Research Article

Effect of Raspberry Ketones and L-carnitine on Oxidative Stress and Body Weight in Iraqi Obese Patients

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ABSTRACT

This study aimed to evaluate the effect of two natural products; raspberry ketones (RK) and L-carnitine on oxidative stress parameter and body weight in Iraqi obese patients. Sixty obese women aged 20- 40 with a BMI ≥ 30 were randomly divided into three groups twenty patients in each; group one received raspberry ketones 500 mg capsule , the second received L-carnitine 1000 mg hard capsule and the third was control without treatment. All patients were informed about Standard diet advice (low calorie diet) the physical activity needed. The treatment course was 12 weeks. For each group, the body weight and body mass index (BMI) were measured at baseline and after 12 weeks; oxidative stress markers (MDA, GSH and 8-isoprostane f2α) were measured at the same manner. The percent of reduction in body weight was highest with L-carnitine group (7.38 %) and lowest with control group (3.79%), although, RK was (5.10%). The effects of the two drugs on changes in oxidative stress parameter were as follows: the RK was most effective in increasing glutathione activity (37.85%) comparing to L- carnitine (26.15 %) and control group (23.92 %). While the percent of MDA reduction parameter were sorted as RK (56.57 %) more than L-carnitine (43.31 %) and L-carnitine more than control (26.95 %) groups. However, 8-isop f2α was (8.71%) for L-carnitine,(4.32 %) for RK and (2.49%) for control group respectively. Raspberry ketones is the most potent anti oxidant herbal treatment among two groups and can reduce the oxidative stress in obese patients.

Keywords: L-carnitine, Oxidative stress, Raspberry Ketones.

INTRODUCTION

Obesity is a public health problem that has raised concern worldwide. According to the World Health Organization (WHO), there will be about 2.3 billion overweight people aged 15 years and above, and over 700 million obese people worldwide in 2015.[1] According to STEPS study(2006), two thirds (66.9%) of the Iraqi population aged (25- 65) were found to be overweight or obese. The rate of overweight among female was higher than male (69.6 Vs 63.6), nearly one third of the respondents were obese. Obesity was proportionately higher than overweight among female, whereas overweight supervened among male.[2] The WHO has classified overweight and obesity in adults based on various BMI cutoffs.[3] These cutoffs are set based on co-morbidities risk associated with BMI. However, the use of BMI does not distinguish between weight associated with muscle and weight associated with fat, and the relationship between BMI and body fat content varies according to body build and proportion.[4] In contrast, the measure of intra abdominal or central fat accumulation to reflect changes in risk factors for cardiovascular diseases and other forms of chronic diseases is better than BMI.[5] Therefore, an assessment of central fat accumulation greatly assists in defining obesity. Obesity is associated with increased oxidative stress and low-grade chronic inflammation.[6] Both events contribute to metabolic abnormalities occurring in the obesity-associated metabolic syndrome[7] and play a critical role in the pathogenesis of various diseases such as atherosclerosis, cardiovascular disease, diabetes type 2 and cancer.[8] Recent research has shown that weight loss attenuates inflammation and leads to improvement in adipokine profiles.[9] Although associations of overweight and obesity with increased oxidative stress have been reported, the effects of weight loss on oxidative stress markers are rarely described in literature.[10] In adults, BMI, total body fat, and waist circumference have been shown to be positively correlated with urinary F2-isoprostane levels and inversely correlated with paraoxonase (PON1) activity.[11] The use of allopathic and pharmacological drugs has become a popular means to overcome excess weight gain.[12] While these drugs generally are effective, severe adverse toxicities may limit their overall usefulness.[13] A nutritional based intervention is being hailed as an inexpensive alternative to aid weight loss, and weight management.[14] Medicinal herbal supplements are being extensively utilized due to their effectiveness in managing many chronic disorders. They are cost effective, and exert less to no toxic side effects in comparison with many chemically synthesized drugs.[15] Accordingly, preliminary reports suggested that herbs with a long history of use and other natural substances less likely to produce severe toxicity might be effective in reducing appetite and promoting significant weight loss are encouraging.[16] L- carnitine the name carnitine originates from the Latin word for flesh or meat, the mechanism of action of carnitine in the body is facilitation lipid oxidation by transporting long-chain fatty acids into the inner mitochondria region where they undergo β-oxidation.[17] Raspberry ketones also known as [4-(4-hydroxyphenyl) butan-2-one] is a compound extracted from red

raspberries that is usually used as a scenting and flavoring agent in foods and cosmetics.[18] The structure of Raspberry ketone has a vaguely similar structure to Ephedrine and Synephrine, where the butanone-substituted phenyl group of raspberry ketone replaces ethylamine group of ephedrine or synephrine. There is also some structural similarity to Capsaicin, with para-substituted phenolic and ketone.[19]

