

## EFFECT OF SOME ANTIHYPERTENSIVE DRUGS AND MEDICINAL PLANTS ON LIPID PROFILE IN PATIENTS WITH ESSENTIAL HYPERTENSION

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### ABSTRACT

**Background:** Hypertension is the most common disease and it is probably the most important public health problem in developed countries. **Objective:** To evaluate the effects of medicinal plants garlic (G), *Nigella sativa* (NS) and *Hibiscus sabdariffa* (HS) to improve the serum lipid levels in patients with essential hypertension (EHT). **Methods:** One hundred-ten patients were complaining from mild, moderate, severe EHT and treated by various regimens of antihypertensive drugs and medicinal plants (G, NS, HS). Serum lipid levels were measured before and after treatment. **Results:** Administration of G in combination with antihypertensive drugs caused a significant reduction in some serum lipid levels, while using NS, HS alone or in combination caused no significant improvement in serum lipid profile of most hypertensive patients groups. **Conclusion:** A combination of antihypertensive drugs and G caused improvement in some serum lipid levels in most groups, but using NS, HS in combination or alone caused no improvement in serum lipid levels.

**Keywords:** Garlic, *Nigella sativa*, *Hibiscus sabdariffa*, lipid profile.

### INTRODUCTION

Essential hypertension (EHT) is the most common type of hypertension. The consequences of the actual level of blood pressure in person will depend not only on the measured level but also upon certain risk factors such as aging, (23) , race, smoking, coffee drinking (11) , toxic metals, alcohol intake, obesity (17),(24) heredity (6) , glucose tolerance, cholesterol. Atherosclerosis is an important risk factor for the development of hypertension, so there was a linear relationship between coronary heart disease and serum cholesterol level that dependent on LDL-cholesterol, major atherogenic lipoprotein (14).

The efficacy of treatment hypertensive patients was tested in three well-known medicinal plants namely, G, NS, HS and their effects on the serum lipid levels.

### METHODS

This prospective study carried out in Baghdad / the Al-Kadimiya Teaching Hospital. (110)

patients were involved in the study with the range of age between (50.3-52.4) years for (72) females and (38) males, with mean body weight (80) kilograms and complaining from moderate to severe EHT before therapy. The patients were alienated in thirteen groups treated by antihypertensive drugs (ramipril, amlodipine, valsartan and metoprolol). Table (1) which were use alone or in combination, duration of treatment are (4) weeks. Three medicinal plants (G, NS and HS) were used daily in a dose of 5g of fresh G, as a tablets orally, 1g of NS as capsules orally, 15g of HS as infusion orally before breakfast.

These plants were used plus the above drugs related to each group which was used for another (4) weeks of treatment. The patients related to groups (11,12,13) have (5 patients) for each group suffered from mild to moderate EHT and treated by one medicinal plant which was used alone for duration of (4) weeks. To determine the serum lipid profile (S. cholesterol (CH), S. triglycerides (TG), S. HDL, S. LDL, S.VLDL) blood samples were taken

from all the patients who have no prior treatment after 12-14 hours fasting period and

then every (2) weeks after onset treatment for (4,8) weeks.

**Table 1: Showed the groups of hypertensive patients which were treated orally by different regimens of drugs**

Group No.	No. of patients + different regimens of drugs
Group one	(12 patients) were treated by combination of ramipril 5mg+ amlodipine 2.5 mg.
Group two	(11 patients) were treated by amlodipine 5 mg.
Group three	(12 patients) were treated by amlodipine 10 mg.
Group four	(11 patients) were treated by valsartan 160 mg.
Group five	(6 patients) were treated by ramipril 10 mg.
Group six	(12 patients) were treated by valsartan 80 mg.
Group seven	(12 patients) were treated by combination of amlodipine 5 mg + valsartan 160 mg + metoprolol 50 mg.
Group eight	(6 patients) were treated by combination of valsartan 80 mg + amlodipine 10 mg + metoprolol 50 mg.
Group nine	(6 patients) were treated by combination of amlodipine 5 mg + metoprolol 50 mg.
Group ten	(7 patients) were treated by combination of amlodipine 10 mg + metoprolol 50 mg.

