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Effect of *curcuma longa* L. on fatty liver induced by oxytetracycline in albino rats.

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Abstract

Background: *Curcuma longa* has been shown to be a potent anti-inflammatory, antioxidant and anticarcinogenic agent. The present investigation aimed at examining the possible potential protective effect of curcuma against oxytetracyclin-induced fatty liver in an attempt to understand its mechanism of action, which may pave the way for possible therapeutic applications.

Material and Methods: Albino rats were divided into two major groups, 15 rats for each. The first group was divided into three sub-groups: a) control, b) fatty liver group; that was injected intraperitonealy with oxytetracycline (120mg/kg) for three consecutive days resulting in steatosis and c) curcuma treated group; which was treated with curcuma (0.4 % of diet) for 30 days after fatty liver induction . All animals were scarified after 33 days of the beginning of the experiment.
The second group was divided into three subgroups: a) control, b) fatty liver group and c) drug protection group; which received curcuma for 15 days before induction of fatty liver, then sacrificed after induction of fatty liver (3 days). Blood samples were collected for biochemical analysis. Liver specimens were obtained and fixed in 10 % formalin for histological study.

Results: Fatty liver groups showed high significant increase in serum glucose, cholesterol, triglycerides, LDL cholesterol, ALT, AST, GGT, LDH, total protein, albumin, globulin, urea and creatinine while HDL cholesterol and A/G ratio were significantly decreased compared to control group.
Histopathological changes were detected in liver tissue of fatty liver rats. The treatment with curcuma ameliorated the biochemical parameters and histological changes. The pre-treatment with curcuma before the induction of fatty liver also ameliorated the results but they did not turn back to the normal values.

Conclusion: It is recommend to using curcuma as diet additive for fatty liver patients or those people who have hyperlipidemic family history.

Keywords: Fatty liver, curcuma, lipid profile, Albino rats, physiological parameters, histopathology.

Introduction:

Any organ of body is a potential target for injurious effects from chemicals but some organs are more vulnerable to adverse effects than others. The liver is often a target organ for a number of reasons. First, most toxicants enter the body *via* the gastrointestinal tract and after absorption they are carried by the hepatic portal vein to the liver. Thus the liver will be exposed to the highest concentrations of these chemicals (Reed, 1994; Lu, 1996). Chemicals encountered by other routes of exposure may also reach the liver through its blood supply from the hepatic artery as well as the portal vein (Stacey *et al*., 1993; Kulkarni and Byczkowski, 1994). Second, the liver has the ability to concentrate, biotransform and excrete chemicals, irrespective of routes of exposure (Plaa and Hewitt, 1982).

Fatty liver is a reversible condition where large vacuoles of triglycerides accumulate in liver cells by the process of steatosis (Reddy and Rao, 2006).

Fat in liver usually causes no damage by itself. However, it may be a sign of more harmful condition (Angulo, 2000).
Fatty liver can occur in diabetes mellitus, obesity, pregnancy and induced by certain drugs

or toxins as tetracyclines and carbon tetrachloride (Araya, 2006).

Tetracycline is one of a group of drugs known to induce micro vesicular steatosis when triglyceride accumulation was concomitant with the inhibition of mitochondrial lipid metabolism. (Amacher and Martin, 1997).

Recent researches have examined the effects of plants used to support liver functions and treat diseases in the liver. *Curcuma longa* (Zingibaeraceae) rhizomes, commonly known as turmeric, have been traditionally used as a source of coloring matter for foods, cosmetics, and as amedicinal formulations in India and other countries and is claimed as effective in folklore ailments; system of medicine for several common ailments (Sarah *et al*., 2009).

Curcumin is the major secondary metabolites of turmeric which has been responsible for the pharmaceutical activities of turmeric powder (Sharma, 1979). The main activities have been found to be anti-inflammatory, hepatoprotective, anti-microbial, wound healing, anti-cancer, anti-tumor and anti-viral (Sikora *et al*., 2011).

Fatty liver causes were markedly increased in Egyptian people throughout last years. In Egyptian folk medicine curcuma is a most preferable plant used to treat fatty liver.

This study aims to examine if curcuma can treat fatty liver and if it can protect against factors that experimentally induced fatty liver. It investigates also its effect on some vital organs as liver, kidney and heart.

Material and Methods:

*** Chemicals:**

Curcuma was obtained in the form of powder and mixed with food.

Oxyteracycline was obtained from El-Nile pharmaceutical company (Cairo, Egypt).

*** Animal groups and treatment:**

30 female albino rats of local strain weighting 150 - 200 gm were obtained from El-Nile pharmaceutical company (Cairo, Egypt). All rats were fed on balanced diet and water and allowed to be acclimatized for 10 days before the beginning of the experiment. The animals were housed in metal cages (5 per cage) and maintained under prevailing atmospheric conditions with continuous cleaning and observation.

The rats were divided into two groups, the first group was to follow up the effect of curcuma as curative plant for fatty liver, while the second group was to illustrate the effect of this plant as protective agent against fatty liver. Each group was subdivided into three subgroups.

The first group was divided into:

1- Control group:

Five rats injected intraperitonealy with saline (120 mg / kg) for three consecutive days.

