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Abstract:

This study aims to investigate the effect of Shea seeds on the rat infected with fatty liver. This study was conducted on twenty five male albino rats weighing (150g± 10) and classified into five groups as follows: control (−ve), control (+ve), 5% Shea seeds powder, 10%shea seeds powder and Shea seeds extract rats groups. The last four groups were given by oral with amitriptyline (1m−mL−100g/ rats) for two weeks of the experiment to induce hepatic injury. The study was assigned for seven week. The results revealed that, the control (+v) rat group showed significant increase in final weight, body weight gain, TC, TG, LDL−c, VLDL−c, MDA, AST, ALT, Creatinine, Uric acid, Urea and Total Bilirubin. However showed significant decrease in HDL−c, TAC and T. Protein, by comparing with control (−v) group. On the other hand the treated groups with Shea seeds and Shea extract showed significant decrease in final weight, daily feed intake, body weight gain, TC, TG,

LDL-c, VLDL-c, MDA, AST,ALT, Creatinine, Uric acid, Urea and Total Bilirubin while showed significant increase in HDL-c, TAC and T. Protein, when comparing with control (+v) group. This result showed that Shea seeds and Shea extract play an important role to enhance our public health.

Key words: Shea, fatty liver, amitriptyline.

Introduction:

Fatty liver was produced by the accumulation of fat in body, which promotes liposuction and results in damage to hepatic cells in the chronic stages. Medicinal herbs were used to reduce blood lipids, which helps reduce cardiovascular disease and in liver metabolism alterations (**Jayasooriya *et al.*, 2000**).

A liver disorder caused by metabolic syndrome is a cause of fatty liver which increase due to obesity increased outcomes. Fatty liver does not represents any riskiness on health, but transmitted fatty liver to steatohepatitis is considerable health problem one of the prevalent causes of liver carcinoma and cardiovascular diseases (**Al-Okbi *et al.*, 2015**).

Amitriptyline hydrochloride 3-(10,11-dihydro-5 dibenzo"a", "d"cycloheptene-5-ylidene)-N, N-dimethyl-1-propanamine, effect is a dibenzo cycloheptene-derivative tricyclic antidepressant (TCA) and analgesic. Amitriptyline is the treatment belongs to the treatment group of addiction to the treatment of disorders of the nervous system (neurodrenaline) in the central nervous system, which helps to modify

the patient's mood and remove depression. Amitriptyline elevated liver enzymes by receiving it continuously (**Selim *et al.*, 1999**).

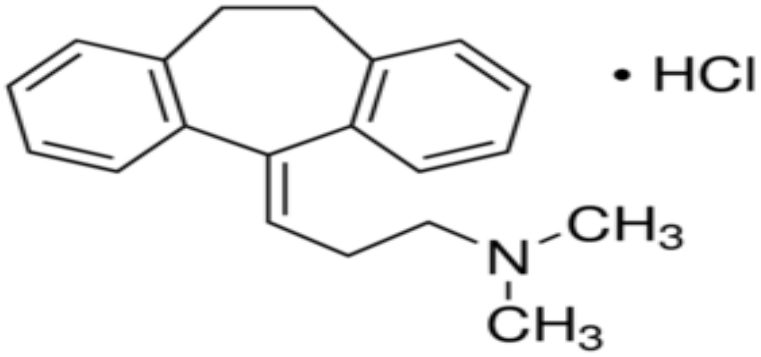

Figure (1): Amitriptyline structure

Amitriptyline caused mitochondrial membrane potential reduction (**Taziki *et al.*, 2013**). Although studies on antidepressant-induce liver injury are rare, 0.5%-3% of antidepressant traded patients may have a symptomatic mild elevation of levels of serum aminotransferase. All antidepressants can induce liver injury, particularly with polypharmacy in elderly patients. Most of liver damage cases are unpredictable and idiosyncratic, and it is mostly unlinked to drug dosage. Onset of liver damage is generally between several days and 6 month after treatment initiation and antidepressant-induced liver injury has been described as life threating including fulminant liver failure or death (**Perlemuter *et al.*, 2014**).

Salvia hispanica L., commonly known as Shea, is an annual plant belonging to the Lamiaceae family. Originating in such countries as

Guatemala, Mexico and Colombia, Shea seed was used and consumed as a source of energy and incorporated into a number of foods in the diet of the indigenous Aztec civilization (**Ulbricht *et al.*, 2009**).

Shea tree grows from Senegal in the west to Ethiopia at the east within a soudano-sahelien landscape known as Shea belt. It's divided in to 2 subspecies: nilotica and paradoxa (**Biochem 2007**). Shea seed is a great source of nutrients, especially polyunsaturated fatty acids like α-linolenic acid which, can be converted into eicosapentaenoic and docosahexaenoic acids, both essential fatty acids to the organism.Linoleicacid (c18:3-w36-20%). Also, it contained presenting a very low n-6/n-3 ratio (around 0.30).

