Anti hyperlipidemic effect of Vara Asanadi Kwatha against high fat diet induced hyperlipidemic rats

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Abstract

Changing life style and diet patterns along with significant role played by genetics made Hyperlipidemia/ Dyslipidemia as one of the most common metabolic aberration of lipids among people all over the world. An extra cavernous research study on classical Ayurvedic formulations which are not in the limelight of routine clinical practice is essential to explore the most effective and target oriented anti hyperlipidemic drugs. Objective of this study was to evaluate anti-hyperlipidemic activity of *Vara asanadi kwatha* against high fat diet induced hyperlipidemic Wistar strain albino rats. Wistar strain albino rats of either sex weighing 180 ± 25 g six animals were selected and housed with each cage containing 6 animals. Test drug treated animals were managed with *Vara Asanadi Kwatha* at a dose of 8 ml/kg in which the efficacy of medicine has been assessed on various serum biochemical parameters, histopathological sections and weights of liver, kidney and heart. One group kept as cholesterol control and the remaining as water control. Findings are in favour of mild anti hyperlipidemic and significant hepato-protective and nephro-protective activities of the test formulation. *Vara Asanadi Kwatha* is a mild anti-hyperlipidemic and potent hepato-protective as well as renoprotective drug.

Introduction

Lipid and lipo-protein abnormalities have become enormously common in the general populace. The metabolic aberrations of lipids are linked as risk factor with numerous numbers of serious systemic illnesses including cardio vascular disorders and metabolic syndrome [1]. Obesity and hyperlipidemia often exist together clinically and share much in common from the etio-pathology to the complications [2]. *Vara Asanadi Kwatha* [VAK] is a classical Ayurvedic formulation in the form of decoction, which claims to be effective in the management of overweight and obesity [3]. An experimental evaluation of the drug on its anti-hyperlipidemic action will certainly give thoughts regarding the efficacy of Vara Asanadi Kwatha in counteracting the ill effects of dyslipidemia. With this judgment the present experimental study was carried out to screen anti-hyperlipidemic potential of VAK in experimental animals.

Materials and Methods

Test formulation:

The ingredient wise composition of Vara Asanadi Kwatha has been provided in Table 1.

Each raw constituent of VAK was subjected to pharmacognostical identification and was certified as genuine and of good quality in the Department of Pharmacognosy, Institute of Post Graduate Teaching and Research in Ayurveda (IPGT and RA), Gujarat Ayurved University, Jamnagar. The test drug was prepared by adding one part of the crushed raw drug to sixteen parts of water, boiled and reduced to half. Thin sheets of Iron was added during the boiling period of kwatha and later removed while filtering. The prepared drug was procured from Ayurveda Pharmacy, Kannur, Kerala.

Animals:

Wistar strain albino rats of either sex weighing 180 ± 25g were obtained from animal house attached to Pharmacology Laboratory of IPGT and RA Gujarat Ayurved University, Jamnagar. Six animals were housed in each cage made up of poly-propylene with stainless steel top grill. The dry wheat (post hulled) waste was used as bedding material and was changed every morning. The animals were exposed to 12 hour light and 12 hour dark

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Table 1: Composition of *Vara Asanadi Kwatha*

Plant and part used	*Latin name*	*Quantity*
Triphala		
Haritaki	*Terminalia chebula*	1 part each
Bibhitaki	*Terminalia bellerica*	
Amalaki	*Emblica officinalis*	
Asana	*Pterocarpus marsupium*	1 Part
Citraka	*Plumbago zeylanica*	1 Part
Haridra	*Curcuma longa*	1Part
Lohaptra	(Fe_2O_3)	Thin sheets added on boiling kwatha (Decoction) and removed afterwards

cycle with the relative humidity of 50 to 70% and the ambient temperature during the period of experimentation was 22 ± 03°C. Animals were fed with Amrut brand rat pellet feed supplied by Pranav Agro Mills Pvt. Limited.

For their drinking purpose tap water ad libitum was used. The experiment was carried out after obtaining the permission from institutional animal ethics committee. (Approval number; IAEC (Institutional Animal Ethics Committee) 06/09-11/PhD/05).

Dose fixation and schedule:

The human dose of *Vara Asanadi Kwatha* is 45ml twice a day (90 ml per day) [4]. The suitable dose for rats was calculated by referring to table of Paget and Barnes (1964) [5] and the dose obtained thus was 8 ml/kg rat. The test formulation was administered with the help of oral catheter attached to a disposable syringe.

