EVALUATION OF ANTI HYPERLIPEDIMIC ACTIVITY OF AMARANTHUS TRICOLOR EXTRACT IN EXPERIMENTAL ANIMAL MODEL

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ABSTRACT: ***To investigatetheanti diabetic and anti Hyperlipidemic activityof methanolextractof Amaranthusin male Wistarrats.Inthismodel of Hyperlipidemia,30 adult malewistarrats(150-200gms)were evenlydivided into 5groups in both groups.Group-1andGroup-2servedas untreated and modelcontrolsrespectively, whileGroup-3, 4and5 werethetreatments groups which were simultaneously treated with standard, 200and400mg/kgextract respectively along with High Fat Diet and Triton x 100. Onlastday, blood samples for biochemical parameters,were obtained under inhaled dietheranaesthesia.HFD and Triton x 100 treatment caused Hyperlipidemiaas evidenced by marked elevationinCholesterol, Triglycerides, LDL, VLDL and decrease in HDL levels.Co-administration of extractwith HFD and Triton x 100decreased rise Cholesterol, Triglycerides, LDL, VLDL and increase in HDL levels. It was observed that the methanolextract of Amaranthusconferred Anti-Hyperlipidemia activity by biochemical observation against HFD and Triton-x-100induced Hyperlipidemiainrats. In the near future could constitute aleadto discovery of anoveldrug for treatment of drug induced Hyperlipidemia.***

I. INTRODUCTION

Hyperlipidemia is a condition when abnormally high levels of lipids i.e.,the fatty substanceare found in theblood.This condition is also called hypercholesterolemia/hyperlipoproteinemia[1].Human body is complex machinery and for maintaining the homeostasis of various organ and organ system.Any undesirable change will disturb the balance resulting in diseased state[2].Lipids are fats in the blood stream,commonly divided into cholesterol and triglycerides. Cholesterol circulates in the bloodstream and is involved in the structure andfunction of cells.Triglycerides(TG) are best viewed as energy that is either used immediately or stored in fat cells.TG are manufactured in the liver from the foods or by being absorbed from the intestine[3].Virchow in 19thcentury who identified cholesterol crystals in atherosclerotic lesion and stated that endothelial cell injury initiates atherogenesis2.In a modification of this hypothesis it was proposed that the endothelium normally influences the behavior of arterial smooth muscle cells by providing a barrier to the passage of plasma proteins, and that the major effect of haemodynamic or other factors that injure endothelium is to reduce the effectiveness of the barrier[4].Arteries are normally smooth and unobstructed on the inside, but in case of increased lipid level, a sticky substance called plaque is formed inside the walls of arteries. This leads to reduced blood flow, leading to stiffening and narrowing of the arteries. It has been proved that elevated plasma levels of cholesterol and of LDL are responsible for atherosclerosis in man, and epidemiological data suggests that elevated plasma levels of HDL have a protective effect[5.]

II. MATERIALS AND METHODS:

I. Plat Material

The leaves of plant *Amaranthus* was collected from hilly region of Chittoor district, Tirupathi, A.P, India. The plant was authenticated by Dr. K. Madhav Chetty, Asst. Professor, Dept. of Botany, Sri Venkateshwara University, Tirupathi.

II. Drugs And Chemicals

Amaranthus, all other chemicals and diagnostic kits were provided by Sigma Institute of Clinical Research and Administration.

III. Phytochemical Screening

Preliminary phytochemical investigation was carried out on Methanol extract of *Amaranthus* leaf for detection of

various phytochemicals by standard methods[90]

IV. Determination Of Acute Oral Toxicity

Acute toxicity studies were performed according to OECD-423 guidelines category IV substance (acute toxic class method). Albino rats (n=3) of either sex selected by random sampling technique were employed in this study. The animals were fasted for 4 hrs with free access to water only. The plant extracts of *Amaranthus* were administered orally with maximum dose of 2000 mg/kg body weight. The mortality was observed for three days. If mortality was observed in 2/3 or 3/3 of animals, then the dose administered was considered as a toxic dose. However, if the mortality was observed only one rat out of three animals then the same dose was repeated again to confirm the toxic effect. If mortality was not observed, the procedure was then repeated with higher dose (Organization for economic Co-operation and development, 2001).