### Statistical analysis

The Complete Randomized Design (CRD) A NOVA with Statistical Analysis System (SAS) was used to study the effect of period before treatment, 1, 2, 3, 4, weeks on different traits. LSD test and Duncan's multiple range was used to comparative significant differences between the means (20).

### RESULTS

Table (2) demonstrated that after treatment with different regimens of drugs there was a significant reduction in serum CH levels in groups (3, 4, 6, 7) while there was a significant elevation in serum CH levels in groups (1,5), serum TG, VLDL levels were significantly decrease in groups (4, 6) at the same time a

significant elevation of serum TG and VLDL levels in groups (3, 7, 10), also a significant increase in serum HDL levels as in groups (2,7) and a significant decrease in serum HDL levels in groups (4,6) regarding the serum LDL levels were significantly decrease as in groups (3,6,7) and significantly increase in groups (1,4,5)

A combination of antihypertensive drugs and G as in groups (1, 2, 3, 5, 7, 9,) provided a significant improvements in some serum lipid profile levels Table (3), while after using NS, HS alone or in combination with different drugs, there was no significant improvement in most serum lipid profile of groups (1, 2, 3, 4, 6, 12) and groups (4, 6, 7, 10, 13). Tables (4, 5) respectively.

**Table 2: The means of serum Lipid Cholesterol ( CH ), Triglyceride ( TG ), High Density Lipoprotein ( HDL ), Low Density Lipoprotein ( LDL ), Very Low Density Lipoprotein (VLDL) mmol/L in patients with EHT throughout the four weeks of treatment by different regimens of drugs.**