Anti-hyperlipidemic activity:

The effect of test formulation on diet induced hyperlipidemia was carried out as per previous study [6]. The selected animals were divided into three groups of six animals each. First group was kept as normal control (NC) which received only tap water.

To second group hyperlipidemic diet was administered and served as cholesterol control (CC) group. Third group received hyperlipidemic diet and *Vara Asanadi Kwatha* (VAK). Test drug was administered at morning hour and hyperlipidemic diet (to second and third group) was administered at evening hours for 20 consecutive days. The hyperlipidemic diet includes hydrogenated vegetable oil (Vanaspati Ghee - 'Raag' brand, Batch No. BA 76, Adani Wilmar Ltd., Gujarat) and cholesterol extra pure powder (Batch No. 14036 Suvidhnath Laboratories, Baroda) made in to 20% suspension in coconut oil (Parachute coconut oil, Batch No. GSW002, Ponda-Goa.).The suspension was administered at the dose of 0.5 ml/100 g rat. On the 21st day after overnight fasting, the animals were weighed again and blood was collected from retro-orbital plexus under ether anaesthesia. From separated serum; biochemical parameters like glucose [7], serum total cholesterol [8], serum triglyceride [9], and serum high density lipoprotein cholesterol (HDL-C) [10], Serum low density lipoprotein cholesterol+ very low density lipoprotein cholesterol (LDL-C + VLDL-C) were estimated. Serum (LDL+VLDL) was calculated by subtracting HDL cholesterol value from total cholesterol instead using both values separately, as in rats whose serum cholesterol is <100 mg/dl Friedewald formula overestimates LDL levels [11]. Further blood urea [12], serum creatinine [(13], serum glutamic oxaloacetic transaminase (S.GOT), serum glutamic pyruvic transaminase (S.GPT) [14], alkaline phosphatase [15], total bilirubin [16], direct bilirubin [17] and serum uric acid[18] were also estimated as per standard procedure. Further, all the rats were sacrificed by overdose of ether anesthesia and from the sacrificed animals liver, kidney, heart and aorta were excised out. The liver, kidney and heart were weighed and fixed in 10% buffered neutral formalin solution. After fixation, tissues were embedded in paraffin and serial sections were cut and each section was stained with hematoxylin and eosin [19]. The slides were viewed under trinocular research microscope (Germany) at various magnifications to note down the changes in the microscopic features of the tissues studied.

Statistical analysis:

The results were presented as mean ± SEM for six rats in each group. Statistical comparisons were performed by unpaired student's t test by using Sigma stat software (version 3.1) for all the treated groups with the level of significance set at $P<0.05$.

Results

Data related to effect of VAK on body weight of albino rats have been provided in Table 2.

Table 2: Effect on body weight

Treatment	*Body weight (g)*			
	Initial body weight (g)	*Final body weight (g)*	*Actual change in body weight (g)*	*% change in comparison to control*
NC(n=6)	203.00±6.38	218.00±6.97**	15.00±3.49	–
CC(n=6)	174.67±8.88	196.00±11.41*	21.33±5.74	42.20↑
VAK(n=6)	177.00±3.96	217.33±7.04**	40.33±6.56	89.08↑

Data: Mean ± SEM (standard error of mean), ↑- Increase, *P<0.05, **P<0.01,
(Compared with initial body weight, paired t test)

In normal control rats a progressive gain in body weight was occurred in comparison to its initial values. In contrast to this, significant increase in body weight was occurred in cholesterol control group in comparison to both initial values. In VAK treated group also significant increase in body weight was occurred in comparison to its initial value. Marginal increase of relative weight of liver and heart was found in cholesterol control group in comparison to normal control group which is found to be statistically non-significant (Table 3).

Table 3: Effect on weight of important organs

Treatment	Weight of liver (mg/100g)	Weight of heart (mg/100g)	Weight of kidney (mg/100g)
NC (n=6)	3061.83±102.96	335.13±6.26	605.00±08.04
CC(n=6)	3356.85±185.82	337.39±14.58	642.19±10.32*
VAK(n=6)	2934.58±43.99	330.69±6.93	591.58±10.42$^{\alpha\alpha}$

Data: Mean SEM

*P<0.001(Compared with normal control group, unpaired t test)

$^{\alpha\alpha}$ P<0.01(Compared with cholesterol control group, unpaired t test)

Treatment with VAK attenuated weight of these organs in non-significant manner. Further, cholesterol control group significantly increased the kidney weight and treatment with VAK significantly attenuated it. The data related to the effect of VAK on serum biochemical parameters were provided in Table 4.

