL level, during prophylaxis and after Triton 1339 WR

β-Carotene Bleaching Assay

β-Carotene stock (10 mg/100 ml chloroform) was prepared. 3 ml stock, 25 mg linolenic acid, and 215 mg Tween 20 were added to a flask, and chloroform was evaporated under N2. The residue was reconstituted in 75 ml water. Absorbance was measured at 470 nm and 700 nm. Rutin standard (300 µg/mL, 3 mg/10 ml methanol) was serially diluted (0.6-19 µg/mL and 15-500 µg/mL). For ELISA, 275 µL β-carotene/linolenic acid solution plus 25 µL extract, methanol (control), or rutin standard were added to wells. All solutions were incubated at 50 °C for 1 hour. Absorbance was quantified at 470 nm and 700 nm at 0, 1, and 2 hours.

Statistical Analysis

Statistical analysis was performed using Design-Expert 8.0.6 (Statease, USA) and MODDE 12.1 (Sartorius Inc., USA). Analysis of variance (ANOVA) was used, with significance defined as $P < 0.05$. Data are expressed as mean ± standard deviation.

RESULTS

Yield of Extraction

The yield of turmeric extract and bitter gourd extract was 2% and 3%, respectively, using dried powder of turmeric rhizome and bitter gourd.

In-vitro antioxidant activities

Table 1 illustrates that bitter gourd, and turmeric extracts have notable DPPH free radical scavenging activity, with IC_{50} values of 193.6 ± 2.2 µg/ml and 56.2 ± 1.6 µg/ml, respectively. The IC_{50} value for ascorbic acid was 2.89 ± 0.08 µg/ml. The beta-carotene bleaching (BCB) experiment demonstrates that both extracts effectively inhibited the oxidation of β-carotene. The IC_{50} values for bitter guard and turmeric extract were 69.55 ± 2.35 and 30.50 ± 1.09 µg/ml, respectively, but the IC_{50} value for rutin was 3.89 ± 0.59 µg/ml in the inhibition of beta-carotene oxidation. The extract demonstrated the capacity to operate as a hydrogen atom donor, transforming the purple hue of DDPH into the yellowish DPPH-H form.

In-vivo studies

Baseline biochemical values of 5 main groups before Triton administration are shown in Table 2. After 21 days of feeding the extract, the biochemical parameters were measured: mean serum level (ALT, AST, HDL, LDL, TC, TG, creatinine, and glucose) in each group studied (Table 3) show the mean serum levels of biochemical parameters. After the injection of Triton WR1339, the biochemical parameters were measured again, which are mentioned in Table 3. The mean and standard deviation were calculated and mentioned. Table 2 shows that a combination of turmeric and bitter gourd extract (group E) significantly lowered LDL, total cholesterol, and ALT levels compared to group A (negative control), while the other parameters did not change. Biochemical alterations were not observed in the negative control (group A), positive control (group B), or other groups. Biochemical characteristics of Triton WR 1339 challenge are shown in Table 3. In comparison to group A, group B exhibited markedly elevated ALT levels (Fig. 1), whereas groups C, D, and particularly E had dramatically reduced ALT levels. Group A exhibited

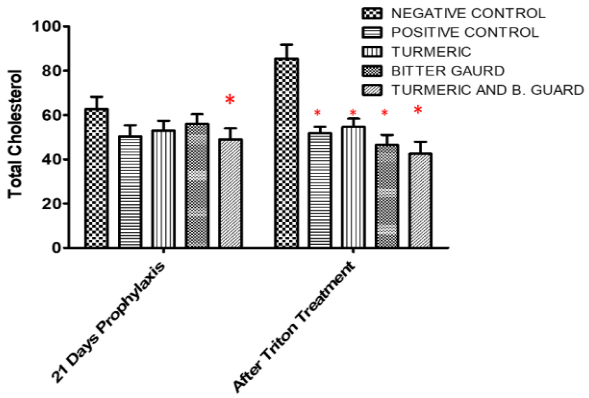

Figure 5: Medication effects on the total cholesterol level, during prophylaxis and after Triton 1339 WR

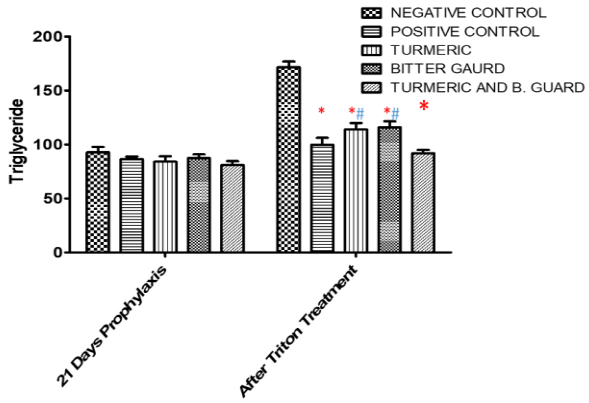

Figure 6: Medication effects on the triglyceride level, during prophylaxis and after Triton 1339 WR

