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Effect of crude extract of *Bombyx mori* coccoons in hyperlipidemia and atherosclerosis

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ABSTRACT

The silkworm is the larva or caterpillar of the domesticated silkmoth, *Bombyx mori* and being a primary producer of silk is an economically important insect. These days the silk is emerging as a resource for solving a broad range of biological problems. The silk (*Abresham*) is popularly known as *Abresham muqriz* (muqriz means cut) in Unani medicine. Its cocoons are extensively used as an ingredient of various Unani formulations like Khameer-E- Abresham Sada, Khameere Abresham Hakeem Arshad Wala, Khameere Abresham Ood Mastagi Wala etc. and are used to treat many cardiac and nervous disorders. The hypolipidemic activity of this drug, along with *Nepata Hindostana* (Badranjboya) and *Terminalia Arjuna* (Arjan) has been documented. But action of extract of *Bombyx mori* cocoons as a single drug is not documented. That's why; it was decided to study its effect on hyperlipidemia and atherosclerosis. The Male New Zealand White rabbits all of 1.5kgs were selected for the study. After stabilization period (2 weeks) the rabbits were divided into 3 groups (Group I - Control, Group II Lesion Control and Group III treated with extract of *Bombyx mori* silk cocoon). Hyperlipidemia and atherosclerosis were induced with 1% cholesterol diet. After induction of hyperlipidemia and atherosclerosis for twelve weeks, Group III rabbits were treated with *Bombyx mori* for 6 weeks (45 days). A significant decrease in hyperlipidemia was seen within 4 weeks of treatment. Histopathologically, the atherosclerotic plaques showed reduction in size. The third group showed a significant increase in the body weight and also an increase in the HDL cholesterol levels. The study concludes that extract of *Bombyx mori* cocoons has a significant effect on hypercholesterolemia and atherosclerosis probably because of its antioxidant and hypolipidemic effect.

Key words: Atherosclerosis, *Bombyx Mori*, hyperlipidemia, silk worm, cocoon extract

INTRODUCTION

Atherosclerosis is a killer disease and is a major cause of death throughout the world. It can affect any blood vessel supplying the vital organs. For example, it can affect the carotid artery and cause severe neurological complications leading to cerebral infarction and stroke or coronary arteries causing ischemic heart disease, which, if left untreated, can lead to complications such as myocardial infarction and death.[1] A strong association between certain types of dyslipidemia, including hypercholesterolemia, hypertriglyceridemia and combined hyperlipidemia with development of atherosclerotic lesions has been documented by a number of clinical trials and epidemiological and experimental studies.[2-4]

Atherosclerosis has a ubiquitous prevalence throughout the world, especially in the elderly. Epidemiological studies correlated in India have suggested a higher incidence of atherosclerosis in the recent past.[5]

The true frequency of atherosclerosis is difficult, if not impossible, to accurately determine because it is a predominantly asymptomatic condition for a long and considerable period of time. In the United States, about 80 million people, (36.3% of the population), have existing cardiovascular diseases. In addition 795,000 people suffer new or recurrent strokes each year.[6] The frequency of coronary heart disease in the Far East is significantly lower than that documented in the West. Ill-defined genetic reasons for this phenomenon may

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exist, but significant interest surrounds the role of diet and other environmental factors in the absence of clinical atherosclerotic vascular disease in these populations. Atherosclerotic cardiovascular disease is also rare on the African continent, although growing evidence indicates that this too is changing as a result of rapid westernization and urbanization of the traditionally rural and agrarian African populations. The prevalence of coronary heart disease is also increasing in the Middle East, India, and Central and South America.[7]

Unani concept of atherosclerosis and hyperlipidemia

According to the unani concept derangement of the functions of the liver (su-e-mizaj-e-jigar) directly affects the quality and consistency of the Kilth – e – balgam, which in the modern concept fits the prototype of lipid abnormalities. Lipid abnormalities are balgam – e – ghair tabaee. The abnormal kilth thus produced can be more viscid rather than dry and may make the flow of blood through the smaller vessels difficult and thus may cause ischemia. If derangement of liver continues, the kilth e balgam is subject to more heat (Ihtiraq –burn) and kilth e balgam may burn to change into Sauda (Vata),[8] which is very dry and may affect the blood vessels leading to obstruction of blood flow causing ishcemia and infarction.[9]