Furthermore, it contained high levels of protein (16 - 26%, mainly prolamins, glutelins, globulins, albumins) with nine essential amino acids in considerable amounts (the most abundant is glutamic acid). Carbohydrate content ranges from 23 to 41%. The dietary fiber includes cellulose, lignin, hemicellulose, pectin, mucilage, gums and other oligosaccharides and polysaccharides with a range of 85% of insoluble fiber. Moreover, it rich in vitamins such as vitamin C, E,B complex thiamine, riboflavin, niacin and folic acid and minerals such as calcium, iron, phosphorus, magnesium, zinc, potassium and selenium. Moreover, Shea seed yield edible oil which is rich source of polyunsaturated fatty acids (PUFA) along with protein, dietary fiber, minerals and polyphenolic compounds (**Capitani *et al.*, 2012**). Shea seed contain about 25-38% oil and have the highest known percentage (60%) of alpha-linolenic fatty acid (**Ixtaina *et al.*, 2011**). In addition Shea seed oil extraction

residue is a good source of dietary fiber and phenolic compounds with antioxidant capacity **Reyes-Caudillo *et al.*, (2008)**.

Material and Methods:

Material:

Shea (*Salvia hispanica*.L) seed powder was purchased from herbal center Cairo-Egypt.

Amitriptyline:

Drug product by Kahira /MAD Company for pharmaceuticals and Medicinal Egypt.

Experimental rats:

Twenty five male of albino rats (Sprague Dawley) weighting 150 ±10 g, provided from National Research Center, Cairo, Egypt. Rats were housed as groups in wire cages under the normal laboratory conditions.

Methods:

Preparation of aqueous extract of Shea seeds:

The powder of Shea seed (10g) were soaked in (500 mL) redistilled water at (60°C) for 12 hrs then filtrated by the filter paper and stored at -5°C until used according to **Singh *et al.*, (2001)**.

Chemical composition of shea seeds powder:

Moisture, protein, fat, fiber and ash contents were determined according to **AOAC (2010)**. The carbohydrate was calculated by difference, according to **Chatfeld and Adamas, (1940)**.

Determination of fatty acids:

The fatty acids of Shea seed oil were determined according to **ISO 5508 (1990)** and **ISO 5509 (2000)** by gas chromatography (GC) as described by **Nath, (1996).**

HPLC analysis of polyphenols and total phenols of Shea seeds:

Phenolic compounds of Shea seed were studied using the reference HPLC method by comparing experimental retention times with reported reference values **Sakakibara *et al.*, (2003).** Total phenols of Shea seeds were determined according to **Singletion and Rossi (1965)** and **Akin *et al.*, (2008).**

Experimental animals:

The study was carried out at the animal house of National Research center, Cairo. Twenty five male albino rats weighing (150g± 10) were fed a standard diet for 7 day as an adaptation period. The standard diet was formulated according to **Reeves *et al.*, (1993).** Salts mixture and Vitamin mixture were prepared according to **Hegested, (1941)** and **Campbell (1961).** The composition of basal diet was casein 12%, corn oil 10%, salts mixture 4%, vitamin mixture 1%, cellulose 5%, and starch 67.5%. The rats were housed in wire cages under the normal laboratory conditions.

Every day the rats were observed for the external appearance, shape, color and distribution of hair and physical activity. The diets were introduced to rats in special food cups to avoid loss of food and contamination. Also water was provided to rats by glass tube projecting

through wire cages from inverted bottles supported to one side of the cage. Food and water provided were checked daily.

Experimental design:

The first main group (n=5 rats): were fed on basal diet and keep on as negative control group. The second main group (n=20 rats) : were taken orally with amitriptyline (1ml /100g rats) for two weeks then two rats were taken to detect the accumulation of fat in the liver by histopathological examination, divided intofour groups as following:

Group (2): Rats fed on standard diet and saved positive control group

Group (3): Rats fed on standard diet containing 5% Shea/kg /diet.

Group (4): Rats fed on standard diet containing 10%Shea /kg/ diet.

Group (5): Rats fed on standard diet & with l ml/100g of aqueous Shea extract (orally)

All rats were weighted at the beginning and the end of experiment to determined body weight gain (BWG). Liver, were removed and washed in saline solution. Body weight gain and relative organs weight were calculated according to **Chapman *et al.*, (1959)** using the following formulas:

BWG (g) = final weight – initial weight

Relative organ weight = body weight of rat / organs weight ×100

Blood sampling:

Blood samples were collected after 12 hour fasting at the end of the experiment (7weeks). Using the retro-orbital by means of a micro

capillary glass tubes, blood was collected into a dry clean centrifugal tube and left to clot in a water bath (37 ℃) at room temperature for half an hour. the blood was centrifuged for 10 minutes at 3000 rpm to separate the serum and transferred into clear quit fit plastic tubes and kept frozen at (−2 ° C) until analysis.