Patient and Methods

In this Prospective randomized single blind clinical study, sixty (60) obese women aged 20- 40 with a BMI ≥ 30 who referred to (Obesity Research & Therapeutic Center in Al – Kindy Medical College). The study protocol was approved by Research Ethical Committee in Al Nahrain University \ College of medicine. All patients had informed consent and statement of confidentiality. Exclusion criteria: patients had hypertension, diabetes, smoking, ischemic heart disease, or any active inflammatory disease, renal disease and chronic liver disease; female on contraceptive pills, pregnant or lactating, or had thyroid or hormonal disturbance and patients on any other drug or therapy that affect oxidative stress like (vit. E, vit C). the patients were randomly divided to three groups twenty patients in each; group one received raspberry ketones 500 mg capsule once daily (Raspberry Ketones 500mg pure ketones 500® from VITATRIX LLC USA), the second received L-carnitine 1000 mg hard capsule once daily (L-Carnitine 1000 mg from ULTIMATE NUTRITION USA) and the third was control without treatment group. All patients were informed about Standard diet advice (low calorie diet) and they must increase their physical activity. The treatment course is 12 weeks. In day zero (D0) all patients underwent an initial assessment that included a medical history, physical examination and vital signs. In addition anthropometric parameters (weight, height, BMI and waist circumference) were taken. Visit 1 , blood aspirated for oxidative stress marker [measurement of serum malondialdehyde (MDA), serum 8- isopro f2α (we use 8-isoprostane ELISA Kit from Abcam USA), serum glutathione (GSH) levels]. After one month patients return to the obesity center to evaluate the tolerability, all adverse events were recorded and then in the last visit (after 12 weeks from the beginning) clinical examination with all investigations were done. Statistical analysis: The results were expressed as mean ±SD. Student t-test for paired and unpaired sample and ANOVA test was used to examine the degree of significance, P-value less than 0.05 considered significant.

RESULTS

All patients were matched in age, weight, waist circumference and BMI before treatment table (1), that revealed no significant differences in these parameter between all groups and control patients. A total of four patients had been quitted during this study, due to poor compliance, side effect and they not attended (the data of these patients have been excluded from the data at the base line).

In control group, the mean and standard deviation (±SD) for baseline clinical parameter of weight, Waist circumference and BMI were 88.061 ± 9.383, 97.166 ±6.767 and 34.833 ±2.991 respectively. And the results after three months intervention for all the parameter mentioned above were 84.844 ± 8.943, 94.722 ± 6.488 and 33.622 ±2.756 respectively, all parameters show high statistical difference between pre and post intervention table (2); the percent of reduction were (3.79%), (2.57%) and (3.58%) respectively.

Table 1: Comparison of pretreatment clinical parameter for all groups the present study

Groups	Age (year)	Weight (Kg)	WC (cm)	BMI Kg/m^2
Control (no=18)	32.72 ±7.002	88.061 ± 9.383	97.166 ± 6.767	34.833 ± 2.991
Lcarnitine (no=18)	33.11 ± 6.533	86.68 ± 6.930	96.444 ±5.782	34.588 ± 2.774
Raspberry ketones (no=20)	31.75 ±5.580	89.46 ± 9.014	100.00 ± 7.920	35.415 ± 3.341
Control				
L-carnitine	NS	NS	NS	NS
Raspberry ketones	NS	NS	NS	NS
L-carnitine compared with				
Raspberry ketones	NS	NS	NS	NS

WC: Waist circumferences; BMI: Body Mass Index; NS = Non significant (p> 0.05).

Table 2: The mean, ±SD of clinical parameter for control group pre and after 3 months and the percent of reduction

Clinical parameter	Control (n=18) mean ± SD	Control after 3m (n=18) mean ± SD	Percent of reduction %
Age (year)	33.111±6.5333	--------	------
Ht. (cm)	158.33 ±2.950	--------	------
Weight (kg)	88.061 ± 9.383	84.844 ± 8.943 **	3.79
WC (cm)	97.166 ±6.767	94.722 ± 6.488 **	2.57
BMI	34.833 ±2.991	33.622 ±2.756 **	3.58

WC: Waist circumferences; Ht: Height in centimeter; BMI: Body Mass Index; ** = Highly Significant difference from control,(P<0.001).