Bombyx mori is an economic insect whose silk is emerging as a resource for solving a broad range of biological problems.[10] It is known as *Abresham* popularly known as *abresham Muqirz* (Muqriz means cut) in Unani[11] and is used as a raw drug in various formulations like *Khameer-E- Abresham Sada, Khameere Abresham Hakeem Arshad Wala, Khameere Abresham Ood Mastagi Wala etc.*, for many cardiac and neurological disorders.[14] The protective role in hyperlipidemia of the crude extract of *Bombyx mori* cocoons along with two other drugs *Nepata hindostana (Badranjboya)*[12] and *Terminalia arjuna (Arjan)*[13] has been documented and found to have a good antioxidant effect and protective role in hyperlipidemia.[15] In Unani pharmacopoeia several formulations with large or small number of ingredients have been described for the treatment of cardiovascular and cerebrovascular disorders. Some of the formulations are being used in Unani medicine with good results.[16] But there is no definitive data regarding dose and effect relationships for the extract of cocoons of *Bombyx mori*. This led to the logical decision of evaluating the role of extract of silk cocoons of *Bombyx mori* as a single drug in induced hyperlipidemia by experimental methods.

MATERIALS AND METHODS

18 New Zealand white rabbits, all male procured from Tamil Nadu Veterinary and Animal Science University (TANUVAS) Tamil Nadu, cholesterol powder (sigma, USA) and *Bombyx mori* cocoons were used for this study.

Properties of *Bombyx mori* silk *(Abresham Muqriz)*

Bombyx mori silk is attributed the qualities 'hot' and 'dry' in its temperament (Garm – o – Khushk). It is a cardiac tonic and a nervous stimulant. It is an expectorant and removes excess 'Kapha' from the blood (Blagam – e – ghair tabaee – Pathological Phlegm).[17] Recent advancement have shown that It is been used to treat palpitations, hypertension and heart diseases, which occurs due to hardening of arteries (Salabath – e – Shiryani).[18]

METHODS

Male New Zealand White rabbits (1.5 kg weight) were housed individually in separate steel cages under temperature-controlled conditions. During the 2-week period of adaptation, the rabbits were fed standard rabbit chow and water ad libitum. Food intake was assessed on a daily basis. After stabilization the rabbits were divided into 3 groups, each group containing 6 rabbits as follows:

Group I

Rabbits were fed with standard diet pellets.

Group II

Rabbits received cholesterol powder (1%) with coconut oil (4%) in addition to their standard diet pellets for all the 18 weeks.

Group III

Rabbits received cholesterol powder (1%) with coconut oil (4%) in addition to their standard diet pellets for 12 weeks. Extract of *Bombyx mori* cocoons 50mg/100gm body weight was added to it for remaining 6 weeks (45 days).

All the 12 New Zealand White Rabbits (Group II and III) were fed with atherogenic diet, i.e 1% cholesterol powder (supplied by sigma, USA) mixed with coconut oil 4%)[19] was drawn in to a sterile syringe fed slowly through per oral route through out the experiment for 18 weeks.

Reason for using male New Zealand white rabbits

Numerous epidemiological studies suggest that estrogens protect women against cardiovascular diseases before the age of menopause. After menopause, the cardiovascular risk of female becomes progressively closer to that of men, suggesting an atheroprotective effect of estrogens.[20,21] In experimental models of atherosclerosis, generally studied at the initial stages of the atherosclerotic process, i.e. at the stage of fatty streak constitution in various animal species, Estradiol (E2) treatment prevents the development of fatty streaks.[22,23]

Determination of serum cholesterol

Blood samples were collected from marginal ear vein, after an overnight fast, at an interval of 2 weeks. Serum was analyzed for cholesterol, triglycerides, LDL, HDL, SGOT, SGPT and creatinine. Serum was analyzed for total cholesterol by an automatic analyzer at the laboratory of International Centre for Cardio thoracic and Vascular Diseases.