Biochemical analysis:

Determination of liver functions:

Alanine amino transferase (ALT), aspartate amino transferase (AST) enzymes were measured according to the methods described by **Bergmeyer and Harder (1986).** Total bilirubin, and total protein were determined according to the methods described by **Tietz, (1976), Henry, (1974), Moss, (1982), Gowenlock *et al.*, (1988) and Doumas *et al.*, (1973)** respectively.

Determination of lipid profile:

Total cholesterol (TC), Triglycerides (TG) and high density lipoprotein (HDL_c) were determined in serum according to the methods described by **Allain (1974), Fassati and Prencipe (1982)** and **Schmidt-sommerfled (1981)** respectively. Atherogenic Index (Total cholesterol / HDL-cholesterol) were calculated according to the equation of **Golay *et al* (1990).** Low density lipoprotein (LDL) and very low density lipoprotein (VLDL) were determined according to the method of **Lee and Nieman (1996)** and calculated using the following equations:

LDL (mg/dl) = Total cholesterol − (HDL + VLDL).

VLDL (mg/dl) = Triglycerides /5.

Determination of kidney functions:

Urea, uric acid and Creatinine were determined according to the method of **While *et al.,* (1970)** and **Henry (1974)** and **Houot (1985)** respectively.

Determination of antioxidant status:

Total antioxidant capacity (TAC) and Malondialdehyde (MDA) were determined according to the methods described by **Hu (1994), Aebi (1974)** and **Jentzsch *et al.*, (1996),** respectively.

Histopathological examination:

Histopathological examination was performance in pathology department veterinary college, Cairo Univ. according to the method of **Carlton *et al.*, (1967).** Where Hand E X 400.

Statistical analysis:

The obtained data were statistically analyzed using analysis of variance (ANOVA) SPSS. Version 19

Results and Discussion:

Proximate composition (dry basis %) of shea seed:

Table (1) summarized the results of chemical composition of Shea seeds (dry basis). The values of Shea chemical composition were 42.1, 30.7, 16.5, 30.4, 4.6 and 4.8 for total carbohydrates, fat, protein, crude fiber, moisture, and ash, respectively. These results agree with **Maira *et al,* (2014)** who reported that Shea seeds were rich in protein, fats, and dietary fiber.

Table (1): Chemical composition of dry basis of Shea seed

Constituents (g/100g)	**Value**
Moisture	4.6
Protein	16.5
Fat	30.7
Ash	4.8
Dietary fiber	30.4
Total carbohydrates	42.1

Values are expressed as mean of three triplicates

Total phenols and antioxidant activity of shea seed:

Table (2) showed the total phenols and antioxidant activity in Shea seeds. The data showed that the total phenols levels was 1.014 while the antioxidant activity was5.43 for Shea seeds. The presence of phenols and antioxidant in Sheamay be gives importance to the alleviation of fatty liver.

Table (2): Total phenols and antioxidant activity of shea seeds

Samples	**Test results**	
	Total Phenols mg/gm	**Antioxidant activity%**
Shea	**1.014**	**5.43**

Fatty acids in Shea seeds:

Data in **table (3)** showed the main essential fatty acids in Shea seeds. Analysis revealed significant levels of polyunsaturated fatty acids which qualified as essential fatty acids Shea seeds are particularly rich in monounsaturated oleic acid and polyunsaturated linoleic acid. In addition stearic acid the precursor of unsaturated fatty acids confirms the good nutritional quality with functional properties of Shea seed oil. In general, unsaturated fatty acids represented for 55.6% of total Shea acids.

Table (3): Fatty acids in Shea seeds

Fatty acid	**Common Name**	**Mean** SD
14:0 SFA	Myristic acid	0.11± 0.03
16:0 SFA	Palmitic acid	6.82± 1.11
18:0 SFA	Stearic acid	22.91± 3.94
20:0 SFA	Arachidic acid	0.74± o.14
16:1 USFA	Palmitoleic acid	0.14± 0.05
18.1	Oleic acid	54.76±

USFA		4.72
18.2 USFA	Linoleic acid	8.4± 2.5
18.3 USFA	Linolenic acid	3.42± 1.03
22.1 USFA	Erucic acid	0.65± 0.20
UnKnowns	C	2.05± 0.40

Values reported as means ± SD of three replicate analyses; USFA: unsaturated fatty acids.

The composition of Shea seeds from flavonoids:

The present data in **table (4)** showed Shea seeds contain seventeen elements of flavonoids. The highest value was Apig 6-rhamnose 8-glucose it worth was 68.34 followed by Rutin, Apig 6- arabinose 8-glactose their values were 65.18 and 41.42 respectively while the remaining elements give relatively small proportions.