Feeding of cholesterol diet led to significant increase in serum glucose in comparison to normal control group and treatment with VAK non-significantly attenuated it. Further blood urea, serum creatinine and serum lipid profiles were significantly increased by feeding with hyperlipidemic diet. These parameters were also non-significantly attenuated by administration of VAK. S.GOT, Aspartate transaminase (AST) and alkaline phosphatase activities were significantly enhanced by feeding with hyperlipidemic diet in rats. VAK significantly attenuated activity of these enzymes in comparison to cholesterol control group. Further total bilirubin and serum uric acid levels were also elevated by feeding of hyperlipidemic diet and VAK significantly attenuated them.

Histopathological sections from control group shows normal cytoarchitecture of liver, kidney and heart (Fig. 1A, 2A and 3A). In contrast, hyperlipidemic diet produced perivascular cell infiltration and micro fatty changes in liver, cell infiltration and fatty changes in kidney and cell infiltration and fatty changes in majority of sections of heart (Fig. 1B, 2B and 3B). Simultaneous treatment with VAK significantly attenuated cholesterol induced pathological changes in all the three organs (Fig. 1C, 2C and 3C).

Table 4: Effect of on various serum bio-chemical parameters

Parameters	*NC*	*CC*	*% change in comparison to NC*	*VAK*	*% change in comparison to CC*
Blood sugar (mg/dL)	87.50± 4.33	111.67± 3.34**	27.62↑	101.67± 7.44	08.95↓
Cholesterol (mg/dL)	58.00± 2.87	87.33± 2.40***	50.56↑	80.50± 4.93	07.82↓
Triglyceride (mg/dL)	81.16± 2.54	164.00± 16.54***	102.06↑	134.16± 15.07	18.19↓
LDL+VLDL (mg/dL)	27.50± 2.51	42.80± 4.42*	55.63↑	42.73± 4.38	–
HDL (mg/dL)	30.50± 1.25	39.50± 3.08*	29.50↑	38.00± 3.58	03.79↓
Apolipoprotein B	19.83± 0.30	14.16± 0.94***	28.59↓	17.16± 0.87$^{\alpha}$	21.18↑
Blood urea (mg/dL)	75.33± 3.48	42.33± 1.05***	43.80↓	44.80± 4.56	06.37↑
Serum creatinine (mg/dL)	0.48± 0.03	0.68± 0.03***	41.66↑	0.60± 0.03	11.76↓
S.GOT (IU/L)	152.50± 5.29	236.00± 25.73**	54.75↑	144.66± 11.22$^{\alpha\alpha}$	38.70↓
S.GPT (IU/L)	46.67± 3.31	73.00± 4.81**	56.41↑	63.66± 2.01$^{\alpha}$	12.79↓
Alkaline phosphatase (IU/L)	211.16± 9.81	526.33± 22.64***	149.25↑	402.50± 28.00$^{\alpha\alpha}$	23.52↓
Bilirubin total (mg/dL)	0.35± 0.02	0.55± 0.06*	57.14↑	0.35± 0.05$^{\alpha}$	36.36↓
Bilirubin D (mg/dL)	0.15± 0.02	0.20± 0.02	33.33↑	0.15± 0.02	25.00↓
Uric acid (mg/dL)	0.90± 0.10	1.35± 0.15*	50.00↑	0.80± 0.11$^{\alpha}$	40.74↓

Data: Mean ± SEM, ↑ - Increase; ↓ - Decrease,

*P<0.05, **P<0.01, ***P<0.001(Compared with normal control group, unpaired 't' test)

$^{\alpha}$ P<0.05, $^{\alpha\alpha}$ P<0.01 (Compared with cholesterol control group, unpaired 't' test)