Bombyx mori crude extract

The *Bombyx mori* cocoons used in this study were obtained from reputed commercial sources for indigenous drugs. The silk cocoons were cut open. The remains of the silk moth were removed. Then the cocoons were cut into fine pieces. Daily 10gms of these finely cut flakes of cocoon were soaked in 1% Nacl solution and kept in a shaker of 100 rpm overnight.[24] The next day the solution were transferred into centrifuge tubes and centrifuged at 3000rpm. Next the supernatants were collected. Protein estimation was done through standard Lowry's method[25] in order to know the protein concentration and calculate the dosage. 10gms of cocoons yielded 7 to 8gms of crude extract. This crude extract of *Bombyx mori* was given to the experimental rabbits of GROUP III with the dose of 50mg/100gm (Tajuddin *et al* 2008)[15] of body weight orally for 45 days.

The rabbits were weighed every second week with a standard digital balance [Tables 1, 2 and 3].

Statistical method

Statistical method – SPSS (Statistical Package for Social Sciences)

Tests – Linear model repeated measurement technique was applied using SPSS software.

RESULTS

Weight

At the beginning of the experiment, the rabbits of group I, were within normal range each weighing above 1.5 kgs with an average mean value of 1.17±0.18 to 1.81±0.08 kgs (P<0.05) (SPSS) [Tables 1 and 4].

Group II rabbits in the beginning of the experiment (0 week) had a weight of 1.69±0.18 kgs [Table 1] showed an increase in the 12th week was recorded. This later decreased to 1.52±0.06 kgs [Tables 2 and 4] in the 18th week. The rabbits of Group III gained significant weight. The weight was increased from 1.97±0.12kg to 2.30±20 kg (P<0.05) [Tables 2 and 4] when compared to the Group II and I

Lipid profile

The average fasting total cholesterol levels in all the six rabbits of group I were 37.17±5.64 (P<0.05) in the 0 week [Tables 1 and 4] and in the 12th [Table 1] and 18th week it remained with in normal limits with an average of 83.83±22.56 mg/dl [Tables 1 and 4].

In Group II the fasting Cholesterol levels were 51.50±16.07mg/dl (P<0.05) The triglyceride levels were 65.67±20.05mg/dl (P<0.05), HDL (High Density Lipoproteins) were 30.83±6.31mg/dl (P<0.05), LDL (Low Density Lipoproteins) were 23.83±5.34mg/dl (P<0.05) in the 0th week. It increased to 1958.17±99.24 mgs/dl, TGL

Table 1: Weight, lipid profile, liver enzymes and creatinine of the New Zealand white rabbits of the group I (Control) taken every two weeks for 18 weeks

Group I (Control)					
	0Week	2Week	4Week	6Week	8Week
WEIGHT - Kg	1.71±0.18	1.71±0.20	1.74±0.18	1.73±0.16	1.71±0.17
TC – mg/dl	37.17±5.64	38.97±6.91	40.67±6.35	42.17±7.76	45.50±12.32
TGL – mg/dl	44.83±10.50	43.17±11.03	45.50±8.36	43.83±16.07	30.83±9.97
HDL – mg/dl	23.00±3.46	22.15±4.99	27.50±5.96	27.67±6.65	28.50±8.62
LDL- mg/dl	24.83±2.40	50.00±58.88	26.83±4.88	25.00±10.18	26.33±8.57
SGOT – IU/l	34.00±4.47	34.47±6.59	38.83±4.83	45.83±16.67	41.17±5.23
SGPT – IU/l	42.05±4.53	45.90±5.49	48.67±6.35	52.00±6.60	55.00±8.00
CREATININE mg/dl	1.00±0.14	1.13±0.21	1.22±0.13	1.27±0.34	1.42±0.23
Group I (Control)					
	10Week	12Week	14Week	16Week	18Week
WEIGHT - Kg	1.74±0.11	1.75±0.16	1.78±0.12	1.77±0.09	1.81±0.08
TC – mg/dl	46.17±8.54	47.33±10.46	79.17±20.75	79.83±21.28	83.83±22.56
TGL – mg/dl	48.33±16.22	37.17±11.13	48.83±15.38	36.33±8.45	34.33±7.84
HDL – mg/dl	27.13±6.41	28.17±7.03	29.83±6.18	31.33±7.12	31.83±6.91
LDL- mg/dl	30.00±11.52	33.33±11.06	32.67±9.33	33.33±9.37	33.67±7.37
SGOT – IU/l	41.83±5.31	41.50±6.06	40.67±5.65	41.83±6.43	42.50±5.01
SGPT – IU/l	55.67±9.69	56.00±11.05	56.83±17.72	57.83±15.29	61.50±18.03
CREATININE mg/dl	1.42±0.13	1.33±0.25	1.33±0.39	1.45±0.40	1.35±0.41