Table (4): The composition of Shea seeds from flavonoids

Flavonoids	**Test results flavonoids(ppm)**
	Shea
Apig 6- arabinose 8- glactose	41.42
Apig 6-rhamnose 8- glucose	68.34
Naringin	8.98
Luteolin 7 –glucose	-
Hespirdin	-
Rutin	65.18
Apig 7-O- neohespirosid	9.49
Kamp.3.7 – dirhsmoside	2.05
Quercetrin	7.42

Apigenin –7 – glueose	1.99
Acacetin – 7 – neo hesperside	1.01
Kaempfero13–(2–p–comaroyl) glucose	5.45
Acacetinneo.rutinoside	2.25
Quercetrin	1.27
Naringenin	0.67
Hespirtin	4.75
Kaempferol	0.58
Rhmnetin	4.80
Apegnin	1.58

The composition of Shea seeds from phenolic compounds:

The present data in **table (5)** showed Shea seeds contain twenty elements of phenolic compounds. The highest value were Catectein, pyrogallol, protocatchein, Ferulic, Benzoic, 4–amino–benzoic, p–oh–benzoic and Catechol their values were 19.22, 17.93, 15.33, 15.31, 9,76, 7,95, 6.62 and 6.39 respectively 17.93, 15.33 and 15.31 respectively while the remaining elements given approximate proportions.

Table (5): The composition of Shea seeds from phenolic compounds

Phenolic compounds	Test results of phenolic compounds(ppm)
	Shea
Gallic acid	0.17
Pyrogallol	17.93
4-amino-benzoic	7.95
Protocatchein	15.33
Catectein	19.22
Chlorogenic acid	1.63
Catechol	6.39
Caffeine acid	1.00
p-oh-benzoic	6.62
Caffeic acid	2.38
Vanillie	1.17
p-coumaric acid	4.95
Ferulic	15.31
los-ferulic	0.64
Alpha – coumaric	-
Ellagic acid	4.25
Benzoic acid	9.76

Body weight gain, final weight feed intake and feed efficiency ratio:

Data presented in **Table (6)** showed the mean values of body weight gain, feed intake and feed efficiency ratio of experimental rats. The untreated group (positive control) showed significant increase in final weight, weight gain and feed intake compared with negative control group and all treated groups. But in feed efficiency ratio showed significant increase in the treated group fed on (5% and 10%shea) compared with positive control group.

On the other hand in final weight and weight gain treated groups fed on 5%, 10% Shea and Shea extract showed significant gradually increase compared with normal control group. Whilst in feed intake and feed efficiency ratio of treated groups fed on 5% and 10% Shea showed significant increase expect treated group fed on Shea extract showed same nearly result compared with normal control group. Shea seeds, (*Salvia hispanica*. L) rich in a linoleic acid improves adiposity dietary Shea seed reduced the visceral adiposity **Adriana *et al.*, (2008)**. Weight loss is also due to the containment of the shea seeds on a high percentage of Mono unsaturated fatty acid, which amounts to 54.9%, which helps reduce the volume of fat in the body.

Table (6): Mean values ± SD of initial weight, body weight gain, feed intake and FER for experimental rats

Groups	Normal control	Positive control	Groups treated with		
			5%SSP	10%SSP	SSPE

Variables					
Initial weight (g)	142.05± 9.70 ^{a}	147.00± 6.16 ^{a}	149.00± 5.41 ^{a}	148.05± 5.62 ^{a}	147.25± 3.10 ^{a}
Final weight (g)	251.75± 10.35 ^{cd}	297.00± 14.52 ^{a}	284.00± 13.98 ^{a}	279.75± 12.30 ^{a}	263.04± 11.11 ^{bc}
Weight gain (g)	109.25± 10.06 ^{c}	149.50± 14.82 ^{a}	135.00± 13.08 ^{a}	131.25± 13.89 ^{a}	116.56± 11.92 ^{c}
Daily feed intake (g)	15.25± 1.25 ^{b}	19.50± 1.29 ^{a}	15.50± 1.03 ^{b}	14.75± 1.95 ^{c}	15.02± 1.12 ^{bc}
Feed efficiency ratio(FER)	0.073± 0.001 ^{b}	±0.078 0.002 ^{a}	0.080± 0.001 ^{b}	0.093± 0.001 ^{b}	0.077± 0.001 ^{b}

Significant with control group * $p<0.05$ ** $p<0.01$ *** $p<0.001$

Mean values in each column having different superscript (a, b, c, d) are significant.