Group No.		Before treatment	After two weeks of treatment	After four weeks of treatment	LSD
Group one	S.CH	B 6.03 ± 0.48	A 6.51 ± 0.24	A 6.45 ± 0.24	**0.28
	S.TG	A 3.03 ± 0.59	A 2.97 ± 0.59	A 2.97 ± 0.58	0.48
	S.HDL	A 1.30 ± 0.12	B 1.19 ± 0.12	A 1.38 ± 0.11	**0.09
	S.LDL	B 3.37 ± 0.45	A 3.97 ± 0.28	A 3.72 ± 0.27	**0.29
	S.VLDL	A 1.37 ± 0.27	A 1.35 ± 0.27	A 1.35 ± 0.26	0.22
Group two	S.CH	A 4.69 ± 0.59	A 5.13 ± 0.68	A 4.67 ± 0.71	0.58
	S.TG	A 1.56 ± 0.32	A 1.41 ± 0.35	A 1.28 ± 0.34	0.30
	S.HDL	B 1.23 ± 0.16	BA 1.34 ± 0.13	A 1.37 ± 0.11	*0.12
	S.LDL	A 2.76 ± 0.70	A 3.15 ± 0.73	A 2.72 ± 0.76	0.64
	S.VLDL	A 0.70 ± 0.15	A 0.64 ± 0.16	A 0.58 ± 0.16	0.13
Group three	S.CH	A 6.25 ± 0.88	BA 6.00 ± 0.68	B 5.43 ± 0.71	*0.63
	S.TG	B 1.05 ± 0.08	B 1.22 ± 0.22	A 1.50 ± 0.29	**0.18
	S.HDL	A 1.06 ± 0.21	A 1.00 ± 0.17	A 1.09 ± 0.16	0.15
	S.LDL	A 4.72 ± 0.82	A 4.44 ± 0.63	B 3.66 ± 0.76	**0.61
	S.VLDL	B 0.48 ± 0.04	B 0.55 ± 0.10	A 0.68 ± 0.13	**0.08
Group four	S.CH	A 5.39 ± 0.38	BA 5.24 ± 0.24	B 5.01 ± 0.23	*0.26
	S.TG	A 3.44 ± 0.55	B 2.74 ± 0.88	B 2.19 ± 0.44	**0.57
	S.HDL	A 2.42 ± 0.52	BA 2.30 ± 0.51	B 1.98 ± 0.39	*0.42
	S.LDL	B 1.41 ± 0.51	BA 1.70 ± 0.30	A 2.04 ± 0.43	**0.37
	S.VLDL	A 1.56 ± 0.25	B 1.24 ± 0.40	B 0.99 ± 0.20	**0.26
Group five	S.CH	B 5.37 ± 0.21	A 6.03 ± 0.64	A 6.53 ± 0.53	**0.61
	S.TG	A 2.73 ± 0.51	A 2.25 ± 0.50	A 2.41 ± 0.48	0.62
	S.HDL	A 1.31 ± 0.39	A 1.39 ± 0.41	A 1.38 ± 0.15	0.42
	S.LDL	B 2.83 ± 0.71	A 3.62 ± 0.39	A 4.06 ± 0.50	**0.68
	S.VLDL	A 1.24 ± 0.24	A 1.02 ± 0.23	A 1.09 ± 0.22	0.28
Group six	S.CH	A 6.02 ± 0.02	B 5.88 ± 0.09	C 5.01 ± 0.01	**0.04
	S.TG	A 1.09 ± 0.01	A 1.07 ± 0.01	B 0.99 ± 0.12	**0.06
	S.HDL	A 1.59 ± 0.03	B 1.54 ± 0.03	C 1.48 ± 0.07	**0.04
	S.LDL	A 3.93 ± 0.03	B 3.86 ± 0.05	C 3.07 ± 0.02	**0.03
	S.VLDL	A 0.49 ± 0.02	A 0.49 ± 0.02	B 0.45 ± 0.02	**0.01
Group seven	S.CH	A 5.45 ± 0.04	B 4.95 ± 0.04	B 4.91 ± 0.09	**0.05
	S.TG	C 1.16 ± 0.02	B 1.75 ± 0.03	A 1.84 ± 0.03	**0.02
	S.HDL	C 0.90 ± 0.03	B 0.98 ± 0.02	A 1.64 ± 0.03	**0.02
	S.LDL	A 4.03 ± 0.03	B 3.18 ± 0.15	C 2.43 ± 0.03	**0.08
	S.VLDL	C 0.52 ± 0.03	B 0.79 ± 0.03	A 0.83 ± 0.03	**0.02
Group eight	S.CH	A 4.45 ± 0.68	A 4.42 ± 0.63	A 4.28 ± 0.54	0.76
	S.TG	A 1.27 ± 0.28	A 1.23 ± 0.25	A 1.12 ± 0.22	0.31
	S.HDL	A 1.09 ± 0.19	A 1.11 ± 0.15	A 1.17 ± 0.51	0.40
	S.LDL	A 2.79 ± 0.79	A 2.76 ± 0.74	A 2.16 ± 0.73	0.93
	S.VLDL	A 0.57 ± 0.13	A 0.56 ± 0.12	A 0.51 ± 0.10	0.14
Group nine	S.CH	B 5.45 ± 0.44	A 6.27 ± 0.57	BA 5.65 ± 0.59	*0.66
	S.TG	A 1.70 ± 0.37	A 2.01 ± 0.40	A 1.72 ± 0.47	0.51
	S.HDL	A 1.09 ± 0.18	A 1.08 ± 0.14	A 1.13 ± 0.07	0.17
	S.LDL	B 3.59 ± 0.56	A 4.28 ± 0.47	BA 3.74 ± 0.47	*0.62
	S.VLDL	A 0.77 ± 0.17	A 0.91 ± 0.18	A 0.78 ± 0.21	0.23
Group Ten	S.CH	A 5.24 ± 0.62	A 5.18 ± 0.63	A 4.99 ± 0.63	0.77
	S.TG	B 0.99 ± 0.04	BA 1.02 ± 0.06	A 1.06 ± 0.06	*0.06
	S.HDL	A 1.56 ± 0.08	A 1.52 ± 0.07	A 1.49 ± 0.06	0.09
	S.LDL	A 3.23 ± 0.61	A 3.21 ± 0.62	A 3.03 ± 0.62	0.76
	S.VLDL	B 0.45 ± 0.02	BA 0.46 ± 0.02	A 0.48 ± 0.02	*0.03

**Table 3: The means of serum Lipid Cholesterol (CH), Triglyceride(TG), High Density Lipoprotein (HDL), Low Density Lipoprotein(LDL), Very Low Density Lipoprotein (VLDL)mmol/L in patients with EHT throughout the four weeks of treatment by different regimens of drugs in groups(1, 2, 3, 5, 7, 8,9)plus(G)or treatment by(G)alone in group(11).**