Table 2: Weight, lipid profile, liver enzymes and creatinine of the New Zealand white rabbits of the Group II (Lesion control) taken every two weeks for 18 weeks

Group II (Lession control)					
	0 Week	2Week	4Week	6Week	8Week
WEIGHT - Kg	1.69±0.18	1.74±0.14	1.89±0.16	1.87±0.16	1.89±0.10
TC – mg/dl	51.50±16.07	389.33±106.70	460.00±131.49	529.83±91.04	950.83±100.94
TGL – mg/dl	65.67±20.05	99.33±36.78	149.33±48.88	282.50±105.15	411.67±98.91
HDL – mg/dl	30.83±6.31	28.50±7.97	31.67±8.91	30.67±14.90	31.33±12.96
LDL- mg/dl	23.83±5.34	332.83±68.15	398.17±99.31	581.00±141.35	586.33±129.04
SGOT – IU/l	27.50±7.45	34.67±10.71	39.50±10.25	64.33±16.49	64.33±16.49
SGPT – IU/l	36.67±5.16	38.50±11.11	46.33±11.29	55.33±12.71	54.67±12.68
CREATININE mg/dl	0.92±0.26	0.77±0.29	0.78±0.19	1.03±0.16	0.92±0.17
Group II (Lession control)					
	10Week	12Week	14 Week	16Week	18Week
WEIGHT - Kg	1.81±0.12	1.72±0.11	1.74±0.11	1.66±0.08	1.52±0.06
TC – mg/dl	1411.67±225.43	1958.17±99.24	1791.33±348.24	1857.67±346.90	1955.33±317.20
TGL – mg/dl	516.00±43.02	736.33±124.74	561±74.25	601.83±78.65	538.83±54.41
HDL – mg/dl	36.83±14.05	43.83±14.37	16.67±3.56	17.33±2.25	19.00±3.35
LDL- mg/dl	627.33±152.98	685.17±194.74	709.17±60.36	761.67±66.17	852.17±60.78
SGOT – IU/l	66.67±21.59	70.33±17.47	53.00±14.34	52.67±7.39	55.83±10.53
SGPT – IU/l	59.33±13.98	67.00±11.87	51.67±13.22	53.33±14.32	62.00±4.69
CREATININE mg/dl	1.23±0.23	1.40±0.23	1.50±0.32	1.48±0.33	1.50±0.24

Table 3: Weight, Lipid Profile, Liver Enzymes and Creatinine of the New Zealand White Rabbits of the Group III (extract of *Bombyx mori* cocoons + Atherogenic Diet) taken every two weeks for 18 weeks