Lipid profile:

Table (7) showed the effect of Shea seeds powder with different levels on lipid parameters of fatty liver rats. There was significant increase of TC, TG, LDL-c and VLDL-c in untreated group (positive control) when compared with negative control group ($p <0.05$). However there was significant descending decreases on TC, TG, LDL-c and VLDL-c levels of all the treated rat group feeding on Shea seeds powder 5%, 10%shea and Shea extract when compared with positive control group ($p<0.05$).While we found a significant increase on (HDL_c) in treated group feeding

on 5%, 10% Shea and Shea extract comparing with positive control group. On other hand there were significant increases on TC, TG, LDL-c and VLDL-c levels of treated rat group feeding on Shea seeds powder 5%,10% Shea and Shea extract) when compared with negative group. While we found a significant decrease on (HDL) in treated group feeding on (5%SSP, 10%SSP, and 10%SSPE) comparing with negative control group. While the serum levels of HDL-C tended to decline in positive control group comparing with negative group. After treatment with Shea and quinoa seeds powder with different doses, TC, TG, LDL-c and VLDL-c levels were decreased compared to the untreated fatty liver group ($P < 0.05$).

Induction of liver toxicity by amitriptyline led to significant increases ($p<0.05$) in the total cholesterol (TC), triglycerides (TG), low density lipoprotein cholesterol (LDL-c) and very low density lipoprotein cholesterol (VLDL-c) levels as shown table (3) in agreement with the findings of (**Kaplowitz 2013**). Shea seeds contain the essential acid; it is induced lipid redistribution with lipid trafficking away from the visceral fat and liver (**Rafaela *et al.*, 2015**).

Table (7): Mean values ± SD of lipids profile for experimental rats

Groups / Variables	*Normal Control*	*Positive Control*	*Groups treated with*		
			5%SSP	10%SSP	SSPE
T C	63.80±	107.55±	92.35±	84.31±	70.02±

Mg\dl	7.34 ^{f}	12.75 ^{a}	8.62 ^{b}	7.43 ^{d}	7.22 ^{e}
TG Mg\dl	75.77± 9.49 ^{e}	119.38± 11.61 ^{a}	104.11± 10.22 ^{ab}	97.07± 10.21 ^{b}	87.51± 7.53 ^{c}
HDL-c Mg\dl	39.57± 3.71 ^{a}	26.15± 2.43 ^{e}	27.12± 2.61 ^{e}	32.02± 1.75 ^{d}	38.12± 2.74 ^{a}
LDL-c Mg\dl	22.17± 2.04 ^{e}	40.30± 3.92 ^{a}	33.03± 2.12 ^{b}	30.32± 2.66 ^{c}	22.97± 2.88 ^{e}
VLDL-c Mg\dl	15.15± 1.90 ^{d}	24.68± 2.32 ^{a}	20.63± 2.14 ^{b}	19.41± 2.04 ^{b}	17.50± 1.51 ^{c}

Significant with control group * $p<0.05$ ** $p<0.01$ *** $p<0.001$

Mean values in each column having different superscript (a, b, c, d) are significant.

Relative weight of Liver, Kidney, Heart and Spleen:

The presented data in **Table (8)** showed Liver, Kidney, Heart ratio and Spleen relative weight of Liver injury rat groups. The untreated group (positive control) showed significant increase in Liver, Kidney, Heart relative and Spleen Ratio weight in comparison to normal control group. Also the untreated group (positive control) showed significant increase in Liver, Kidney, Heart Ratio and Spleen Ratio weight in comparison to all treated group.

Whereas the liver weight of treated groups fed on 5%shea and Shea extract showed significant increase in liver weight in comparison to negative control group. However the treated group feeding on Shea extract showed the same result in liver weight in comparison to normal control group. On the other hand the treated group feeding on 5%, 10%Shea and Shea extract showed the nearly result in kidney weight in comparison to negative control group.

These results are in agreement with **Essengue *et al.*, (2009); Das *et al.*, (2012)** and **Compton *et al.*, (2017)** they reported that Shea oil consider as a good source of important vitamins and minerals including vit. E, vit C, D which they are function as a free radical scavengers and protect body from oxidative stress and free radicals which can cause a wide damage of the organs.

Table (8): Mean values ± SD of relative weight of liver, kidney, heart and spleen for experimental rats

Groups / Relative weight of	Normal control	Positive Control	Groups treated with		
			5%SSP	10%SSP	SSPE
Liver Ratio	4.3± 0.43 b	5.23± 0.38 a	4.5± 0.22 b	4.26± 0.27 b	4.46± 0.20 ab
Kidney Ratio	0.97± 0.12 b	0.99± 0.11 a	0.97± 0.10 b	0.98± 0.12 ab	0.96± 0.10 ab
Heart Ratio	0.315± 0.055 c	0.355± 0.063 a	0.341± 0.036 b	0.335± 0.026 b	0.338± 0.037 b
Spleen Ratio	0.311± 0.056 c	0.338± 0.053 a	0.328± 0.051 ab	0.345± 0.078 a	0.328± 0.053 ab

Significant with control group * $p<0.05$ ** $p<0.01$ *** $p<0.001$

Mean values in each column having different superscript (a, b, c, d) are significant.