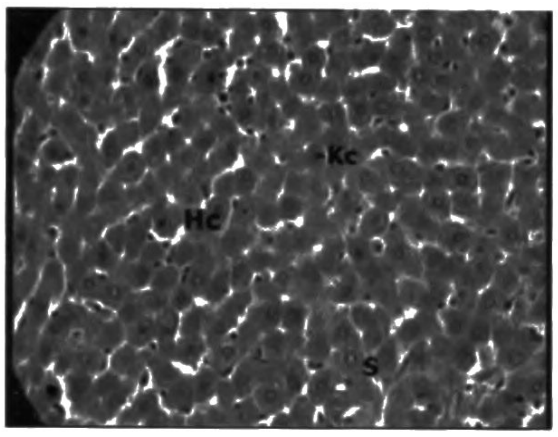

Figure 1A. NC-liver showing normal cytoarchitecture, Hc-hepatocytes Kc-Kupffer cell, S-sinusoid.

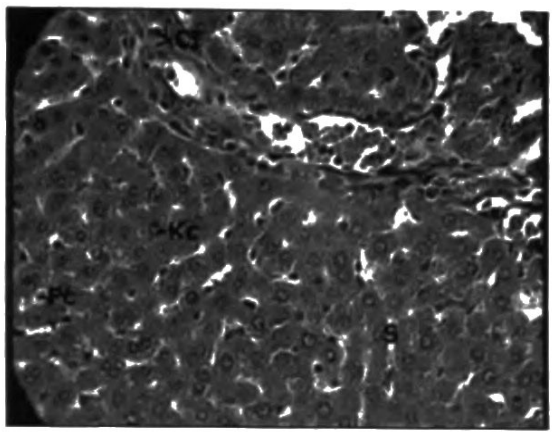

Figure 1B. CC-liver showing micro and macro (Fc) fatty changes, (CI) cell infiltration.

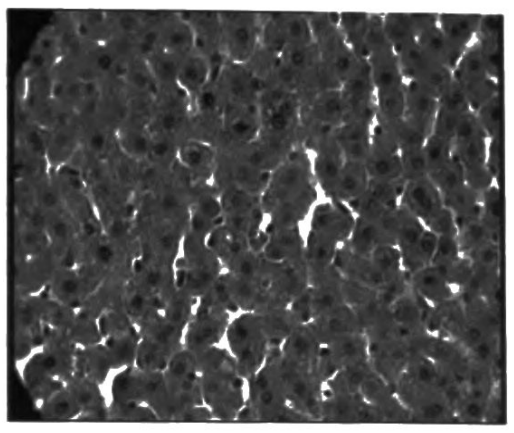

Figure 1C. VAK liver showing almost normal cytoarchitecture.

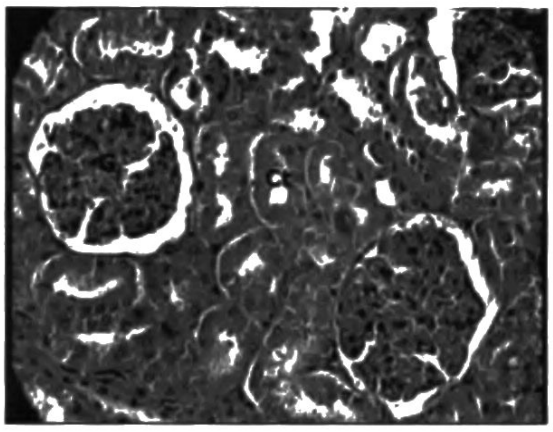

Figure 2A. NC-kidney showing normal cytoarchitecture G-glomerulus; Ct-convoluted tubule.

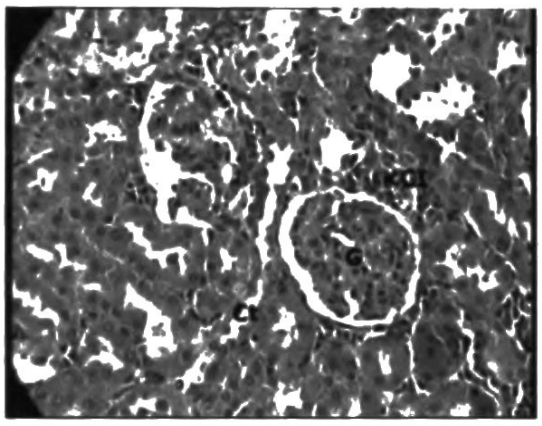

Figure 2B. CC-kidney showing micrb (Fc) fatty changes, and (CI) cell infiltration.