GROUP III (extract of *Bombyx mori* cocoons + Atherogenic Diet)					
	0 Week	2Week	4Week	6Week	8Week
WEIGHT - Kg	1.97±0.21	2.01±0.19	2.03±0.19	2.08±0.18	2.04±0.19
TC – mg/dl	52.83±13.27	273.83±74.48	443.83±106.07	606.83±121.87	941.67±168.27
TGL – mg/dl	52.17±16.24	161.50±45.49	248.50±54.73	351.50±42.61	398.33±58.44
HDL – mg/dl	16.00±4.98	15.67±4.55	18.67±5.43	21.50±3.62	15.50±5.36
LDL- mg/dl	28.17±8.13	246.50±34.07	329.83±38.49	444.50±39.06	508.50±52.27
SGOT – IU/l	30.33±7.17	27.12±6.62	41.17±26.54	33.83±24.57	39.67±29.06
SGPT – IU/l	43.00±5.55	52.67±12.13	72.33±12.13	48.17±12.19	39.50±4.59
CREATININE mg/dl	0.94±0.18	1.20±0.36	1.00±0.13	0.82±0.19	1.13±0.18
GROUP III (extract of *Bombyx mori* cocoons + Atherogenic Diet)					
	10Week	12Week	14Week	16Week	18Week
WEIGHT - Kg	2.09±0.16	2.09±0.20	2.15±0.19	2.24±0.18	2.30±0.20
TC – mg/dl	1395.00±238.55	1848.17±242.03	1476.17±234.82	779.17±126.42	422.00±74.79
TGL – mg/dl	448.17±45.21	510.67±56.55	381.17±42.61	289.33±57.47	181.67±35.25
HDL – mg/dl	15.33±6.12	15.33±4.08	24.00±4.60	30.67±3.93	42.17±6.11
LDL- mg/dl	555.00±32.49	621.67±25.32	467.17±38.91	362.83±47.69	228.50±46.28
SGOT – IU/l	34.00±21.67	41.17±18.14	50.00±23.79	42.67±27.83	29.83±10.80
SGPT – IU/l	53.00±11.21	40.67±5.05	49.00±8.65	39.50±10.52	36.33±12.48
CREATININE mg/dl	1.17±0.23	1.15±0.32	1.27±0.38	1.00±0.27	1.25±0.33

736.33±124.74 mgs/dl, LDL 685.17±194.74mg/dl and HDL dropped to 43.83±14.37 mg/dl in the 12th week of experiment. At the end of 18th week the values remain the same [Tables 2 and 4] confirming the atherogenic diet on the lipid profile.

The fasting total cholesterol levels in Group III rabbits started with a mean average of 52.83±13.27mg/dl (P<0.05), triglycerides was 52.17±16.24mg/ dl(P<0.05), HDL (High Density Lipo Proteins) was 16.00±4.98mg/dl(P<0.05) and the LDL (Low Density Lipoproteins) was 28.17±8.13mg/dl(P<0.05) [Tables 3 and 4]. During the 12th week the serum levels of total cholesterol, triglycerides, HDL and LDL increased. The serum cholesterol levels were found up to 1848.17±242.03mg/dl (P<0.05), triglycerides was 510.67±56.55mg/dl (P<0.05), HDL came down to 15.33±4.08 (P<0.05) and the LDL levels shot up to 621.67±25.32 (P<0.05) and the values of total cholesterol,

triglycerides and LDL were very high and matched those in the Group II (Lesion Control) vide [Tables 2 and 4]. However, the most remarkable part of this experiment was seen after the 14th week where these values started steadily coming down to 1467±234.82mg/dl, 779.17±126.42mg/dl in the 16th week and to 422.00±74.79mg/dl [Tables 3 and 4] during 18th week of the experiment. Similar remarkable changes were seen in the TGL, HDL and LDL values as well. Improvement in the HDL was recorded during 16th and 18th week, which increased from 30.67±3.93mg/dl to 42.17±6.11mg/dl (P<0.05) [Tables 3 and 4]. When compared to Group II the levels were significant. These observations suggest that the action of the crude extract of *Bombyx mori* cocoons is slow to begin with, however has an extremely desirable effect within 16th week and dramatic improvement in the weight from 1.97±0.21 kg in the 0th week to 2.30±0.20kg(P<0.05) [Tables 3 and 4] in the 18th week.

