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Research Article

Reduction of Oxidative Stress by Layman

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Abstract

Regional body fat distribution has an important influence on metabolic and cardiovascular risk factors. Increased abdominal (visceral) fat accumulation is a risk factor for coronary artery disease (CAD), dyslipidemia, hypertension, stroke, and type 2 diabetes. The recent emphasis on treatment of the dyslipidemia of the metabolic syndrome (hypertriglyceridemia, reduced high-density lipoprotein, and increased small, dense low-density lipoprotein particle number) has compelled practitioners to consider lipid-lowering therapy in a greater number of their patients, as one in two individuals over age 50 has the metabolic syndrome. Individuals with the metabolic syndrome typically have normal low-density lipoprotein cholesterol levels, and current lipid-lowering guidelines may underestimate their cardiovascular risk. Two subgroups of patients with the metabolic syndrome are at particularly high risk for premature CAD. One, individuals with type 2 diabetes, accounts for 20-30% of early cardiovascular disease. The second, familial combined hyperlipidemia, accounts for an additional 10-20% of premature CAD. Familial combined hyperlipidemia is characterized by the metabolic syndrome in addition to a disproportionate elevation of apolipoprotein B levels. The measurement of fasting glucose and apolipoprotein B, in addition to the fasting lipid profile, can help to estimate CAD risk in patients with the metabolic syndrome. In this research we compared allopathic medication and medicinal herb in treating hyperlipidemia.

Place of Research: It was conducted at Ghurki trust teaching hospital, Lahore Pakistan from January 2016 to August 2016.

Consent: Written and already approved consent was taken from all enrolled participants.

Method: Ninety hyperlipidemic patients were selected from cardiology and medical wards of the hospital.

Exclusion Criteria: patients suffering from peptic ulcer disease, hypo or hyperthyroidism, kidney disease, any liver disease and cigarette smokers were excluded from the research study. They were divided in three groups, one at placebo therapy, another on Kalonji and third one on Vitamin B3.

Results: After one and half month, significant changes (p value ranging from <0.05 to <0.001) were observed in their LDL and HDL-cholesterol.

Conclusion: Kalonji is as good as vitamin B-3 in treating hyperlipidemia.

Keywords: coronary artery disease; dyslipidemia; hypertension; stroke

Introduction

Regional body fat distribution has an important influence on metabolic and cardiovascular risk factors. Many prospective studies have shown that increased abdominal (visceral) fat accumulation is an independent risk factor for CAD, hypertension, stroke, and type 2 diabetes (DM2) [1-5]. The strong link between increased abdominal (visceral) fat and hyperinsulinism, insulin resistance, elevated plasma free fatty acid (FFA) levels, hypertension, predisposition to thrombosis, hypertriglyceridemia, small, dense LDL particles, and reduced HDL has been recognized for decades, but until

recently there has been no uniform definition of this disease complex. The National Cholesterol Education Program (NCEP) and others have recently suggested the use of the term metabolic syndrome to identify the common cluster of metabolic abnormalities, defined as three or more of five criteria: 1) abdominal obesity (waist circumference, >102 cm in men and >88 cm in women), 2) hypertriglyceridemia [≥1.69 mmol/liter (≥150 mg/dl)], 3) low HDL <1.04 mmol/liter (<40 mg/dl) in men and <1.29 mmol/liter (<50 mg/dl) in women], 4) hypertension (≥130/85 mm Hg), and 5) elevated fasting glucose [≥6.1 mmol/liter (≥110 mg/dl) [6]. Even normal weight individuals with increased amounts of abdominal adipose tissue can be metabolically obese, with insulin resistance and dyslipidemia [7-12]. High Lipids levels can be treated by allopathic medications like statins, and vitamin B-3 [13]. Medicinal plants play remarkable role to control hyperlipidemia [14]. Hypolipidemic effects of Nigella Sativa are well known in medical science research groups [15]. Kalonji prevent absorption of dietary cholesterol from GIT, which reduce triglycerides levels in liver, thus VLDL are not synthesized which are main lipoproteins involved in biotransformation of LDL particles in human body [16]. Nigella sativa seeds used as a herbal medicine has many effects including cholerectic activity. It is reported that the Holy Prophet Muhammad (Peace be upon him) said, “Black seeds i.e. the seeds of Nigella sativa is a remedy for every disease except death” [17].