Antioxidant capacity (TAC) and Malondialdhyde (MDA):

The presented data in **Table (9)** the untreated group (positive control) showed significant decrease in total antioxidant capacity in comparison to normal control group. Whilst the same group gave a significant increase in Malondialdhyde in comparison to normal control group. On the other hand the all treated groups fed on Shea seeds showed

significant increase in total antioxidant comparing with positive control group whilst the all treated group showed significant decrease in total antioxidant comparing with normal control group. However in Malondialdhyde the groups treated fed on Shea seeds showed significant decrease comparison with positive control group. While the all treated group showed significant increase in Malondialdhyde comparison with normal control group.

This result is due to analysis of the analysis of the seeds in the table (3) Shea seed and oil is an excellent source of antioxidants such as tocopherols, phytosterol, carotenoids and phenolic compounds, chlorogenic acid, caffeic acid, myricetin, quercetin and Kaempferol (**Ixtaina *et al.*, 2011**); **Reyes-Caudillo *et al.*, 2008** and **Capitani *et al.*, 2012**). Several reports demonstrate potent antioxidant property of shea seed among in vitro assays (**Alvarez-Chavez *et al.*, 2008**).

Table (9): Mean values ± SD of total antioxidant capacity (TAC) and Malondialdhyde (MDA) for experimental rats

Groups / Variables	Normal control	Positive Control	Groups treated with		
			5%SSP	10%SSP	SSPE
T A C Mmol\g	4.10± 0.22 ^{a}	1.51± 0.16 ^{c}	2.68± 0.15 ^{c}	3.75± 0.07 ^{b}	3.87± 0.06 ^{b}
MDA Nmol\g	39.46± 5.71 ^{e}	89.31± 10.33 ^{a}	51.33± 8.71 ^{b}	48.22± 7.11 ^{c}	44.18± 7.10 ^{d}

Significant with control group * p<0.05 ** p<0.01 *** p<0.001

Mean values in each column having different superscript (a, b, c, d) are significant.

Liver and kidney function parameters:

The presented data in **Table (10)** showed liver and kidney function parameters of fatty liver rat groups feeding on Shea seeds powder. The untreated group (positive control) showed significant increase in AST, ALT, Creatinine, Uric acid, Urea and Total bilirubin at p <o.05 in comparing with normal control group. And showed significant decrease in t. protein when comparing with normal control. Whilst the treated group showed significant decrease in AST, ALT, creatinine, uric acid, urea and total bilirubin at p <o.05 in comparing with positive control group.

Shea seeds contains important quantities of protein, minerals, fiber, polyphenols, and polyunsaturated fatty acids (PUFAs) and is currently known as one of the best plant sources of the omega_3 (n−3) fatty acid, a−linolenic acid (ALA) (5, 6, 7, 8 and 9).

Long term dietary intake of Shea seed is associated with increased bone mineral content and improved hepatic and intestinal morphology in sprague−dawley rats (**Montes Chani *et al.* 2018**).

Mean values ± SD of liver and kidney function for Table (10): experimental rats

Groups / Variables	Normal control	Positive Control	5%SSP	10%SSP	SSPE
A S T lu\ml	41.52± 5.16 ^{e}	75.12± 9.21 ^{a}	56.55± 4.92 ^{b}	43.80± 2.76 ^{d}	45.80± 5.83 ^{e}
A L T lu\ml	16.55± 1.95 ^{e}	30.37± 3.43 ^{a}	25.11± 2.03 ^{b}	20.22± 2.11 ^{c}	15.31± 3.66 ^{e}
Creatinine Mg\dl	0.85± 0.02 ^{d}	2.74± 0.21 ^{a}	2.23± 0.52 ^{a}	1.94± 0.11 ^{b}	0.78± 0.18 ^{e}
Uric acid Mg\dl	2.12± 0.33 ^{c}	4.44± 0.33 ^{a}	3.35± 0.45 ^{b}	3.08± 0.19 ^{b}	2.14± 0.28 c
Urea Mg\dl	24.80± 1.26 ^{d}	46.10± 3.08 ^{a}	34.05± 2.75 ^{b}	27.85± 2.17 ^{c}	24.75± 1.19 ^{d}
Total Bilirubin Mg\dl	0.29± 0.01 ^{e}	1.12± 0.12 ^{a}	0.75± 0.05 ^{b}	0.49± 0.03 ^{c}	0.29± 0.05 ^{e}
T.Protein g\dl	7.55± 1.51 ^{a}	4.01± 0.91 ^{c}	6.91± 1.11 ^{b}	6.72± 1.31 ^{b}	7.51± 1.11 ^{a}

Significant with control group * $p<0.05$ ** $p<0.01$ *** $p<0.001$

Mean values in each column having different superscript (a, b, c, d) are significant.