SGOT, SGPT and creatinine

The liver enzymes and creatinine levels in the group III were normal and matched with Group I confirming that the crude extract of *Bombyx mori* cocoons was neither hepatotoxic or nephrotoxic [Tables 1,3 and 5]

Table 4: Lipid profile and weight of the rabbits Cholesterol, phospholipid and triglyceride values of Group I, II and III drug treated rabbits (mg/ dl*)

	0 week	12th week	18th week
Weight			
Group I (Control)	1.71±0.18	1.75±0.16	1.81±0.08
Group II (Lesssion Control)	1.69±0.18	1.72±0.11	1.52±0.06
Group III(*Bombyx mori*).	1.97±0.21	2.09±0.20	2.30±0.20
Cholesterol			
Group I (Control)	37.17±5.64	47.33±10.46	83.83±22.56
Group II (Lesssion Control)	51.50±16.07	1958.17±99.24	1955.33±317.20
Group III (*Bombyx mori*)	52.83±13.27	1848.17±242.03	422.00±74.79
Triglyceride			
Group I (Control)	44.83±10.50	37.17±11.13	34.33±7.84
Group II (Lesssion Control)	65.67±20.05	736.33±124.74	538.83±54.41
Group III (*Bombyx mori*)	52.17±16.24	510.67±56.55	181.67±35.25
HDL			
Group I (Control)	23.00±3.46	28.17±7.03	31.83±6.91
Group II (Lesssion Control)	30.83±6.31	43.83±14.37	19.00±3.35
Group III(*Bombyx mori*)	16.00±4.98	15.33±4.08	42.17±6.11
LDL			
Group I (Control)	24.83±2.40	33.33±11.06	33.67±7.37
Group II (Lesssion Control)	23.83±5.34	685.17±194.74	852.17±60.78
Group III (*Bombyx mori*)	28.17±8.13	621.67±25.32	228.50±46.28

P=<0.05

Histopathology

Routine Hematoxylin and Eosin staining method was followed.

Histopathologically the aorta of group I was normal: No atherosclerotic plaque was seen in them [Figure 1]. The sections of the aorta of group II showed extensive atheromatous plaques filled with foam cells. Our study matched with H.P. Shaila *et al* 1998[26] where there were enormous atherosclerotic plaques in the experimental rabbits in which cholesterol only was fed. Section of aorta of Group III [Figure 3] rabbits treated with the crude extract of *Bombyx mori* cocoons and atherogenic diet showed focal areas of atherosclerotic lesion on comparison with the lesion control Group II [Figure 2] rabbits, the size of the atherosclerotic plaques was seen to be significantly reduced in size [Figure 3].

Areas of sclerosis were seen in the sections of aorta stained with Elastic Vangeson stain in both group II and group III, but the sclerosis was found less in the group III [Figure 4 – areas of sclerosis marked with yellow arrow].

DISCUSSION

Weight of the rabbits in group I was 1.81±0.08 kgs at the end of 18th week. This was normal as served as control.

Table 5: Liver enzymes and creatinine of the experimental rabbits SGOT, SGPT and Creatinine values of Group I, II and III rabbits in (U/L) and (mg/dl)

	0 week	12th week	18th week
SGOT			
Group I (Control)	34.00±4.47	41.50±6.06	42.50±5.01
Group II (Lesssion Control)	27.50±7.45	70.33±17.47	55.83±10.53
Group III (*Bombyx mori*)	30.33±7.17	41.17±18.14	29.83±10.80
SGPT			
Group I (Control)	42.05±4.53	56.00±11.05	61.50±18.03
Group II (Lesssion Control)	36.67±5.16	67.00±11.87	62.00±4.69
Group III (*Bombyx mori*)	43.00±5.55	40.67±5.05	36.33±12.48
Creatinine*			
Group I (Control)	1.00±0.14	1.33±0.25	1.35±0.41
Group II (Lesssion Control)	0.92±0.26	1.40±0.23	1.50±0.24
Group III (*Bombyx mori*)	0.94±0.18	1.15±0.32	1.25±0.33

(*P*=<0.05)