Material & Method

The study was conducted at Ghurki trust teaching hospital, Lahore Pakistan from January 2016 to August 2016. Ninty patients were selected for study. Consent was taken from all participants. Inclusion criteria was primary and secondary hyperlipidemic patients. Exclusion criteria was patients suffering from any kidney, liver and thyroid related disease. Name, age, gender, occupation, residential address, phone/contact number, previous medical history, disease in family history, drug history were recorded in specific Performa. Three groups I, II, and III were made (30 patients in each group). Group-I was allocated for placebo, to take placebo capsule once daily, after breakfast for six weeks. Group-II was advised to take 2 tea spoons of kalonji after breakfast for the period of six weeks. Group-III was on Niacin 2 grams in divided doses, after breakfast, lunch and dinner for 6 weeks. Their base line LDL-cholesterol and HDL-cholesterol level was estimated at the start of research work. Their serum was taken at follow up visits, fortnightly for lipid profile. Data were expressed as the mean±SD and ‘t’ test was applied to determine statistical difference in results. A p-value > 0.05 was considered as non-significance and P-value < 0.001 was considered as highly significant change in the differences. Serum LDL-cholesterol was calculated by formula (LDL-Cholesterol=Total Cholesterol-(Triglycerides/5 +HDL-Cholesterol). Serum HDL-cholesterol was determined by using kit Cat. # 3022899 by Eli Tech Diagnostic, France.

Data Inference & Results

Numerical values and results of all parameters of participated patients were analyzed biostatistically. In placebo group, LDL-cholesterol decreased from 189.15±3.90 mg/dl to 186.75±2.08 mg/dl, change in the parameter is 2.40 mg/dl. This difference in pretreatment and post treatment value is non-significant, ie; P-value > 0.05. HDL-cholesterol in placebo group increased from 36.11±2.11mg/dl to 37.17±1.51mg/dl. The difference in parameter was 1.06mg/dl. Statistically this change in parameter was nonsignificant, ie; P-value > 0.05. In Nigella sativa group, out of 30 hyperlipidemic patients, 27 patients completed over all study period. LDL-cholesterol in this group decreased from 202.45±1.54mg/dl to 189.52±2.21mg/dl. The difference in pretreatment and postreatment mean values is 12.93 mg/dl. Statistically this change in two mean values is highly significant, with p-value < 0.001. HDL-cholesterol in this group increased from 38.81±3.90 42.19±3.32mg/dl. Change in two mean values was 3.38mg/dl. Statistically this change is significant, with probability value <0.01. In group III, 28 patients completed the research. LDL-cholesterol in this group decreased from 212.65±2.32 to 185.61±3.43 mg/dl in six weeks treatment. Change in pre and post treatment mean values is 27.04mg/dl. Statistically this change is highly significant, i.e., P-value < 0.001. HDL-cholesterol increased from 39.19±2.01 to 43.00±3.07 mg/dl in six weeks. Change in two parallel values is 3.49mg/dl, which is significant with P-value <0.01.