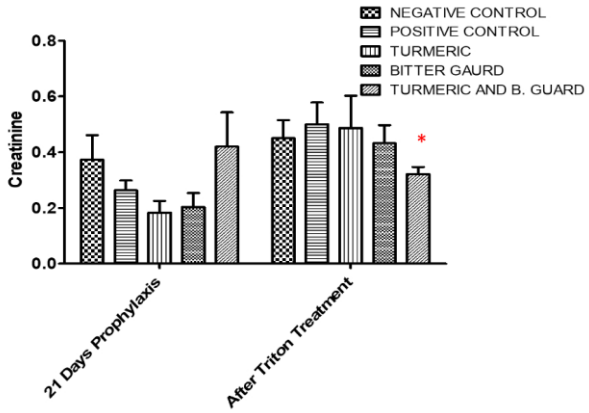

Figure 7: Medication effects on the creatinine level, during prophylaxis and after Triton 1339 WR

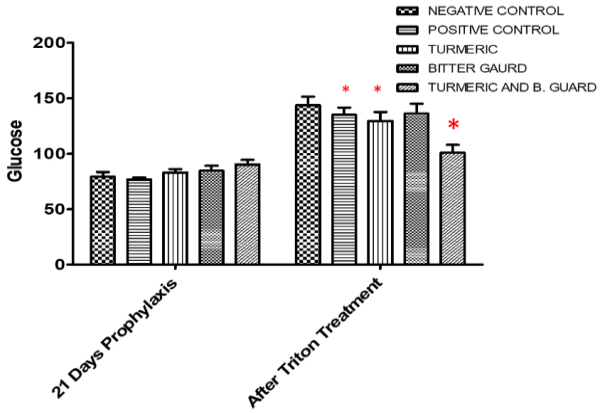

Figure 8: Medication effects on the glucose level, during prophylaxis and after Triton 1339 WR

markedly elevated AST levels (Fig. 2) compared to groups C, D, and particularly E; values in groups B and C were also considerably higher relative to group A. HDL levels (Fig. 3) were significantly lower in group A compared to groups B and C. HDL values were comparable across groups B, C, D, and E. LDL levels (Fig. 4) were markedly reduced in all treatment groups (B–E) relative to control group A and were similar across groups B, C, D, and E.

The combination group (E) exhibited a significant reduction in total cholesterol (Fig. 5) compared to group A. Triglyceride levels (TG) (Fig. 6) were markedly reduced in groups B, C, D, and particularly E, in comparison to group A. The medication did not markedly decrease triglyceride levels. The TG levels in groups B and E were comparable. Atorvastatin significantly reduced triglyceride levels in comparison to turmeric or bitter gourd extract alone. Creatinine levels (Fig. 7) were significantly reduced in group E compared to group A. Animals in the combination group (E) exhibited regulated glucose levels (Fig. 8) in comparison to other groups. Glucose levels showed no significant differences across groups B, C, and D. The combination therapy of bitter gourd and turmeric, represented in group E, exhibited a significantly pronounced impact, as illustrated in Table 2 and Figures 1-8. This combination successfully reduced lipid profile levels to normal values after 21 days of continuous treatment. Group E exhibited significant biochemical improvements compared to group A. The combination reduced LDL by 6% to 20 mg/dl, approaching normal levels, and lowered total cholesterol by 25% to 49 mg/dl from the negative control's 62.67 mg/dl, effects like the positive control. ALT levels were also significantly decreased ($p<0.05$) compared to both control groups (Table 2). Biochemical parameters did not significantly differ among groups A, B, C, and D. Triton WR 1339 significantly elevated ALT, AST, LDL, creatinine, glucose, TC, and TG levels in experimental animals ($p<0.05$) (Tables 2 and 3). Turmeric extract, bitter gourd, and their combination significantly reduced ALT, AST, LDL, TG, and TC levels ($p<0.05$), as shown in Figures 1-8 and Table 3. Compared to the negative control group, the atorvastatin group exhibited significantly elevated ALT and AST levels ($p<0.05$). Atorvastatin significantly reduced LDL and TC levels. The bitter gourd extract reduces the ALT level exclusively ($p<0.05$). The combination extract demonstrated health benefits by normalizing ALT, LDL, TC, and TG levels as a prophylactic measure ($p<0.05$). Atorvastatin markedly decreases the levels of LDL, total cholesterol, glucose, and triglycerides ($p<0.05$). Atorvastatin significantly increased HDL levels ($p<0.05$) compared to the control group (Fig. 1-8). However, the atorvastatin has no significant effect on AST level, when hyperlipidaemia was induced by Triton WR 1339 ($P>0.05$). It is interesting to mention that the