Table 3: The mean, ±SD and the percent of reduction% of clinical parameter for L-Carnitine pre and after 3 months of treatment

Clinical parameter	L- Carnitine (n=18) mean ± SD	L- Carnitine after 3months. (n=18) mean ± SD	Percent % of reduction
Age (year)	33.111±6.5333	------	------
Ht. (cm)	158.33 ±2.950	------	------
Weight (kg)	86.68± 6.930	80.74± 6.440**	7.38
WC (cm)	96.444±5.782	89.611 ± 6.545**	7.73
BMI	34.588±2.774	32.205 ±2.503**	7.41

WC: Waist circumferences; Ht: Height; ** = Highly Significant difference (P<0.001).

Table 4: The mean, ± SD and percent of reduction in clinical parameter of all groups of the study (control, L-carnitin and raspberry ketones) after (12) weeks intervention

Clinical parameter	Control	L-carnitine	(RK)	% of reduction control	% of reduction L-carnitine	%of reduction (RK)
Weight (kg)	84.844 ± 8.943	80.74 ± 6.440	85.2 ± 9.258 b	3.79 %	7.38 % a b	5.10 %
WC (cm)	94.722 ± 6.488	89.611 ± 6.545	95.9 ± 7.992 b	2.57 %	7.73 % a b	4.31 % a
BMI	33.622 ± 2.756	32.205 ± 2.503	33.7 ± 3.371 b	3.58 %	7.41 % a b	5.08 % ā

WC: Waist circumferences; BMI: Body mass index; RK: Raspberry ketones; a = highly significant difference from control group ($p < 0.001$); ā = significant difference from control group ($p < 0.05$); b = significant difference of L-carnitine from RK ($p < 0.05$).

Table 5: The mean, ±SD and the percent of reduction of clinical parameter for RK pre and after 12 weeks intervention

Clinical parameter	RK (n=20) mean ± SD	RK after 3m (n=20) mean ± SD	Percent of reduction %
Age (year)	31.75 ±5.580	------	------
Ht. (cm)	158.925 ±4.091	------	------
Weight (kg)	89.46± 9.014	85.2± 9.258**	5.10
WC (cm)	100.00 ±7.920	95.9± 7.992**	4.31
BMI	35.415 ±3.341	33.723± 3.371**	5.08

RK: Raspberry keton; WC: Waist circumferences; Ht: Height; BMI: Body Mass Index; M=Months; ** = Highly Significant difference (P<0.001).

Table 6: The mean, ± SD of oxidative stress (OS) parameter for control group pre and after 3 months and the percent of changes (reduction or increment)

OS parameter	Control (n=18) Mean ± SD	Control after 3m. (n=18) mean ± SD	Percent of changes %
S. GSH (µmol/l)	0.445 ± 0.0492	0.5483 ± 0.04409 **	23.92 % ↑
S. MDA (µmol/l)	2.093 ±0.5229	1.6622 ± 0.40273**	26.95 % ↓
S. 8- isop. pg/mL	184.22 ±10.53	179.72 ± 9.85 **	2.49 % ↓

** = Highly Significant difference (P<0.001); M = Month; GSH = glutathione; MDA: Malandaldihyd; 8-isop = 8- isoprostane; m=month; OS= oxidative stress.

Accordingly, in L-carnitine group, the mean, ± SD for baseline parameter of weight, Waist circumference and BMI, were 86.68± 6.930, 96.444 ±5.782 and 34.588±2.774 respectively. All the parameters were reduced after the intervention (after 3 months treatment) and the results were 80.74± 6.440, 89.611 ± 6.545 and 32.205 ±2.503respectively. It has been noted after statistical analysis that all results differ in highly significant manner ($p<0.001$) from the baseline. The percent of reduction were 7.38%, 7.73% and 7.41 table 3.

Moreover the L-carnitine group had high significant difference ($p<0.001$) from control group in percent of reduction for all clinical parameter table 4.

Table 5 showed the effect of raspberry ketones in obese women after three months intervention. The mean and ±SD of baseline clinical parameter for this group were 89.46± 9.014, 100.00 ±7.920 and 35.415 ±3.341 respectively for these parameters: weight, Waist circumference and BMI respectively. While the results after intervention were 85.2± 9.258, 95.9± 7.992 and 33.723± 3.371 respectively. All clinical parameter after treatment were differ with high significance ($p<0.001$) from baseline, and the percent of reduction were as follows: weight (5.10%), WC (4.31%) and BMI (5.08%) respectively.