Group No.		Before treatment Plus (G)	After two weeks of treatment plus(G)	After four weeks of treatment plus(G)	LSD
Group one No.(6)	S.CH	A 6.45± 0.26	A 6.25± 0.25	B 5.74± 0.31	**0.34
	S.TG	A 3.15±0.58	A 2.93± 0.58	A 2.73± 0.54	0.70
	S.HDL	B 1.41± 0.14	A 1.63± 0.15	A 1.74±0.15	**0.18
	S.LDL	A 3.61± 0.20	B 3.28± 0.21	C 2.76± 0.27	**0.28
	S.VLDL	A 1.43± 0.26	A 1.32± 0.26	A 1.24±0.24	0.31
Group two No.(5)	S.CH	A 4.80± 0.59	A 4.64± 0.47	A 4.18± 0.50	0.72
	S.TG	A 1.30± 0.26	A 1.20± 0.22	B 0.90 ± 0.10	*0.28
	S.HDL	B 1.37± 0.16	A 1.59± 0.15	A 1.70±0.14	*0.21
	S.LDL	A 2.85±0.68	AB2.51±0.52	B 2.07±0.45	*0.77
	S.VLDL	A 0.59±0.12	A 0.54± 0.10	B 0.40 ±0.04	*0.13
Group three No.(6)	S.CH	A 5.32± 0.80	A 5.14± 0.73	A 4.67± 0.69	0.91
	S.TG	A 1.45± 0.34	B1.27± 0.20	C 1.07± 0.15	**0.18
	S.HDL	B 1.17± 0.19	BA 1.31± 0.12	A 1.51± 0.19	*0.21
	S.LDL	A 3.51±0.82	A3.23 ± 0.78	A 2.67± 0.79	0.98
	S.VLDL	A 0.66±0.02	BA 0.58± 0.10	B0.50± 0.10	*0.10
Group five No.(6)	S.CH	A 6.53± 0.53	A 6.21±0.54	A 5.88± 0.55	0.66
	S.TG	A 2.41± 0.48	A 2.22± 0.58	A 2.00± 0.59	0.68
	S.HDL	B 1.38±0.15	A 1.58± 0.15	A 1.77±0.19	**0.20
	S.LDL	A4.06±0.50	BA3.62± 0.55	B 3.20± 0.39	*0.60
	S.VLVL	A1.09±0.22	A1.01±0.26	A 0.91± 0.27	0.31
Group seven No.(6)	S.CH	A 5.15± 0.71	BA 4.63± 0.68	B 3.90± 0.51	*0.79
	S.TG	A 1.91±0.12	BA 1.69± 0.32	B 1.40± 0.27	*0.31
	S.HDL	B 1.63±0.17	BA 1.71 ± 0.16	A 1.85± 0.09	*0.18
	S.LDL	A 2.65±0.70	A 2.16± 0.56	B 1.42± 0.44	**0.71
	S.VLDL	A 0.87 ± 0.06	BA 0.76 ± 0.14	B 0.63 ± 0.13	*0.14
Group eight No.(6)	S.CH	A 4.28 ± 0.54	A 4.16 ± 0.55	A 4.00 ± 0.52	0.66
	S.TG	A 1.12 ± 0.22	A 1.04 ± 0.21	A 0.95 ± 0.15	0.24
	S.HDL	A 1.17 ± 0.51	A 1.29 ± 0.50	A 1.51± 0.49	0.62
	S.LDL	A 2.61 ± 0.73	A 2.40 ±0.73	A 2.06 ± 0.72	0.89
	S.VLDL	A 0.51 ± 0.10	A 0.47 ± 0.09	A 0.44 ± 0.07	0.11
Group nine No.(6)	S.CH	A 5.65 ± 0.59	A 5.28 ± 0.53	B 4.00 ± 0.51	**0.67
	S.TG	A 1.72 ± 0.47	BA 1.48 ± 0.49	B 1.03 ± 0.21	*0.50
	S.HDL	C 1.13 ± 0.07	B 1.24 ± 0.06	A 1.36 ± 0.06	**0.08
	S.LDL	A 3.74 ± 0.47	A 3.37 ± 0.36	B 2.18 ± 0.44	**0.52
	S.VLDL	A 0.78 ± 0.21	BA 0.67 ± 0.22	B 0.47 ± 0.10	*0.23
Group eleven No.(5)	S.CH	A 5.26 ± 0.46	A 5.00 ± 0.48	A 4.86 ± 0.5	0.66
	S.TG	A 1.65 ± 0.50	A 1.40 ± 0.38	A 1.26 ± 0.35	0.57
	S.HDL	B 1.05 ± 0.21	BA 1.28 ± 0.16	A 1.41 ± 0.15	*0.24
	S.LDL	A 3.47 ± 0.52	A 3.08 ± 0.41	A 2.88 ± 0.44	0.63
	S.VLDL	A 0.75 ± 0.23	A 0.63 ± 0.18	A 0.57 ± 0.16	0.26