V. High Fat Diet Composition[94]

Table 5.1 :High Fat Diet Composition

Composition	Normaldiet (gm)	High Fatdie (gm)
Protein(Milkpowder)	12	10
Carbohydrates(Wheatflour)	71	61
Sugar	05	05
Fat(Butter)	05	16
Salts	04	04
Vitamins	01	02
Fibers	02	01
Cholesterol	--	01
TotalWeight	100g	100g

VI. Experimental Animals

Wistar albino adult male rats weighing 200-250g were obtained from the animal house. The animal were grouped and housed in polyacrylic cages (38x 23x 10 cm) with not more than five animals per cage and maintained under standard laboratory under standard laboratory conditions (temperature 25+2oC) with dark and light cycle (14/10 hour). They were allowed free access to standard dry pellet diet (Hindustan Lever, Kolkata, India) and water *ad libitum*. The animals were acclimatized to laboratory condition for 10 days before commencement of experiment.
The experimental protocol was approved by Institutional Animal Ethical Committee (IAEC) constituted under CPCSEA.

VII. Induction of Hyperlipidemia by Triton-x-100

Hyperlipidemia was induced in Wistar albino rats by single intraperitoneal injection of freshly prepared solution of Triton-X-100 (100 mg/kg) in physiological saline solution after overnight fasting for 18 h[95].
The animals were divided into five groups of six rats each.

I. The first group was given standard pellet diet, water and orally administered with 2% Tween 80.
II. The second group was given a single dose of triton administered at a dose of 100mg/kg, i.p. After 72 hours of triton injection, this group received a daily dose of 2% Tween 80 (p.o) for 7 days.
III. The third group was administered a daily dose of Atorvastatin 10 mg/day
IV. Fourth group Amaranthus200mg/kg suspended in 2% Tween 80, p.o., for 7 days, after inducing hyperlipidemia.
V. Fifth group was administered with the Amaranthus400 mg/kg, p.o. for 7 days[96] .

VIII. Induction of Hyperlipidemia by High Fat Diet

The animals were divided into five groups. Each group contains six animals.

Grouping is as follows:

Group 1: Normal Group (Tween 80)

Group 2: Control Group (HFD)

Group 3: Standard-Atorvastatin + HFD (10 mg/kg)

Group 4: Extract II- *Amaranthus*+ HFD (400 mg/kg)

Group 5: Extract I- *Amaranthus*+ HFD(200 mg/kg)

The study Duration is 14 days

IX. Collection of blood

On the 8thday, blood was collected by retro orbital sinus puncture, under mild ether anaesthesia. The collected samples were centrifuged for 10 minutes. Then serum samples were collected and used for various biochemical experiments. The animals were then sacrificed and the liver collected[97].

X. Bio Chemical Estimations:

A. Cholesterol[98-100]

Clinical significance

Measurement of serum cholesterol levels are useful in evaluation of the risk of the coronary arterial occlusion, atherosclerosis, myocardial infarction , liver function, biliary function, intestinal absorption, thyroid function and adrenal disease.

Principle

The estimation of cholesterol involves the following enzyme catalyzed reactions.

Cholesterol ester $\xrightarrow{CE}$ cholesterol + fatty acid

Cholesterol ester + O_2 $\xrightarrow{CHOD}$ cholest-4-en-3one + H_2O_2

H_2O_2 + 4AAP + phenol $\xrightarrow{POD}$ 4 H_2O + Quinoneimine

CE: cholesterol esterase

CHOD: Cholesterol oxidase

4AAP: 4 aminoantopyrine

Calculation

$$\text{Cholesterol (mg/dl)} = \frac{\text{Abs. of test}}{\text{Abs. of standard}} \text{ concentration of standard (mg/dl)}$$

Procedure

Pipette in to test tubes	Blank	Standard	Sample
Working reagent 1	1000 µl	1000 µl	1000µl
Distill water	10µl	……	……..
Standard	……	10 µl	……..
Sample	…….	……	10 µl

Mix well after each addition and incubate at 37° C for 10 minutes. Read absorbance ofstandard and test against reagent blank at 505/670 nm.

Increase

- Increased levels are found most characteristically, in primary hyperlipoproteinaemias
- In nephrotic syndromes
- Myoxoedema
- Obstructive jaundice

- Diabetes mellitus

Decreases

- Low are frequently obtained in anemia
- In hemolytic jaundice
- In malabsorption syndrome
- severe malnutrition
- Acute infections and in terminal state
- Very low values occur in betalipoproteinemia and to a lesser degree in familial hypobetalipoproteinaemias.