On the other hand table (4) revealed the difference between control and Raspberry ketons groups, there were statistically high significant difference ($p< 0.001$) in percent of changes in WC while the BMI differing only significantly ($p< 0.05$) although, the p value of weight was more than the 0.05 ($p>0.05$) and there was no significant difference. We compared the effect of RK and L- carnitin and the results of comparisons were significant difference favoring L-carnitine in all clinical parameter at the level of percent of changes ($p<0.05$) table (4).In control group, the mean and SD for baseline oxidative stress parameters were; glutathione (0.445 ±0.049), MDA (2.093 ±0.522) and 8-isoprostane (184.22 ±10.53). And the mean and ±SD after 3 months intervention were (0.548 ±0.044), (1.6622 ±0.402) and (179.72 ±9.85) respectively. It is worth noting that the glutathione increased by (23.29%)

from the baseline, however, the MDA, 8-isoprostane were reduced by (26.95%) and (2.49%) respectively. The statistical analysis revealed to there were a highly significant differences (p<0.001) between baseline and after 3 months intervention for all parameters table 6.

Accordingly, the effect of L-carnitine on oxidative stress parameter in baseline were as follwos: GSH (0.440 ±0.04820), MDA (2.2111 ±0.47016) and 8-isoprostane (185.11 ± 8.5466). Further, The mean, ±SD after intervention were (0.5526 ±0.04442), (1.649 ± 0.33874) and (170.33 ± 7.6157) respectively. However, the increment in glutathione was (26.15 %) while there were reduction in MDA and 8-isoprostane by (43.31 %) and (8.71 %) respectively. The statistical analysis revealed there were a highly significant differences (p<0.001) between baseline and after 3 months intervention for all parameters in the table except MDA which was differ only significantly (p<0.05) table 7.

Table 7: The mean, ±SD of oxidative stress (OS) and the percent of changes for L- Carnitine group pre and after 3 months treatment.

OS parameter	L- Carnitine (n=18) Mean ± SD	L- Carnitine after 3m (n=18) mean ± SD	Percent of change %
S. GSH (µmol/l)	0.440 ± 0.04820	0.5526 ± 0.04442**	26.15 % ↑
S. MDA (µmol/l)	2.2111 ± 0.47016	1.6494 ± 0.33874*	43.31 % ↓
S. 8-isop.pg/mL	185.11 ± 8.5466	170.33 ± 7.6157 **	8.71 % ↓

** = Highly significant difference from control (P<0.001); m = Month; GSH = glutathione; MDA = malondialdehyde; 8-isop = 8-isoprostane.

The differences between control and L- carnitine groups after 3months intervention presented in table (8) revealed high significant difference (p< 0.001) for 8-isoprostane. And there were no significant differences for others parameter.

Table 8: Mean, SD and the percent of changes in oxidative stress (OS) parameters for control, L-carnitine and raspberry ketones (RK) after (12) weeks treatment.

OS parameter	Control	L-carnitine	RK	% of change in C.	% of change in L-carnitine	% of change RK
S. GSH (µmol/l)	0.5483 ± 0.04409	0.552 ± 0.044	0.609 ± 0.047a e	23.92 % ↑	26.15 % ↑	37.85 %↑ a e
S. MDA (µmol/l)	1.6622 ± 0.4027	1.6494 ± 0.338	1.495 ± 0.419	26.95 % ↓	43.31 % ↓	56.57↓ ā
S. 8- isop. pg/mL	179.72 ± 9.85	170.33 ± 7.6157 ā bb	180.7 ± 13.07	2.49 % ↓	8.71 % ↓ a bb	4.32 % ↓

GSH = glutathione; MDA = Malondialdehyde; 8-isop = 8- isoprostane; ā = significant difference from control group (p < 0.05); a = highly significant difference from control group; bb = Highly significant difference (P<0.001) of L-carnitine from RK; e = significant difference (p < 0.05) of RK from L- carnitine.

Table 9: The mean, ±SD of oxidative stress (OS) parameter for Raspberry Ketones (RK) group pre and after 3 months treatment. and the percent of changes%.

OS parameter	RK (n=20) Mean ± SD	RK after 3m (n=20) mean ± SD	Percent change %
S. GSH (µmol/l)	0.4430±0.04601	0.6096±0.04735**	37.85 %↑
S. MDA (µmol/l)	2.2200 ±0.44674	1.4955 ±0.41913**	56.57 % ↓
S. 8-isop.pg/mL	188.3 ±11.318	180.7 ± 13.0791	4.32 % ↓

** = Highly Significant difference (P<0.001); m = month; GSH = glutathione; MDA = malondialdehyde; 8-isop= 8- isoprostane.