turmeric extract significantly reduces the ALT, AST, and TG. Bitter gourd extract (150 mg/kg) significantly reduced ALT and TG levels ($p<0.05$), unlike atorvastatin ($p<0.05$). The combined administration of bitter gourd and turmeric significantly reduce the ALT, AST, TC, and glucose level when compared to atorvastatin. Atorvastatin and the combined extract exhibit no significant difference in their effects on HDL, LDL, and TG levels ($p>0.05$) (Figure 1-8). The combination of extracts significantly reduced ALT and AST levels more than atorvastatin did. Currently, it can be stated that this combination positively affects liver enzymes; nevertheless, further trials are necessary to assess its comparative efficacy with atorvastatin.

DISCUSSION

The results above indicate that bitter gourd and turmeric extracts exhibited superior antioxidant properties compared to ascorbic acid and rutin, which serve as antioxidant standards, and are linked to several health benefits. This indicates that their effect as an extract may act as hepatoprotective and can overcome excess glutathione GSH production to get a good antioxidant effect on the blood and liver cells. Combining these two extracts shows promise for natural antioxidant production. Karela therapy improved hypercholesterolemia-induced decreases in serum superoxide dismutase, catalase, and glutathione (GSH), while also reducing increased malondialdehyde levels[10]. Ethanolic bitter gourd extract improved lipid profiles in diabetic Wistar rats, lowering blood cholesterol and triglycerides while increasing HDL cholesterol[27]. Bitter gourd, because of its physiologically active chemicals and antioxidant properties, has been linked to several health advantages[28]. According to[29] The extracts of wild bitter gourd leaves demonstrated antioxidant activity, as shown by their ability to scavenge nitric oxide and hydroxyl radicals. In group (D), which is treated by bitter gourd extract only, the bitter gourd extract showed excellent antioxidant effect in comparison with rutin only as antioxidant standard to be considered as relatively good natural antioxidant.

Curcumin impacts cholesterol in diabetic animals on high-cholesterol diets. Hepatic cholesterol-7-α-hydroxylase and HMG CoA reductase measurements elucidated the mechanism, revealing that curcumin-fed diabetic mice exhibited significantly increased cholesterol-7-α-hydroxylase activity, indicative of enhanced cholesterol catabolism[30]. Turmeric extract alone substantially decreases ALT, AST, and TG in group (C) compared to atorvastatin ($p<0.05$).

The hepatic mitochondrial superoxide levels were markedly reduced in the 5% turmeric group relative to the high-fat (HF) group. Turmeric supplementation also decreased cytosolic and mitochondrial hepatic hydrogen peroxide levels in comparison to the high-fat and cholesterol diet group. Turmeric supplementation significantly reduced mitochondrial hepatic carbonyl values compared to a high-fat, high-cholesterol diet, suggesting that turmeric powder mitigates oxidative damage by activating antioxidant defense systems in rats[31]. In group (C), which received solely turmeric extract, the extract demonstrated superior antioxidant efficacy compared to ascorbic acid and rutin, the usual antioxidants, correlating with several health advantages.

The extracts may mitigate hepatotoxicity caused by Triton WR1339 owing to their antioxidant properties (Table 2). Further studies and studies are necessary to comprehend the process and its impact on the liver.

CONCLUSION

The study found that turmeric and bitter guard extracts dramatically lower animal lipids. These extracts may protect the animals due to their antioxidant activity. The extract significantly affects liver enzymes (ALT, AST) and kidney function (creatinine levels). This extract may produce a hepatoprotective effect and may improve kidney function. It may be suggested at this time that this extract can be used along with atorvastatin in the treatment of hyperlipidemia. It is suggested to conduct another research study to investigate the effect of these extracts along with atorvastatin and investigate the possible herbal drug interaction as well. The bitter guard and turmeric are very commonly used in the Indian peninsula; the combination of the extract with other hypolipidemic agents may change the lipid profile and side effects.