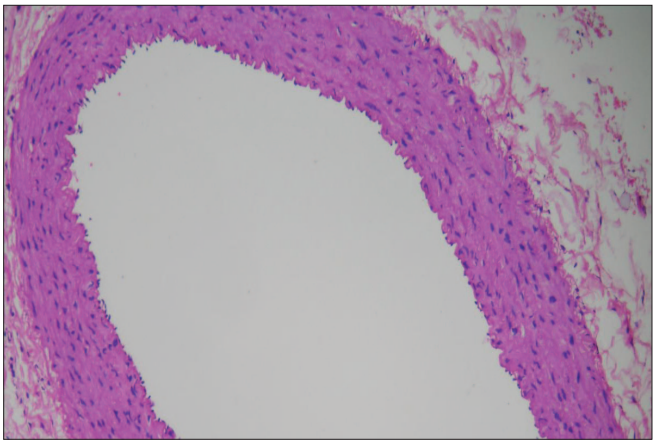

Figure 1: Histopathology results group 1 normal control h&e sections showing a normal aorta

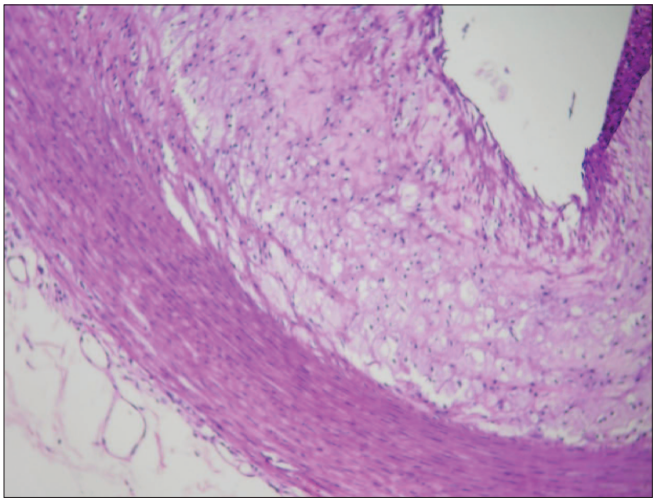

Figure 2: Histopathology results of group 11 lession control h&e sections of aorta showing a huge atherosclerotic plaque

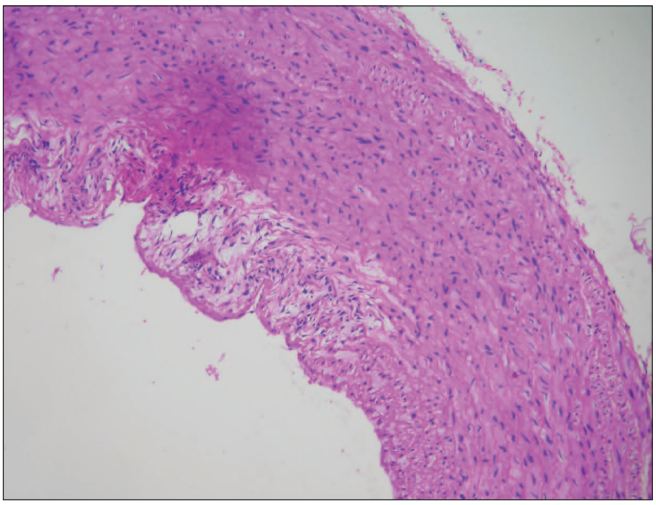

Figure 3: Histopathology results of group 3 *bombyx mori* treated h&e sections of aorta showing a reduced atherosclerotic

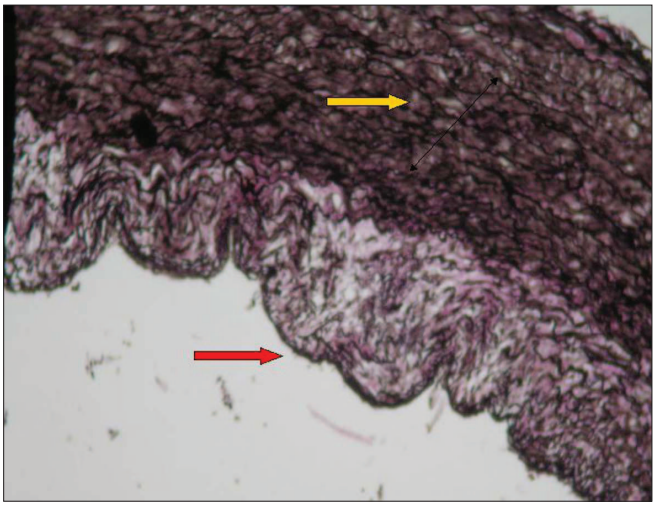

Figure 4: Group 3 *bombyx mori* treated evg sections of aorta showing a reduced atherosclerotic plaque with area of sclerosis (yellow arrow)