The effect of RK on oxidative stress parameter in baseline were as follows: GSH (0.4430±0.04601), MDA (2.2200 ±0.44674) and 8-isoprostane (188.3 ±11.318). Although, The mean and ±SD after intervention were (0.6096±0.04735), (1.4955 ±0.41913) and (180.7 ± 13.0791) respectively. However, the increment in glutathione was (37.85 %) while there were reduction in MDA, 8-isoprostane by (56.57 %) and (4.32%) respectively. The statistical analysis revealed a highly significant differences (p<0.001) between baseline and after 3 months intervention for all parameters, except 8-isoprostane there was no difference and the p value (p>0.05) table 9.

The difference between control and RK groups after intervention was highly significant (p<0.001) in glutathione, however, the significant difference in MDA was present (p<0.05) and there was no significant difference in 8- isoprostane parameter table (8). Continuously, the same table shows the differences between L- carnitin and RK, it was revealed to the increment in the percent of glutathione in RK above the level in L-carnitine by (11.7%) and the statistical analysis showed the significant difference (p<0.05) in this parameter, although there was highly significant difference in 8-isoprostane (p<0.001), it is worth noting that the reduction in this value in L-carnitine group was(4.39%) more than in RK group. The percent of changes (reduction or increment) of the clinical data was summarized in table (4). The percent of reduction in body weight was highest with L-carnitine group (7.38 %) and lowest with control group (3.79%), however, RK was (5.10%). When ranking the drugs according to their weight reduction they were as follows: L-carnitine reduced weight more than RK, and RK reduced weight more than control (placebo) group, and so on for other parameters. finally, the effect of the two drugs on the

percent of changes in oxidative stress parameter were summarized in table (8) which revealed that the RK was most effective in increasing glutathione activity (37.85%) comparing to L- carnitine (26.15 %) and control group (23.92 %). While concerning the percent of MDA reduction; parameter was sorted as RK (56.57 %) more than L-carnitine (43.31 %) and L-carnitine more than control (26.95 %) groups.

DISCUSSION

The present study demonstrated that 1 g/day orally, L-carnitine supplementation in obese women was able to reduce body weight by (7.38%), adipose tissue accumulation (WC) by (7.73%) and BMI by (7.41%), all these anthropometric parameter are reduced significantly in L- carnitine treatment group from baseline and from control group . These results were in agreement with Alipour results[20] and can be due to the positive effect of L-carnitine on β-oxidation of fatty acids and its activation of the pyruvate dehydrogenase complex by decreasing the intra mitochondrial acetyl-CoA/CoA ratio through the trapping of acetyl groups, the simultaneous reduction of acetyl-CoA levels in the cytosol further contributes to activate the glycolytic pathway, and so L-carnitine had a key role in glucose metabolism and assists in fuel-sensing.[21] However, our results disagree with Derosa[22] who suggested that L-carnitine did not give an improvement of body weight, glycemic and lipid profile compared to placebo. There is a paucity of data from clinical studies investigating the role of oxidative stress in obesity and the effects of weight loss on oxidative stress status in metabolically healthy and metabolically abnormal individuals.[23] The results of present study indicated that L-carnitine produced a significant inhibition of MDA production and a significant increase in GSH where Amin and Nagy found that L-carnitine produced a significant inhibition of MDA production and a significant increase in GSH and activity of catalase [16], however, L-carnitine reduces significantly the content of thiobarbituric acid reactive substances (TBARS), and causes marked increase in activity of catalase in skeletal muscles of obese rats.[24] It has been shown that many pathological conditions that resulted in elevation of MDA due to lipid peroxidation were prevented by L-carnitine.[25] Moreover L-carnitine favorably modulates oxidative stress causing a reduction in oxidized LDL cholesterol levels.[26] L-carnitine has the ability to increase the level of nonenzymatic antioxidants such as GSH or vitamins E and C.[27] It was reported that L-carnitine causes increases in vitamin E and C status by increasing the level of GSH.[28] The results of these studies were consistent with the current study. Moreover the level of 8- isoprostane were significantly reduced in L-carnitine group which agree with Ribas findings.[29] Further, Oh and his colleagues concluded that an improvement in hepatic steatosis, inflammatory and oxidative stress levels after regular exercise coupled to diet regimen in non alcoholic fatty liver disease patients[30], which consistent with our results that revealed a decrease in MDA level and increase in glutathione level in control group.