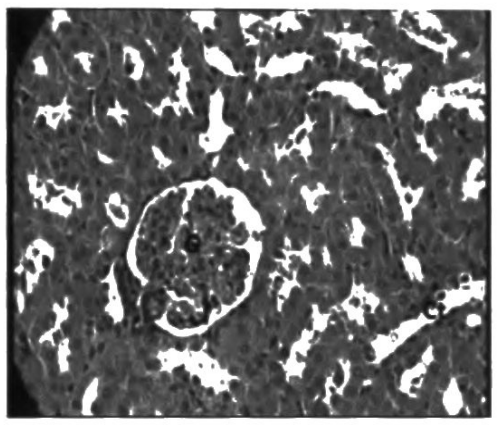

Figure 2C. VAK kidney showing almost normal cytoarchitecture.

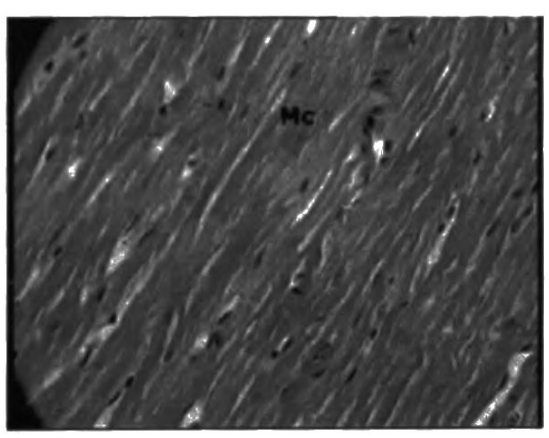

Figure 3A. NC-heart showing normal cytoarchitecture, Mc-myocardium.

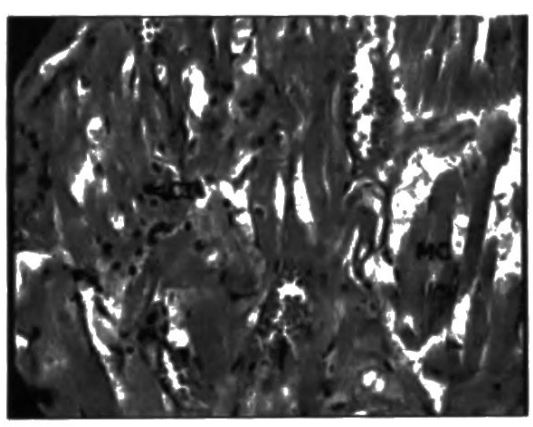

Figure 3B. CC-heart showing CI - cell infiltration.

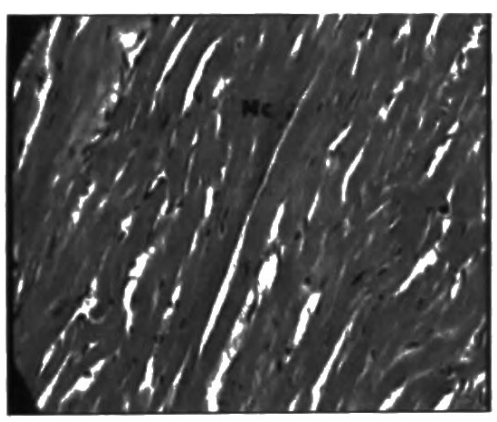

Figure 3C. VAK heart showing normal cytoarchitecture.

Discussion

Elevated levels of different types of lipids have been implicated in the production of atherosclerosis. In this stipulation the blood vessel wall thickens due to cholesterol deposition ensuing to inflammatory reaction. This ultimately leads to loss of elasticity of affected vessel wall and becomes the major pathology involved in the occurrence of a number of serious systemic disorders such as cardio vascular diseases, cerebro-vascular accidents, peripheral arterial disease which account as the significant culprit for mortality/disability in both developed and developing countries. Even after the prescription of dietetic, lifestyle and therapeutic interventions the incidence and prevalence of lipid abnormalities and resultant fatal complications are hiking up. Hence there is huge scope for the introduction of effective hypolipidemic and anti-hyperlipidemic drugs in to existing therapeutic armamentarium.

In the current experimental work, in comparison to cholesterol control group, the VAK treated animals exhibited moderate level of decrease in S.cholesterol, S.triglycerides and HDL-C, but the variations were statistically non significant.