Histological Examination of liver

Liver histopathology:

Microscopically, liver of rats from normal control group revealed the normal histological (Photo1)

Structure of hepatic lobule **(Photo 2)** liver of rats from positive control group for amitriptyline group showed sever and diffuse fatty degeneration in the form of circumscribed vacuolated hepatocytes among the hepatic parenchyma (Photo 3) liver of rats from treated group with 5%SSP showed massively distributed of slightly vacuolated hepatocytes (Photo 4) Examined sections from treated group with 10%SSP showed massively distributed of moderately vacuolated hepatocytes (Photo 5) also liver of rats from treated group with 5% QSP explained moderately dilated blood sinusoids in between the hepatic parenchyma (Photo 6) Moreover, liver of rats from treated group with 10%QSP showed congestion in the central vein with vacuolated hepatocytes (Photo 7) also liver of rats from treated group with 5%S+QSP explained moderately dilated blood sinusoids in between the hepatic parenchyma (Photo 8) Moreover, liver of rats from treated group with 10%S+QSP showed Liver showing slightly dilated blood sinusoids in between the hepatic parenchyma (Photo 9) found in the liver of rats from treated group with 10%SSPE showed slightly dilated blood sinusoids in between the hepatic parenchyma (Photo 10) Examined sections from treated group with 10%QSPE showed massively distributed of moderately vacuolated hepatocytes

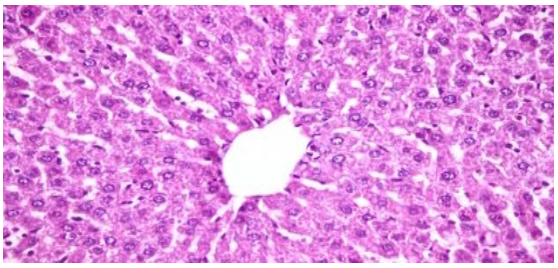

Photo (1): Liver showing normal hepatocytes and parenchyma (H&E X 400).

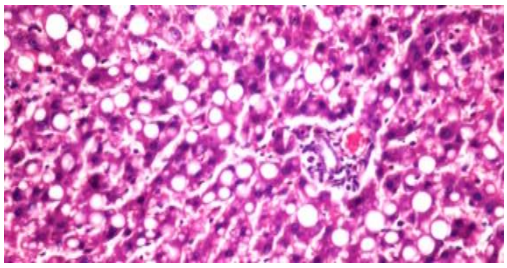

Photo (2): Liver of untreated group, showing sever and diffuse fatty degeneration in the form of circumscribed vacuolated hepatocytes among the hepatic parenchyma (arrows) (H&E X 400).

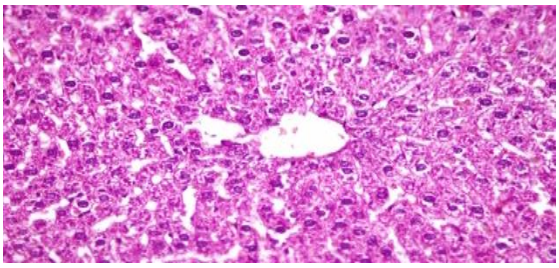

Photo (3): Liver of group feeding on 5%SSP, showing massively distributed of slightly vacuolated hepatocytes (arrows) (H&E X 400).

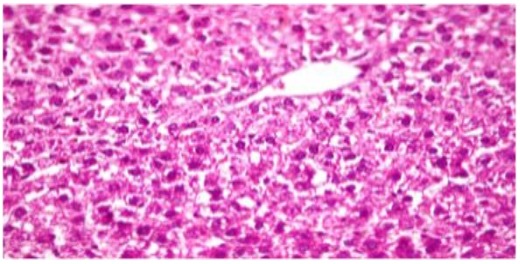

Photo (4): Liver of group feeding on 10%SSP, showing massively distributed of moderately vacuolated hepatocytes (arrows) (H&E X 400).

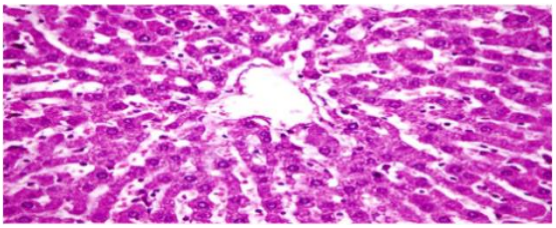

Photo (5): Liver of group feeding on 5% QSP, showing moderately dilated blood sinusoids in

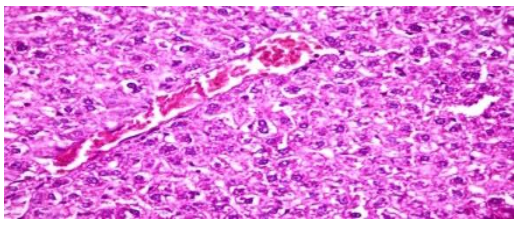

Photo (6): Liver of group feeding on 10% QSP, showing congestion in the central vein

between the hepatic parenchyma (arrows) (H&E X 400).

(arrow) with vacuolated hepatocytes (H&E X 400).