B. Triglycerides[101]

Clinical significance

Triglycerides are a family of lipids absorbed from the diet and produced endogenously from carbohydrates. Measurement of triglyceride is important in the diagnosis and management of hyperlipidemias. These diseases can be genetic or secondary to other disorders including nephrosis, diabetes mellitus and endocrine disturbances. Elevation of triglycerides has been identified as a risk factor for atherosclerotic disease.

Principle

$$\text{Triglycerides} + H_2O \xrightarrow{\text{Lipase}} \text{Glycerol} + \text{Fatty acids}$$

$$\text{Glycerol} + ATP \xrightarrow{GK} \text{Glycerol-3-phosphate} + ADP$$

$$\text{Glycerol-3-Phosphate} + O_2 \xrightarrow{GPO} \text{Dihydroxy phosphate} + H_2O_2$$

$$H_2O_2 + \text{4-aminoantipyrine} + \text{3, 5-DHBS} \xrightarrow{Mg^{2}} \text{Quinoneimine} + 3H_2O$$

GK: Glycerokinase

GPO: Glycerol 3 phosphate oxidase

DHBS: 3, 5-Dichloro-2-hydroxybenzene sulfonate

Lipase hydrolysis serum triglycerides to glycerol and fatty acids. The liberate glycerol is converted to glycerol-3-phosphate in the presence of ATP and glycerokinase. Glycerol-3 phosphate is oxidised by glycerol 3 phosphate oxidase to yield H_2O_2. H_2O_2 thus generated reacts with 4-aminophenazone and 4-chlorophenol under the catalytic influence of peroxidase to form colouredquinoneimine complex. The intensity of colour so developed is proportional to triglyceride concentration and is measured at 505 nm.

Procedure

Pipette in to test tubes	Blank	Standard	Sample
Working reagent 1	1000 µl	1000 µl	1000µl
Distill water	10µl	……	……..
Standard	……	10 µl	……..
Sample	…….	……	10 µl

Mix and incubate for 10 minutes at 37° C. Read absorbance of standard and test against reagent blank at 505 (500-540 nm).

Calculation

$$\text{Triglycerides (mg/dl)} = \frac{\text{Abs. of test}}{\text{Abs of standard}} \text{ concentration of standard (mg/dl)}$$

A. HDL-Cholesterol[102]

Clinical significance

High density lipoprotein (HDL) contains particles of different density including lipid and highest concentration of proteins amongst the different lipoproteins. Includes free and esterified cholesterol, triglycerides, phospholipids and apoproteins A, C and E. HDL cholesterol values are about $1/5^{th}$ of the total cholesterol values and can be expressed as percentage of total cholesterol.

Decreases

There exists an inverse relationship between HDL cholesterol and coronary heart diseases. Low concentration i.e.

below 30 mg/dl is one of the risk factors for cardiac ailments.

Principle

Chylomicrons, LDL and VLDL (low and very low density lipoproteins) are precipitated from serum phophotungstate in the presence of divalent cat ions such as magnesium. The HDL cholesterol remains unaffected in the supernatant and is estimated using Robonik cholesterol reagent.

Phophotungstate
Serum/plasma= ——— HDL + (LDL + VLDL + chylomicrons)
Mg2+ (Supernatant) (Precipitate)

Procedure:

Pipette in to test tubes	Blank	Standard	Sample
Working reagent 1	1000 µl	1000 µl	1000µl
Distill water	10µl	……	……..
Standard	……	10 µl	……..
Sample	…….	……	10 µl

Mix well and allow the reaction mixture to stand for 10 min at R.T. centrifuge at 4000 rmp for 10 min to obtain clear supernatant. Use the supernatant to obtain the concentration of HDL cholesterol in the sample.

Calculation

. Abs. of test
HDL (mg/dl) = ——— x Concen of standard (mg/dl)x Dilution factor
Abs. of standard

B. SGOT/AST[103-104]

Principle

Kinetic determination of the aspartate aminotransferase (GOT) activity :

L - Aspartate + α –Ketoglutarate —GOT→ Oxaloacetate + L-Glutamate

Oxaloacetate + NADH + H —MDH→ L-Malate + NAD

ASSAY PROCEDURE 1: Two Reagent procedures

Pipette in to test tubes	Sample/Control
R1	800µl
R2	200 µl

Mix and Incubate at 37°C for 2 minutes then add

Pipette in to test tubes	Sample/Control
Working reagent	1000µl
Sample/Control	100 µl

Mix and after a 60 seconds incubation at 37°C measure the change of absorbance per minute (ΔA/minute) during 180 seconds.