REFERENCE

1. Lloyd-Jones DM, Morris PB, Ballantyne CM, Birtcher KK, Daly DD, DePalma SM, Minissian MB, Orringer CE, Smith SC. Focused update of the 2016 ACC expert consensus decision pathway on the role of non-statin therapies for LDL-cholesterol lowering in the management of atherosclerotic cardiovascular disease risk: A report of the American College of Cardiology task force on expert consensus decision pathways. J Am Coll Cardiol 2017;70. https://doi.org/10.1016/j.jacc.2017.07.745:1785-1822.
2. Kocaadam B, Şanlier N. Curcumin, an active component of turmeric (*Curcuma longa*), and its effects on health. Crit Rev Food Sci Nutr 2017;57. https://doi.org/10.1080/10408398.2015.1077195:2889-2895.
3. Hyre AD, Krousel-Wood MA, Muntner P, Kawasaki L, DeSalvo KB. Prevalence and predictors of poor antihypertensive medication adherence in an urban health clinic setting. J Clin Hypertens (Greenwich) 2007;9. https://doi.org/10.1111/j.1524-6175.2007.06372.x:179-186.
4. Luca SV, Macovei I, Bujor A, Miron A, Skalicka-Woźniak K, Aprotosoaie AC, Trifan A. Bioactivity of dietary polyphenols: The role of metabolites. Crit Rev Food Sci Nutr 2020;60. https://doi.org/10.1080/10408398.2018.1546669:626-659.
5. Aggarwal BB. Targeting inflammation-induced obesity and metabolic diseases by curcumin and other nutraceuticals. Annu Rev Nutr 2010;30:173-199.
6. Jaafer Al-Tameemi S. Inhibition of the growth of E. coli and candida by silver COMPLEXE (I) With Ligands. European Science Methodical Journal 2024;2:35-41.