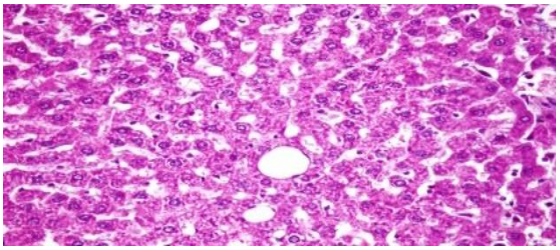

Photo (7): Liver of group feeding on 5%S+QSP, showing moderately dilated blood sinusoids in between the hepatic parenchyma (arrows) (H&E X 400).

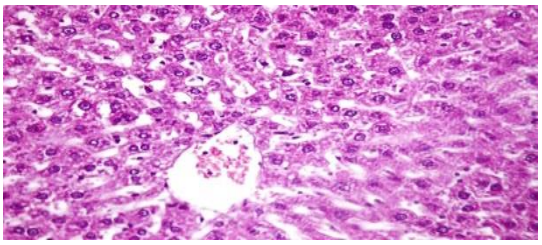

Photo (8): Liver of group feeding on 10%S+QSP, showing slightly dilated blood sinusoids in between the hepatic parenchyma (arrows) (H&E X 400).

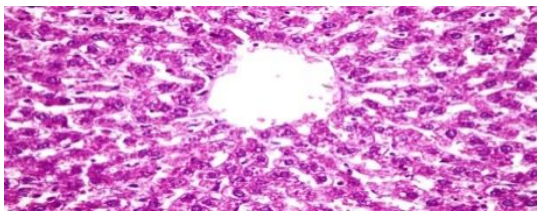

Photo (9): Liver of group administrated 10%SSPE showing slightly dilated blood sinusoids in between the hepatic parenchyma (arrows) (H&E X 400).

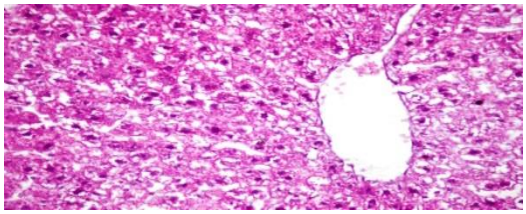

Photo (10): Liver of group administrated 10%QSPE, showing massively distributed of moderately vacuolated hepatocytes (arrows) (H&E X 400).

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تأثير بذور الشيا والمستخلص المائي علي الفئران المصـابه بالكبد الدهني

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تهدف هده الدراسة الى قياس تاثير بذور الشيا والمستخلص المائي لها على الفئران المصابة بالكبد الدهنى اجريت هذه الدراسة على ٢٥ من فئران التجارب تتراوح اوزانهم من ١٥٠ ± ١٠ جرام لمدة ٧ اسابيع وتم تقسيم الفئران الى ٥ مجموعات كالتالى: الضابطة السالبة، والضابطة الموجبة، ٥% بودر بذور الشيا ١٠،% بودر بذور الشيا والمستخلص المائي لبذور الشيا ولاحداث اصابة الفئران بدهون الكبد تم استخدام عقار الاميتربتلين وتم اعطاء ٤ مجموعات الاخيرة عقار الاميتربتلين كمحلول مائى ١٠ مليجرام / كجم من وزن جسم الفأر وفي نهايه التجربه تم أخد عينات الدم وعمل تحاليل: انزيمات الكبد ،انزيمات الكلي ، دهون الدم ومضادات الاكسده وقد اشارت نتائج التجربة الى ان المجموعة الضابطة الموجبة اظهرت ارتفاع معنوى فى الوزن النهائى والمكتسب ونسبه كفاءة الطعام والكلولسترول ودهون الدم، MDA,AST,ALT,Creatinine, acid, Urea Uric and Total Bilirubin مقارنه بالمجموعه الضابطه السالبه والمجموعات المعالجه بينما اظهرت المجموعات المعالجه ببدور الشيا ومستخلص بدور الشيا انخفاض معنوى في نفس التحاليل مقارنه بالمجموعه الضابطه الموجبه وعلي الجانب الاخر اظهرت المجموعات المعالجه ارتفاع معنوي في – TAC, HDL

c,البروتين الكلى مقارنه بالمجموعه الضابطه السالبه بينما اظهرت المجموعه الضابطه الموجبه انخفاض معنوي في نفس التحاليل مقارنه بالمجموعه الضابطه السالبه .

وتوصى الدراسة باستخدام بذور الشيا والمستخلص المائي للحفاظ على الصحة العامة والوقايه من دهون الكبد. و هذا يرجع الي احتواء بذور الشيا علي حمض الاوليك الدي يؤثر مباشره علي منع تكون دهون الجسم ويحمي من الازمات القلبيه المفاجئه ,كما ان احتواء بذور الشيا علي نسبه عاليه من الدهون الغير مشبعه تساعد علي تخفيض نسبه الكوليسترول الضار في الجسم مما يساعد علي عدم تراكم دهون الكبد