Calculation:

Activity of Sample (U/L) = Δ A/Min X1746

C. SGPT/ ALT[105-108]

Principle

Kinetic determination of the GPT activity

$$\text{L - Alanine} + \alpha\text{–Ketoglutarate} \xrightarrow{\text{GPT}} \text{Pyruvate} + \text{L-Glutamate}$$

$$\text{Pyruvate} + \text{NADH} + H^{+} \xrightarrow{\text{LDH}} \text{L-Lactate} + NAD^{+}$$

ASSAY PROCEDURE 1: Two Reagent procedures

Pipette in to test tubes	Sample/Control
R1	800µl
R2	200 µl

Mix and Incubate at 37°C for 2 minutes then add

Table : 10 Kinetic determination of the GPT activity

Pipette in to test tubes	Sample/Control
Working reagent	1000µl
Sample/Control	100 µl

Mix and after a 60 seconds incubation at 37°C measure the change of absorbance per minute (ΔA/minute) during 180 seconds.

Calculation:

Activity of Sample (U/L) = Δ A/Min X 1746

C. ALKALINE PHOSPHATASE[107]

p-Nitrophenyl phosphate is converted to p-nitrophenol and phosphate by alkaline phosphatase. The rate of formation of p-Nitrophenol is measured as an increase in absorbancewhich is proportional to the ALP activity in the sample.

$$\text{p-Nitrophenyl phosphate} \xrightarrow{\text{ALP}} \text{p-Nitrophenol} + \text{Phosphate}$$

Assay procedure 1: Two Reagent procedures

Pipette in to test tubes	Sample/Control
R1	800µl
R2	200 µl

A. Mix and Incubate at 37°C for 2 minutes then add

Pipette in to test tubes	Sample/Control
Working reagent	1000µl

Sample/Control	100 µl

Mix and after a 60 seconds incubation at 37°C measure the change of absorbance per minute (ΔA/minute) during 180 seconds.

Calculation:

At 405 nm with mono reagent procedure and two reagent procedure for 1 cm path light cuvette

Activity of Sample (U/L) = (Δ A/Min) X 2712

A. Estimation of Serum Total Proteins: [108-109]

Principle: The peptide bond of proteins reacts with CU+2 ions in alkaline solution to from a blue violet complex (Biuret reaction), each copper ion complexing with 5 or 6 peptide bonds. Tartarate is added as stabilizer while iodine is used to prevent auto reduction of alkaline copper complex. The colour formed is proportional to the protein concentration and is measured at 546 nm.

Procedure:

Reagents	Blank(µl)	Standard(µl)	Sample(µl)
Working reagent	1000	1000	1000
Distilled water	20	----	----
Standard	----	20	----
Sample	----	----	20

Incubate for 10 min. at 37 [c.] Read absorbance of standard and each sample at 546 nm against reagent blank.

B. Glucose[110]

Glucose estimation by Trinder‟s method

Clinical significance

Accurate measurement of glucose in body fluids is important in the diagnosis and management of diabetes, hypoglycaemia, adrenal dysfunction and various other conditions.

Principle

Glucose in sample is oxidized to yield gluconic acid and hydrogen peroxide in the presence of glucose oxidase.The enzyme peroxidase catalyses the oxidative coupling of 4aminoantipyrine with phenol to yield a colouredquioneimine complex, with absorbance proportional to the concentration of glucose in sample.

$$\text{Glucose} + O^2 + H_2O \xrightarrow{\text{Glucose oxidase}} \text{Gluconic acid} + H_2O_2$$

$$H_2O_2 + \text{Phenol} + 4\text{AAP} \longrightarrow \text{Quinoneimine dye} + 2H_2O$$

Procedure

Pipette in to test tubes	Blank	Standard	Sample
Working reagent 1	1000 µl	1000 µl	1000µl
Distill water	10µl	……	……..
Standard	……	10 µl	……..
Sample	…….	……	10 µl

Increases

- Diabetes mellitus
- In patients receiving glucose containing fluids intravenously, during severe stress and cerebrovascular

accidents.