Little can be found about RK from a scientific perspective studies. In this study there was a significant reduction in anthropometric parameters (body weight, WC and BMI) in the RK group after 12 weeks therapy. However, in one human study that investigate the effects of raspberry ketones there was a significant decrease in body weight, body fat mass, waist and hip girth, while increasing lean mass compared to the placebo. Fat loss of 7.8% relative to the 2.8% in placebo, and weight loss of 2% relative to 0.5% in placebo[31], although in that study multi-ingredient was used but the main was Raspberry ketones, it has been suggested that Raspberry ketones enhance norepinephrine-induced lipolysis in adipocytes and prevent high fat diet induced body weight gain in mice.[32] These postulations were in agreement with the present results. It could be hypothesized that RK might affect in similar ways to Capsaicin, pungent principle of hot red pepper, which has been reported to decrease the adipose tissue weight and serum triacylglycerol content by enhancing energy metabolism.[33] In strategies to prevent obesity, one of the key steps is to inhibit the digestion and absorption of the dietary fat. Morimoto et al., explore this strategy further, they studied the effects of RK on fat absorption. RK at a concentration of 5% reduced the elevation of plasma triacylglycerol after oral administration of a lipid emulsion containing corn oil in rats, although a lower concentration of RK 1% elicited no such effect.[32] It has been suggested that RK suppresses the dietary fat absorption by inhibiting the trioleoylglycerol hydrolysis. Nevertheless, the inhibitory effect of RK on fat absorption is not the main anti obesity mechanism because the minimum RK dose required to exert these effects is much higher than that required to exert anti obese effects.[32] In this study the reduction in body weight were slightly decreased in RK group compared to the control group with no significant differences, this may be due to the small dose of RK that had been used in this study. The percent of reduction in MDA level was (56.57 %) in RK group and the elevation of glutathione level was (37.85 %), compared to the control group (26.95 % and 23.92 %) respectively, these results designate the effect of RK as antioxidant compound and was in agreement with Wang et al.[34] The 8-isoprostane f2α level reduced in RK group, this marker is widely accepted as a stable and reliable index of overall lipid peroxidation.[35]

Many clinical studies that have measured isoprostane (8-isoprostaglandin f2 α) levels by a variety of methods provide a significant body of evidence that many risk factors for chronic heart disease (CHD) increase overall lipid peroxidation, that higher isoprostane levels correlate with greater extent of CHD, that isoprostane levels predict disease outcomes, and its level can be used to assess the effectiveness of various therapies aimed at reducing the level of lipid peroxidation.[36] While low values of glutathione peroxidase along with increased

levels of isoprostane in obese women are indicative of defective protection mechanisms against oxidative stress and consequently carry increased risk for atherosclerosis.[37] The results of present study shows decreased F2-isoprostane with weight loss in all groups that consistent with a previous Davi et al.[38] It has been summarized that oxidative stress can be rapidly reduced and sustained through a modest reduction in caloric intake for a relatively short period.[39] And the reduction in BMI with L-carnitine group for present study was (2.38 kg/m2) while Alshammari [40], show (7.6 kg/m2) reduction which was higher than current study although, the reduction in BMI in a study by Alipour et al. was only (1.8 kg/m^2)[16], the difference in results may be due to that the younger age of patient sample in this study. It has been suggested that with advancing age, carnitine levels decline in all of human tissues and a carnitine deficiency leads to the extensive destruction of body mitochondria, this loss of mitochondrial function is likely to hasten death.[41] The reduction in weight and BMI in Raspberry ketones group in current study were (5.10%) and (5.08%) respectively. While Lopeze et al., registered (2%) reduction for body weight[31], this difference in results may be due to the concentration of RK in the combination of the supplement that used and the period of treatment which was only eight weeks in lopez study. L- carnitine is more effective than RK in reducing weight and BMI, while RK superior as anti oxidant. The percent of reduction in MDA level was (56.57 %) in RK group and the elevation of glutathione level was (37.85 %), compared to the control group (26.95 % and 23.92 %) respectively, these results designate the effect of RK as antioxidant compound which was in agreement with Wang et al.[34]

CONCLUSION

According to the results presented in this study it is conclude that the administration of L-carnitine and Raspberry ketones to obese women enhance the weight reduction and reduce BMI and waist circumference. Raspberry ketones are the most potent anti oxidant herbal treatment among two groups and can reduce the oxidative stress in obese patients.

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