**Table 4: The means of serum Lipid Cholesterol ( CH ), Triglyceride ( TG ), High Density Lipoprotein ( HDL ), Low Density Lipoprotein ( LDL ), Very Low Density Lipoprotein ( VLDL) mmol/L in patients with EHT throughout the four weeks of treatment by different regimens of drugs in groups ( 1, 2, 3, 4, 6) plus ( NS ) or treatment by ( NS ) alone in group ( 12 ).**

Group No.		Before treatment plus (NS)	After two weeks of treatment plus(NS)	After four weeks of treatment plus(NS)	LSD
Group one No.(6)	S.CH	A 6.46± 0.24	BA 6.28± 0.25	B 6.06± 0.26	*0.31
	S.TG	A 2.80± 0.57	A 2.69± 0.56	A 2.52± 0.54	0.69
	S.HDL	A 1.36± 0.07	A 1.39± 0.07	A 1.44± 0.09	0.09
	S.LDL	A 3.83± 0.31	A 3.66± 0.31	A 3.48± 0.30	0.38
	S.VLDL	A 1.27± 0.26	A 1.22± 0.25	A 1.14± 0.25	0.31
Group two No.(6)	S.CH	A 4.56± 0.84	A 4.38± 0.86	A 4.19± 0.86	1.05
	S.TG	A 1.26± 0.43	A 1.13± 0.41	A 0.99± 0.31	0.47
	S.HDL	B 1.37± 0.07	BA1.41± 0.06	A 1.48± 0.05	*0.08
	S.LDL	A 2.61± 0.87	A 2.46± 0.89	A 2.26± 0.88	1.08
	S.VLDL	A 0.57± 0.19	A 0.51± 0.18	A 0.45± 0.14	0.22
Group three No.(6)	S.CH	A 5.53± 0.65	A 5.36± 0.58	A 5.04± 0.57	0.74
	S.TG	A 1.54± 0.43	A 1.40± 0.42	A 1.29± 0.40	0.51
	S.HDL	A 1.03± 0.11	A 1.07± 0.07	A 1.09± 0.08	0.11
	S.LDL	A 3.80± 0.76	A 3.65± 0.74	A 3.37± 0.72	0.91
	S.VLDL	A 0.70± 0.20	A 0.63± 0.19	A 0.58± 0.18	0.23
Group four No.(5)	S.CH	A 4.92± 0.29	A 4.78± 0.28	A 4.60± 0.27	0.39
	S.TG	A 2.26± 0.13	A 2.23± 0.28	A 2.13± 0.27	0.32
	S.HDL	A 1.88± 0.36	A 1.91± 0.52	A 1.94± 0.54	0.66
	S.LDL	A 2.02± 0.10	BA 1.86± 0.19	B 1.70± 0.23	*0.25
	S.VLDL	A 1.02± 0.06	A 1.01± 0.13	A 0.96± 0.12	0.15
Group six No.(6)	S.CH	A 5.38± 0.50	A 5.18± 0.84	A 4.78± 0.76	0.88
	S.TG	A 1.08± 0.11	A 1.05± 0.12	A 1.00± 0.10	0.13
	S.HDL	A 1.35± 0.25	A 1.39± 0.23	A 1.43± 0.19	0.28
	S.LDL	A 3.54± 0.44	A 3.32± 0.62	A 2.90± 0.59	0.68
	S.VLDL	A 0.49± 0.05	A 0.47± 0.05	A 0.45± 0.04	0.06
Group twelve No.(5)	S.CH	A 5.46± 0.29	A 5.36± 0.28	A 5.20± 0.29	0.39
	S.TG	A 1.70± 0.63	A 1.57± 0.60	A 1.30± 0.38	0.75
	S.HDL	A 1.46± 0.27	A 1.50± 0.26	A 1.55± 0.09	0.31
	S.LDL	A 3.27± 0.23	A 3.15± 0.22	A 3.06± 0.28	0.34
	S.VLDL	A 0.77± 0.29	A 0.71± 0.27	A 0.59± 0.20	0.34