Decreases

- On insulin administration, as a result of insulinoma
- In born errors of carbohydrate metabolism or on fasting.

XI. STATISTICAL ANALYSIS: All data were expressed as the mean ± SEM. For statistical analysis of the data, group means were compared by one-way analysis of variance (ANOVA) followed by Dunnett's test, $P<0.05$ was considered significant.

III. RESULTS AND DISCUSSIONS

Table 6.1: ListofInstrumentsused

Sr.No.	NameofInstrument	Description
1.	Autoanalyser	ARTOS, The versatile Autoanalyser, SlNo-SBPL/ 188/06-07
2.	Incubator	REMI Cooling Centrifuge. C-24 BL.
3.	Digital Balance	ACCULAB – Sartorious group.
4.	Flash Evaporator	SUPERFIT, Rotary "VaccumDigital Bath", PMTc – 3040
5.	Deep Freezer	BLUE STAR, Model No.- CHE400, SrNo.-67771
6.	Homogenizer	REMIHomogenizerMumbai. Type – RQ 127A

Table 6.2: ListofChemicalsused

Sr.No	NameofChemical	Description
1.	Gentamicin	Gifted by MicrolabsPvt. Ltd. Bangalore
2.	Heparin	(HEP-5)Gland Pharma Ltd, Hyderabad.Batch – No. UJ918
3.	Chloroform	S.D. Fine – ChemLtd. Mumbai.
4.	KCl	S.D. Fine– ChemLtd. Mumbai. Batch-No: -200Z-0200-1612-09. Mole. Wt:- 74.55
5.	Formalin	FischerScientific. LotNo:- 91026906-5., PdtNo.24005
6.	NaCl	LeoChem, Bangalore. Mole.Wt:- 58.44, Lot No. 126012
7.	EDTA	S.D.Fine ChemLtd. PdtNo:-LO4/10204/2611/13
8.	KH_2PO_4	QualigensfineChemicals, Mumbai. LotNo:-18986711-
9.	K_2HPO_4	Leochem, Bangalore. Lot No- 125176, P-2V829.
10.	H_2O_2(30%w/v)	SDFCL-38694L05,BatchNo-G09A/2209/0807/13

11.	Methanol	SDFCL. Mole Wt:- 32.04, B.P. 64-65.5 ºC, Batch No:-K08A/1308/1211/13
12.	Trichloroacetic acid	Nice Chemicals, Bombay.
13.	Thiobarbituric acid (TBA)	Loba Chemicals, Mumbai.
14.	Sodiumazide	S.D. Fine ChemLtd.
15.	Reduced glutathione	Sigma U.S.A.
19.	UreaKit	Coral Clinical Systems, Verna Goa, India.
20.	Uric acid Kit	Coral Clinical Systems, Verna Goa, India.
21.	Creatinine Kit	Coral Clinical Systems, Verna Goa, India.

Table 6.3:. Preliminary phytochemical screening of *Amaranthus* extract:

SLNO.	TEST	RESULT
1.	**ALKALOIDAL TEST** a.Dragondroffs test b.Mayer's test c.Wagner's test d. Hager's test	 Positive Positive Positive Positive
2.	**CARBOHYDRATES TEST** a.Molish's test b.Fehling's test c.Benedict's test d. Baeford's test	 Positive Positive Positive Positive
3.	**STEROIDS TEST** a.LibermannBuchard test b. Salwoski test	 Positive Positive
4.	**GLYCOSIDES TEST** a.Legal test b.Baljet test c.Killerkilaini test d. Borntagers test	 Positive Positive Positive Positive
5.	**SAPONINS TEST** a.Foam test	 Positive
6.	**FLAVONOIDS TEST** a.Shinoda test	 Positive
7.	**TRITERPINOIDAL TEST**	Negative
8.	**PHENOLICS & TANNINS TEST** a.Ferric chloride test b.Gelatin test c.Lead acetate test	 Negative Negative Negative
9.	**PROTIEN& AMINOACIDS TEST** a.Buret's test b.Ninhydrin test c.Xanthoprotic test	 Positive Positive Positive
10.	**FIXED OIL TEST** a.Spot test	 Positive
11.	**RESIN TEST** a.Acetic anhydride test	 Positive

Table no 6.4: Percentage yield of crude extract of *Amaranthus*

Sl.No.	Solvent	Color and Consistency	Percentage yield
1	Ethanol	Dark brown sticky	10.2%