Statistically significant changes were attained in the values of SGOT, SGPT, alkaline phosphatase, total bilirubin and uric acid revealing the high hepatoprotective and nephroprotective properties of the test formulation. SGOT determination is of immense value in the assessment of coronary artery diseases and myocardial infarction.

Elevated serum enzyme activity associated with cardiac disorder is assumed to reflect activity of enzyme released from the injured cardiac tissue too [20]. The significant change attained in the value of SGOT may also have noteworthy role in the cardio-protective activity of the trial drug. The observations attained in bio chemical parameters are in line with the histopathological findings of this study. The normal cytoarchitecture and absence of cholesterol induced pathological changes in the histopathological sections of liver, heart and kidney shows the efficacy and capability of VAK in the management of dyslipidemia induced complications.

Vara is well known as *triphala* (combination of *Terminalia chebula*, *Terminalia bellerica* and *Emblica officinalis*) in Ayurveda is the foremost ingredient of VAK and Ayurvedic science has identified its benefits in obesity, diabetes mellitus and hepatic disorders. There are many reports with regard to pharmacological effects of triphala, including its anti-hypercholesterolemic, anti-oxidant and hepatoprotective properties [21,22]. *Emblica officinalis (Amalaki)* given in a ration of rabbit at 1g/kg has found to have anti-hypercholesterol activity. In one of the study; *T. arjuna, T. bellerica* and *T. chebula* was fed to rabbits on cholesterol rich diet inducing atherosclerosis which showed that *T.chebula* as the most potent hypolipidemic agent among the three drugs and induced partial inhibition of rabbit atheroma as seen from plasma and tissue lipid content and the lesions of aorta. *Haritaki (T. chebula)* is also well known for its anti-hepatotoxic activities. Hepatoprotective activity of *T.bellerica* is also been reported as the alcoholic extract of fruit of *T. bellerica* in a dose of 30 mg/kg given I/V to dogs showed significant bile stimulant activity and increased solids in bile secretion. Further numerous studies have been conducted on the anti-hyperlipidemic acivity of *Citraka (Plumbago zeylanica)*, which is the one of the ingredient of VAK. In the study conducted on hyperlipidemic rabbits – Plumbagin; the active constituent of *Plumbago zeylanica* reduced serum cholesterol by 53-86% and elevated decreased HDL cholesterol significantly [23].

Curcuma longa (Haridra) is an established hepato protective drug and is been used widely in the management of jaundice and hepatic disorders [24]. *C. longa* prevents the formation of fatty liver by the modulation of expressions of enzymes that are important to fat metabolism [25].

In a usual mutant obesity, *Curcuma longa* had significantly reduced cholesterol and triglyceride concentration, while increasing HDL cholesterol. Advance evidences indicate that it diminishes the oxidation of LDL, blood glucose and renal lesions. It had been demonstrated to reduce smooth muscle cell proliferation and endothelial dysfunction [26]. As per recent research studies Curcumin has been reported to have the nephroprotective effect to improve creatinine and urea clearance and also can protect the chronic renal allograft nephropathy [27].

Furthermore the hypolipidemic and hepatoprotective activities of *Pterocarpus marsupium (Asana)* are also well established by several studies [28,29]. Most of the Ayurvedic drugs such as *E. officinalis* and *C. longa* are stronger and efficient anti-oxidants; which may be helpful in preventing lipid peroxidation [30].

Thus multiple constituents of VAK are reported to have antihyperlipidemic, anti-hepatotoxic and hepato-protective activities. The ingredient such as *Curcuma longa* is having nephroprotective properties also and the same is reflected in present study. The non-significant changes obtained in the most of the values of lipid profile and blood sugar cannot be interpreted negatively, as the results are definitely pointing towards the direction of reduction. The weak action obtained in terms of these parameters may be because of the fact that the drug is administered in the form of decoction. Otherwise most of the individual components of the *Vara Asanadi Kwatha* are proven anti-hyperlipidemic drugs when used in single or in combinations. The alcoholic extract of the same drugs may show more potent and significant anti-hyperlipidemic activity as reported by the various studies in this regard.

Conclusion

From the present study it can be concluded that *Vara Asanadi Kwatha* is having mild anti-hyperlipidemic and remarkable hepato-protective and nephroprotective activities. Exclusive experimental works on hepato-protective and nephroprotective properties of *Vara Asanadi Kwatha* may reveal hidden and highly informative facts regarding this wonderful classical formulation.

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