**Table 5: The means of serum Lipid Cholesterol ( CH ), Triglyceride ( TG ), High Density Lipoprotein ( HDL ), Low Density Lipoprotein ( LDL ), Very Low Density Lipoprotein ( VLDL ) mmol/L in patients with EHT throughout the four weeks of treatment by different regimens of drugs in groups ( 4, 6, 7, 10 ) plus ( HS ) or treatment by ( HS ) alone in group ( 13 ).**

Group No.		Before treatment plus (HS)	After two weeks of treatment plus(HS)	After four weeks of treatment plus(HS)	LSD
Group four No.(6)	S.CH	A 5.08 ± 0.13	A 4.98 ± 0.12	B 4.80 ± 0.16	*0.17
	S.TG	A 2.13 ± 0.60	A 2.35 ± 0.65	A 2.60 ± 0.49	0.72
	S.HDL	A 2.06 ± 0.42	A 2.10 ± 0.43	A 2.17 ± 0.37	0.50
	S.LDL	A 2.06 ± 0.59	A 1.88 ± 0.73	A 1.45 ± 0.47	0.75
	S.VLDL	A 0.97 ± 0.27	A 1.07 ± 0.29	A 1.18 ± 0.22	0.32
Group six No.(6)	S.CH	A 4.65 ± 0.38	A 4.57 ± 0.38	A 4.31 ± 0.36	0.46
	S.TG	B 0.91 ± 0.03	A 1.29 ± 0.18	A 1.34 ± 0.15	**0.17
	S.HDL	A 1.63 ± 0.13	A 1.69 ± 0.17	A 1.75 ± 0.16	0.19
	S.LDL	A 2.61 ± 0.29	BA 2.30 ± 0.32	B 1.95 ± 0.33	**0.39
	S.VLDL	B 0.41 ± 0.02	A 0.59 ± 0.08	A 0.61 ± 0.07	0.08
Group seven No.(6)	S.CH	A 4.54 ± 0.46	A 4.45 ± 0.45	A 4.22 ± 0.15	0.47
	S.TG	B 1.78 ± 0.12	A 2.20 ± 0.15	A 2.35 ± 0.14	**0.17
	S.HDL	B 1.65 ± 0.08	B 1.70 ± 0.07	A 1.86 ± 0.09	*0.10
	S.LDL	A 2.22 ± 0.46	BA 1.75 ± 0.45	B 1.29 ± 0.15	**0.48
	S.VLDL	B 0.81 ± 0.05	A 0.99 ± 0.07	A 1.07 ± 0.06	**0.08
Group ten No.(7)	S.CH	A 4.99 ± 0.63	A 4.88 ± 0.63	A 4.74 ± 0.63	0.77
	S.TG	C 1.06 ± 0.06	B 1.38 ± 0.07	A 1.57 ± 0.10	**0.10
	S.HDL	B 1.49 ± 0.06	BA 1.55 ± 0.08	A 1.62 ± 0.04	**0.08
	S.LDL	A 3.03 ± 0.62	A 2.70 ± 0.64	A 2.41 ± 0.65	0.78
	S.VLDL	C 0.48 ± 0.02	B 0.63 ± 0.03	A 0.71 ± 0.04	**0.04
Group thirteen No.(5)	S.CH	A 4.55 ± 0.18	A 4.45 ± 1.20	A 4.30 ± 0.21	0.27
	S.TG	A 1.48 ± 0.49	A 1.80 ± 0.57	A 2.00 ± 0.59	0.76
	S.HDL	A 1.04 ± 0.11	A 1.09 ± 0.15	A 1.20 ± 0.15	0.20
	S.LDL	A 2.84 ± 0.26	BA 2.53 ± 0.41	B 2.19 ± 0.49	*0.55
	S.VLDL	A 0.67 ± 0.22	A 0.82 ± 0.26	A 0.91 ± 0.27	0.34