I. Evaluation of Anti Hyperlipidemic activity of *Amaranthus* In Rats

Mean And S.E.M Of Parameters Of The Animals

Table 6.5:TRITON-X-100 INDUCED MODEL:

Triton x-100					
TEST	**NORMAL**	**CONTROL**	**STANDARD**	**T1**	**T2**
ALP	74.59±3.107	162.51±1.34***	125.78±1.52***	126.23±0.92***	78.2±1.423
GPT	35.26±1.275	65.80±1.413***	41.11±3.826	40.79±1.385***	35.71±1.671**
GOT	41.50±3.226	53.70±3.894*	41.68±2.426	42.35±2.310	46.32±2.075
TP	40.31±3.128	32.71±1.751***	19.56±2.321***	16.26±2.315***	22.89±1.483***
HDL	53.48±3.652***	23.82±1.516	41.69±3.971**	51.25±2.153	56.85±2.351
TG	51.12±2.128	81.76±1.621***	79.23±1.619***	81.2±2.210***	61.42±3.126***
TC	66.35±2.328	151.01±2.121***	68.05±1.451	98.52±1.612***	92.21±1.811***
VLDL	10.01±0.233	15.59±0.627***	13.85±0.352***	15.45±0.356***	13.30±1.464***
LDL	11.56±2.692	101.63±5.069***	10.63±3.114	33.83±4.159	21.98±2.700
AI	0.41±0.239	3.29±0.358	0.42±0.189	0.96±0.328	0.51±0.496
CRR	3.12±0.521	7.63±0.491	2.68±0.122	2.23±0.214	2.16±0.161

Table 6.6 Body Weight

TRITON X100	NORMAL	CONTROL	STANDARD	T1	T2
Before treatment	173.0±0.96	172.66±0.89	174.0±1.12	173.33±0.88	172.0±0.85
After treatment	183.5±0.76	243.0±0.96***	194.33±0.66***	224.33±0.88***	244.83±1.14***