LDL, HDL’s basic values (pre and after treatment) and their biostatistical significance

No. Of patients	Day-0 values	Day-45 values	Change in basic values	Statistical significance
Placebo (30 pts)	LDL-c=189.15±3.90	LDL=186.75±2.08	2.40	> 0.05
	HDL-c=36.11±2.11	HDL=37.17±1.51	1.06	> 0.05
Kalonji (27 pts)	LDL-c=202.45±1.54	LDL=189.52±2.21	12.93	< 0.001
	HDL-c=38.81±3.90	HDL=42.19±3.32	3.38	< 0.01
Vit B3 (28 pts)	LDL-c=212.65±2.32	LDL=185.61±3.43	27.04	< 0.001
	HDL-c=39.19±2.01	HDL=43.00±3.07	3.49	< 0.01

Table 1: HDL and LDL are measured in mg/dl, n stands for sample size, p-value >0.05 indicate non-significant, <0.01 indicate significant and <0.001 indicate highly significant change in basic values

Discussion

Treatment with three weeks, Kalonji decreased LDL-cholesterol 12.93 mg/dl by six weeks of treatment. HDL-cholesterol increased 3.38 mg/dl by taking this drug for six weeks. The change in both parameters were significant. In placebo group, LDL-C reduction was 2.40 mg/dl and increase in HDL-C was 1.06 mg/dl with P-value >0.05, which proves non-significant change in results. These results match with Akhondian et al [18] who did prove that Nigella sativa is very effective hypolipidemic drug. He tested the drug on 120 hyperlipidemic and diabetic patients by using Nigella sativa for one month. Their results were highly significant when compared with placebo-controlled group. Our results also match with results of Gillani AH et al [19] who proved LDL-Cholesterol reduction from 201.61±3.11 mg/dl to 187.16±2.10 mg/dl in fourty hyperlipidemic patients. Their HDL-C increase was 3.98 mg/dl which also matches with our results. Results of our study are in contrast with results of research work conducted by AH BH and Blunden G [20]. They explained that some active ingredients of Nigella sativa are hypolipidemic but their hypolipidemic effects are very narrow spectrum. Their results showd only 2.11 mg/dl change in LDL-C and 0.92 mg/dl increase in HDL-C of 38 rats. Difference in results may be genetic variants of human and rats. Brown BG et al [21] also described phenomenon of genetic variation in pharmacological effects of Nigella sativa. Burits M & Bucar F [22] have also mentioned wide variety effects of Nigella sativa with different genetic make ups. Our results also match with results of research work of Dehkordi FR & Kamkhah AF [23] and El-Dakhakhany M [24]. Same mechanism of action of drug Nigella sativa is described by El-Din K

et al [25]. In our research Niacin reduced LDL-Cholesterol from 212.65±1.19 mg/dl to 185.61±1.65 mg/dl in six weeks. This reduction in LDL-C was 27.04 mg/dl, which is highly significant change, when analyzed statistically. These results match with resultsn of research work conducted by Afilalo J et al [26] who proved almost same change in LDL-C in 32 hyperlipidemic patients who were cases of secondary hyperlipidemia and used Niacin 2 grams daily for two months. Their LDL-C reduction was 25.55 mg/dl. Their HDL-C increase was 6.65 mg/dl in 2 months. In our results HDL-C increase was 3.81 mg/dl in six weeks use of Niacin. Our results also match with results of research conducted by Whitney EJ et al [27] who proved 27.77 mg/dl reduction in LDL-C in 19 hyperlipidemic patients. Ginsberg HN et al [28] also support our results, as they proved 4.00 mg/dl increase in HDL-C when two grams of Niacin was used in 34 hyperlipidemic patients for six weeks. Our results do not match with results of research conducted by Boden WE et al [29] who proved that 2.5 grams Niacin decreased 10.99 mg/dl LDL-Cholesterol. HDL-C increase was only 1.11 mg/dl. These differences may be considered due to lack of physical exercise and no restriction of use of lipids in their diet. Cholesteryl ester transfer protein (CETP) facilitates the exchange of cholesterol ester in LDL and HDL particles for triglyceride in VLDL particles. The transfer of triglyceride into LDL and HDL particles makes them triglyceride-rich and hence a better substrate for hepatic lipase. Elevated hepatic lipase activity leads to a predominance of small, dense LDL particles and a reduction in HDL2, the more antiatherogenic subspecies of HDL [30].

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