7. Feng W, Wang H, Zhang P, Gao C, Tao J, Ge Z, Zhu D, Bi Y. Modulation of gut microbiota contributes to curcumin-mediated attenuation of hepatic steatosis in rats. Biochim Biophys Acta Gen Subj 2017;1861. https://doi.org/10.1016/j.bbagen.2017.03.017:1801-1812.
8. Al-Akeedi M, Najdawi M, Al-Balas Q, Al-Qazzan MB, Telfah ST. Novel anthraquinone amide derivatives as potential Glyoxalase-I inhibitors. J Med Life 2024;17. https://doi.org/10.25122/jml-2023-0257:87-98.
9. Krawinkel MB, Keding GB. Bitter gourd (*Momordica charantia*): A dietary approach to hyperglycemia. Nutr Rev 2006;64. https://doi.org/10.1301/nr.2006.jul.331-337:331-337.
10. Saad DY, Soliman MM, Baiomy AA, Yassin MH, El-Sawy HB. Effects of Karela (Bitter Melon; *Momordica charantia*) on genes of lipids and carbohydrates metabolism in experimental hypercholesterolemia: Biochemical, molecular and histopathological study. BMC Complement Altern Med 2017;17. https://doi.org/10.1186/s12906-017-1833-x:319.
11. Hassan KH, Al-hussaniy HA, Ibraim Oraibi A, Bashar Al-Qazzan M, Zainee HY, Al-Jashamy K, Imad Hussein M. Thermodynamic and Kinetic Assessment of Cobalt II Adsorption Using Green Synthesized NiO/γ-Al2O3 Nanoparticles. Salud, Ciencia y Tecnología - Serie de Conferencias 2025;4:1167. https://doi.org/10.56294/sctconf20251167.
12. Morton JF. Plant resources of South-East Asia 6. Econ Bot 1995;49. https://doi.org/10.1007/BF02862341:248-248.
13. Al-Zuhairi MI, Al-Tameemi SJ, Yasin YS. Physiological Scrutiny about the Hematological effects of resveratrol as compared with some statins in Rats n.d. DOI. https://doi.org/https://iarconsortium.org/srjp/publications?author=13494#.
14. Wang C-Y, Liu P-Y, Liao JK. Pleiotropic effects of statin therapy: Molecular mechanisms and clinical results. Trends Mol Med 2008;14. https://doi.org/10.1016/j.molmed.2007.11.004:37-44.
15. Hashim WS, Yasin YS, Jumaa AH, Al-Zuhairi MI, Abdulkareem AH. Physiological scrutiny to appraise a flavonol versus statins. Biomed Pharmacol J 2023;16. https://doi.org/10.13005/bpj/2610:289-293.
16. Rallidis LS, Fountoulaki K, Anastasiou-Nana M. Managing the underestimated risk of statin-associated myopathy. Int J Cardiol 2012;159. https://doi.org/10.1016/j.ijcard.2011.07.048:169-176.
17. Bone loss and inhaled glucocorticoids. N Engl J Med 2002;346. https://doi.org/10.1056/NEJM200202143460716:533-535.
18. Fuster V. A new perspective on nonprescription statins: An opportunity for patient education and involvement. Am J Cardiol 2007;100. https://doi.org/10.1016/j.amjcard.2007.06.009:907-910.
19. Kapoor JR. Effect of combination angiotensin-converting enzyme and angiotensin receptor blocker therapy on heart failure mortality and morbidity. Am J Cardiol 2008;101. https://doi.org/10.1016/j.amjcard.2007.10.013:744-745.
20. Guyton JR, Bays HE, Grundy SM, Jacobson TA, The National Lipid Association Statin Intolerance Panel. An assessment by the statin intolerance panel: 2014 update. J Clin Lipidol 2014;8 (Supplement). https://doi.org/10.1016/j.jacl.2014.03.002:S72-S81.
21. Keen HI, Krishnarajah J, Bates TR, Watts GF. Statin myopathy: The fly in the ointment for the prevention of cardiovascular disease in the 21st century? Expert Opin Drug Saf 2014;13. https://doi.org/10.1517/14740338.2014.937422:1227-1239.
22. Muntean DM, Thompson PD, Catapano AL, Stasiolek M, Fabis J, Muntner P, Serban M-C, Banach M. Statin-associated myopathy and the quest for biomarkers: Can we effectively predict statin-associated muscle symptoms? Drug Discov Today 2017;22. https://doi.org/10.1016/j.drudis.2016.09.001:85-96.
23. Banach M, Rizzo M, Toth PP, Farnier M, Davidson MH, Al-Rasadi K, Aronow WS, Athyros V, Djuric DM, Ezhov MV, Greenfield RS, Hovingh GK, Kostner K, Serban C, Lighezan D, Fras Z, Moriarty PM, Muntner P, Goudev A, Ceska R, Nicholls SJ, Broncel M, Nikolic D, Pella D, Puri R, Rysz J, Wong ND, Bajnok L, Jones SR, Ray KK, Mikhailidis DP. Statin intolerance—An attempt at a unified definition. Position paper from an International Lipid Expert Panel. Arch Med Sci 2015;11. https://doi.org/10.5114/aoms.2015.49807:1-23.
24. Parwin A, Najmi AK, Ismail MV, Kaundal M, Akhtar M. Protective effects of alendronate in Triton X-100-induced hyperlipidemia in rats. Turk J Gastroenterol 2019;30. https://doi.org/10.5152/tjg.2019.18076:557-564.
25. Tulini FL, Souza VB, Thomazini M, Silva MP, Massarioli AP, Alencar SM, Pallone EMJA, Genovese MI, Favaro-Trindade CS. Evaluation of the release profile, stability and antioxidant activity of a proanthocyanidin-rich cinnamon (Cinnamomum zeylanicum) extract co-encapsulated with α-tocopherol by spray chilling. Food Res Int 2017;95. https://doi.org/10.1016/j.foodres.2017.03.010:117-124.
26. Khalaf NA, Shakya AK, Al-Othman A, El-Agbar Z, Farah H. Antioxidant activity of some common plants. Turk J Biol 2008;32.
27. Fernandes NPC, Lagishetty CV, Panda VS, Naik SR. An experimental evaluation of the antidiabetic and antilipidemic properties of a standardized *Momordica charantia* fruit extract. BMC Complement Altern Med 2007;7. https://doi.org/10.1186/1472-6882-7-29:29.
28. Tan SP, Kha TC, Parks SE, Roach PD. Bitter melon (*Momordica charantia* L.) bioactive composition and health benefits: A review. Food Rev Int 2016;32. https://doi.org/10.1080/87559129.2015.1057843:181-202.

29. Tsai T-H, Huang C-J, Wu W-H, Huang W-C, Chyuan J-H, Tsai P-J. Antioxidant, cell-protective, and anti-melanogenic activities of leaf extracts from wild bitter melon (*Momordica charantia* Linn. var. abbreviata Ser.) cultivars. Bot Stud 2014;55. https://doi.org/10.1186/s40529-014-0078-y:78.
30. Babu PS, Srinivasan K. Hypolipidemic action of curcumin, the active principle of turmeric (*Curcuma longa*) in streptozotocin induced diabetic rats. Mol Cell Biochem 1997;166. https://doi.org/10.1023/A:1006819605211:169-175.
31. Kim M-S, Chun S-S, Choi J-H. Effects of turmeric (*Curcuma longa* L.) on antioxidative systems and oxidative damage in rats fed a high fat and cholesterol diet. J Korean Soc Food Sci Nutr 2013;42. https://doi.org/10.3746/jkfn.2013.42.4.570:570-576.