N = 6; Significance:*** $P<0.001$, ** $P<0.01$, * $P<0.05$ from control

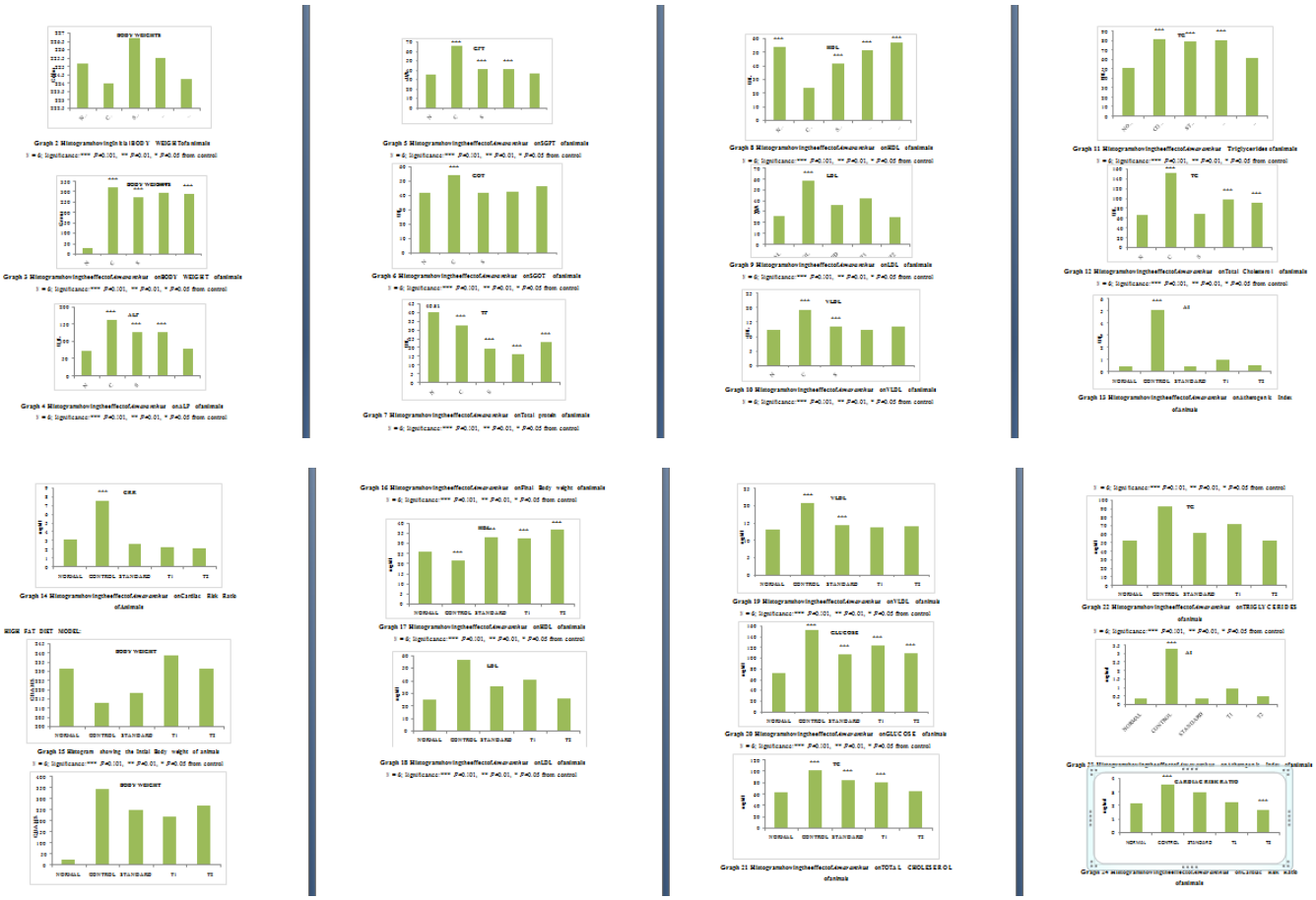
I. HFD DIET INDUCED MODEL

Table no 6.7: Biochemical Parameters of the Animals

HFD diet	NORMAL	CONTROL	STANDARD	T1	T2
B.wB.T	231.28±0.1	213±1.06	218.59±2	238.7±0.43	231.12±0.61
B.wA.T	25.4±0.15	342.61±2.4***	251.29±0.5***	221.43±2.71***	269±2.75***
HDL	26.12±0.23	21.5±0.83**	33.19±0.54**	32.23±0.47**	36.87±0.10***
LDL	25.19±0.56	56.40±0.10***	35.85±0.26***	41.17±1.43***	26.28±1.69
VLDL	13.10±2.63	20.72±0.58***	14.38±0.59***	13.76±0.48	14.22±0.52
GLUCOSE	72.43±0.81	151.2±0.60***	107.1±0.75***	124.1±0.47***	108.1±0.70***
TC	64.02±0.51	102.0±0.19***	86.1±0.60***	81.25±2.55***	65.43±0.39
TG	52.05±1.15	92.01±0.24***	60.94±2.11***	71.55±2.46***	52.89±0.75

AI	1.41±0.574	3.67±0.529***	1.66±0.619	1.69±0.46	0.77±0.98***
CRR	2.21±0.218	3.57±0.529***	2.98±0.328	2.31±0.85	1.75±0.89***

TRITON-X-100 INDUCED HYPERLIDAEMIA MODEL:



IV. CONCLUSION

Phytochemical screening of the extract shows the presence of chemical constituents like Alkaloids, steroids, fixed oils, cardio tonic aglycones, flavonoids ,saponins ,carbohydrates, proteins, resins. Acute toxicity tests were performed according to the OECD guide line no.423, LD50 value was found to be 200mg/kg and 400mg/kg.

Anti Hyperlipidaemic activity was performed by using the high fat diet and Triton-x-100 induced method. In the present study an increase in plasma HDL-cholesterol with a concomitant percentage decrease from other lipid was observed. It can be concluded from the present data that the levels of total serum cholesterol, triglyceride and MDA which are actually raised in high fat diet, can be lowered significantly with *Amaranthus* And total proteinswhich is actually lowered in Triton-x-100 can be raised significantly with *Amaranthus***.** Atherogenic index which actually raisedin atherogenic diet and Triton-x-100, can be lowered significantly with *Amaranthus* and a very good % protection was seen with *Amaranthus* and standard drug.

The extract also show increase in the glucose tolerance of the rats and decrease in the fasting blood glucose level of diabetic rats, showing the hypoglycaemic activity of the plant which is most pronounced in methanol extract.

In nutshell the extract of *Amaranthus* possesses significant hypoglycaemic activity and anti Hyperlipidaemic activity, which is the first claim in this respect.

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