In group II weight had come down being 1.52±0.06kgs and was less when compared with Group I and III. In group III the rabbits gained significant weight. The weight increased from 1.97±0.12kg to 2.30±20 kg (P<0.05) when compared to the Group I and Group II. This remarkable finding is an evidence that the crude extract of *Bombyx mori* cocoons improves the general condition by reducing cholesterol perhaps by increasing the anti oxidant status in the rabbits.[15]

Group I control had no change in the lipid profile (Total Cholesterol 83.83±22.56mg/dl, Triglycerides 34.33 ±7.84 mg/dl, HDL 31.83±6.91mg/dl and LDL 33.67±7.37mg/dl (P<0.05); histopathological appearance were normal and were similar to the findings of H.P. Shaila *et al*.[26]

Group II showed higher degrees of lipid levels (Total Cholesterol 1955.33±317.20mg/dl, Triglycerides 538.83±54.41mg/dl, HDL 19.00±3.35 and LDL 852.17±60.78 mg/dl (P<0.05) at the end of 18th week (135th Day). This was not surprising since this group of rabbits were fed with atherogenic diet (only cholesterol) through the experiment. Histological examination showed huge atherosclerotic plaques in aorta. The body weight was 1.52±0.06 kgs (P<0.05); which was comparatively low.

Group III crude extract of *Bombyx mori* cocoons with Cholesterol diet showed a reduction in lipid levels (Total Cholesterol 422.00±74.79mg/dl, Triglycerides 181.67±35.25mg/dl, HDL 42.17±6.11mg/dl, LDL 228.50±46.28mg/dl (P<0.05)) this findindgs were similar to the findings of Ghule BV *et al* 2006.[27] This shows, that the crude extract of *Bombyx mori* cocoons has successfully lowered the rise of serum cholesterol level caused by the cholesterol rich diet probably due to the presence of amino acids such as histidine, tryptophan, cysteine etc in the silk cocoons which have been reported to act as antioxidants.[15]

In addition to decreased cholesterol levels significant increase in the body weight was noted (2.30±0.20kgs $P<0.05$) in all the rabbits of this group. However the size of atherosclerotic plaque had reduced in the aorta.

In atherosclerosis, there are three main processes at work. Injury of arterial wall and proliferation of its smooth muscle cells, lipid implantation into the arterial wall, and mural thrombosis over the altered wall leading to organization of the thrombus and its incorporation into the wall. The extract of *Bombyx mori* cocoons probably could have inhibited the second step of lipid implantation in the injured arterial wall, by its lipid lowering and anti oxidant property.

The Unani system is based on four – qoon (Rakta), Balgam (Kapha), Safra (Pitta), Sauda (Vata). Extract of *Bombyx mori* cocoons a cardio protectant and expectorant of phlegm and as per unani hyperlipidemia occurs due to accumulation of excess of pathological phlegm aggravated Kapha in the blood, the action of the drug probably may be by removing the accumulation of excess of pathological phlegm form blood thereby bringing down Hyperlipidemia and preventing atherosclerosis.

The above study indicates that the crude extract of *Bombyx mori* cocoons has a fairly good lipid lowering capability. It is also capable of decreasing the extent of atherosclerotic lesions even though not fully.

Thus we can conclude that extract of *Bombyx mori* cocoons has a definite effect on lowering lipid profile. The reduced levels of Cholesterol and increase in the HDL levels and weight probably due to its active component sericin, which is a protein, gives a positive scientific background to name extract of *Bombyx mori* cocoons as a cardio protective and neuro protective drug.

Future Work: More extensive work in an large group of rabbits would substantiate the significant contribution of Extract of *Bombyx mori* cocoons.

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