## DISCUSSION

Table (2) and after treatment with amlodipine group (2) caused a significant elevation in serum HDL, but other studies show no elevation in HDL because differ CCBs seem to have variable effect on lipid levels in hypertensive patients (1) while using amlodipine group (3) caused reduction in serum CH, LDL, levels which compatible with results of (16). Hypercholesterolemia linked with endothelin system and progression of atherosclerosis (3), after treatment with amlodipine there was a significant reduction in ET-1 (22) by up regulation of LDL receptors in the liver with enhanced LDL clearance. Using ramipril group (5) caused a significant elevation in serum CH, LDL, this may differ from results of (27) (12) that ramipril caused decrease in CH, LDL, HDL after 2-4 months and ramipril alone didn't significantly change the lipoprotein and C-reactive protein, so our results due to the short period of treatment. Treatment with valsartan groups (6, 4) produced a significant reduction in serum CH, TG, HDL, LDL, VLDL levels, this were compatible with (8) also reducing in CH and LDL levels, occurred by reducing the expression of inflammatory chemokines and

oxidative stress markers (26), while valsartan group (4) caused a significant elevation in LDL level that compatible to the result of (19) so valsartan alone has no lowering effect on the high sensitivity C-protein that promotes oxidized LDL uptake (21). Combination of ramipril and amlodipine group (1) produced increase in serum CH, LDL levels this attributed to small doses of drugs and short period of treatment. Using a combination of amlodipine, valsartan and metoprolol group (7) produced a significant reduction in serum CH, LDL and elevation in HDL, this may be related to the effect of CCBs and Angls. At the same time there was a significant elevation in serum TG, VLDL that may results from the action of metoprolol (10). On other hand a combination of amlodipine and metoprolol caused a significant elevation in serum TG and VLDL levels group (10) but was still with normal range. Using garlic groups (8, 11) produced non significant changes in serum lipid profile, this may related to normal levels of lipid before treatment with exception of level of LDL, so (28) reported that G is not clinically relevant lipid-lowering in normo-lipidaemic individuals but there was a significant increase in serum

HDL level group (11) while using G in combination with different drugs provided a significant improvement in some serum lipid profile levels groups (1, 2, 3, 5, 7, 9). These effects related to the synergistic action between G and different antihypertensive drugs. Garlic has a sulfur containing compound, the thiosulfonates including allicin which is the active substance (25) so garlic and its constituents inhibit human squalene monooxygenase and HMG – CoA reductase enzyme involved in cholesterol biosynthesis in hepatocytes (18). Garlic produced a lowering effect in TG and CH synthesis (29) so increase Peroxisome proliferators activated receptor, Hepatocyte Nuclear Factor and decrease in cholesterol 7  $\alpha$ -hydroxylase gene transcription, this attenuated the trans activation of CYP7A1 enzyme that regulate the cholesterol conversion to bile acids in the liver, contribute to decrease the TG and CH synthesis which found in HepG2cell. Garlic is this study resulted in a significant increase in serum HDL levels, so G appears to be an important protective factor against heart disease and stroke via its ability to impact the process of atherosclerosis at many steps.

Using G in human lead to increase resistance of LDL oxidation that suppress of LDL oxidation, this is one of the powerful mechanisms for anti atherosclerotic properties of G (13).

After using NS alone or in combination with drugs there was no significant improvement in most serum lipid profile groups (1, 2, 3, 4, 6, 12). These may be related to small dose of NS or short duration of therapy to provide changes in serum lipid levels that differ from the experimental studies of (15) , (2) and (5) demonstrated that NS produced a significant lowering effect in lipid levels and elevation in HDL after treatment by dose 800mg/day, orally for 4 weeks and 30mg/kg BW for 12-20 weeks. Treatment by HS produced a significant reduction in serum LDL group (13) reduction in serum CH group (4), elevation in TG, VLDL, HDL and reduction in LDL levels groups (6, 7, 10) these changes may be related to the effects of drugs involved in the combination, or to the diet of patients, this mean that HS in the present study has no improved effect on serum lipid levels as a result of inadequate dose or short period of treatment, while previous studies demonstrated that HS provide it's effect on serum lipid levels after 10 and 8 weeks of treatment (4) , (7) respectively, also the method of preparing the aqueous extract of HS play an important role to extend effect of plant on the serum lipids (9).

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