2- Oxytetracycline group:

Five rats were injected intraperitonealy with oxytetracycline (120 ml / kg) for three consecutive days for fatty liver induction (Nicola *et al*., 1996).

3- Curcuma curative group:

Five rats were injected intraperitonealy with oxytetracycline (120 mg / kg) for three consecutive days then fed on curcuma (0.4 % of diet) for 30 days.

All animals were scarified after 33 days of the beginning of the experiment.

The second group divided into:

1- Control group:

Five rats were injected intraperitonealy with saline (120 ml / kg) for three consecutive days then scarified.

2- Oxytetracycline group:

Five rats were injected intraperitonealy with oxytetracycline (120 mg / kg) for three consecutive days for fatty liver induction then scarified (Nicola *et al*., 1996).

3- Curcuma recovery group:

Five rats were fed on curcuma (0.4 % of diet) for 15 days then injected intraperitonealy with oxytetracycline (120 mg / kg) for three consecutive days then scarified.

Preparation of Samples:

The animals were anesthetized under light ether anesthesia, blood samples were collected then all animals were scarified. Blood samples were kept for 30 minutes without disturbance then centrifuged for 15 – 20 minutes at 5000 rpm to separate serum. The livers were processed for preparation of 5 µm. thick paraffin sections and

stained with H & E (Drury and Wallington, 1980).

Biochemical Studies:

Serum glucose was estimated according to enzymatic colorimetric method described by Tietz (1986). Alanine amino transferase (ALAT) and aspirate amino transferase (ASAT) were determined by the method of Breuer (1996). Serum γ-glutamyl transfers (GGT) was performed by kinetic method according to Persijn *et al*. (1976). Serum total protein was estimated according to Doumas *et al*. (1975). Serum albumin was determined according to the method of Webster (1977). The globulin value for each sample was obtained by substracting the albumin value from the corresponding total protein value. The A/G ratio for each sample was obtained by dividing the albumin level to globulin level. Total lipids were assayed by the method of Kaplan (1984). Serum total cholesterol (T.C) was performed according to Henry *et al*. (1974). Serum triglycerides (T.G) were determined according to the method of Fossati and Prencie (1982). Serum high density lipoproteins cholesterol (HDL-Cholesterol) was assayed according to Burstein (1970). The concentration of low density lipoproteins cholesterol (LDL-Cholesterol) in serum was estimated by the equation used by Friedewald *et al*. (1972) as follow:

$$LDL-cholesterol\ (mg/dl) =$$

$$Total\ cholesterol - HDL\ cholesterol\ -\left(\frac{T.G}{5}\right)$$

Measurement of Serum Urea was done according to the method of Patton and Crouch (1977). Serum creatinine was evaluated according to the method of Jaffe (1980).

Statistical Analysis:

The results were expressed as mean ± standard error (SE). The significance of differences between means was measured by student's t-test (Snedecor and Cochran, 1980). The P values below 0.05 were considered significant.

Results:

As shown in table (1), fatty liver group showed high significant increase in serum glucose level when compared with control group.

Induction of fatty liver with oxy tetracycline and treatment with curcuma for 30 days showed insignificant change in serum glucose level when compared with control group, while it showed high significant decrease ($P < 0.01$) when compared with fatty liver group.

On the other hand, giving curcuma before induction of fatty liver was associated with hyperglycemia in rats when compared with control and fatty liver groups.

As shown in table (2), fatty liver group showed a high significant increase ($P < 0.01$) in serum total lipids, triglycerides and LDL-cholesterol while HDL-cholesterol was significantly decreased when compared with control group.

After induction of fatty liver with oxy tetracycline followed by treatment with curcuma for 30 days there was insignificant change in serum total lipids, cholesterol, triglycerides, LDL-cholesterol and HDL-cholesterol when compared with control group while significant decrease was recorded in serum total lipids, cholesterol, triglycerides and HDL. LDL was high significantly decreased when compared with fatty liver group.

Protective group recorded significant increase ($P<0.01$) in total lipids and triglycerides when compared with control group but it showed high significant decrease ($P<0.01$) when compared with fatty liver group. Regarding to cholesterol, no significant change was recorded in curcuma group as compared to control. Curcuma also significantly reduced LDL level but it didn't effect HDL reduction which was caused by oxy tetracycline in comparison with control rats.

Table (3) showed that, fatty liver group recorded high significant decrease ($P<0.01$) in serum total protein, albumin and globulin while A/G ratio was significantly increased ($P<0.01$) when compared with control group.

Treating rats with curcuma resulted in ameliorated oxy tetracycline effect on serum total protein, albumin, globulin and A/G ratio when compared with control group.

On the other hand, giving curcuma to rats before induction of fatty liver didn't change the

effect of oxytetracycline on serum protein profile.

Table (4) showed a high significant increase in serum liver enzymes (ALAT, ASAT, GGT and LDH) in fatty liver group when compared with control group.

Treating rats with curcuma recovered ASAT, ALAT and GGT activities to normal values. It also decreased LDH activity where it recorded high significant decrease when compared with fatty liver group.

On the other hand, giving curcuma for 15 days before induction of fatty liver reduced the effect of oxy tetracycline on AST, GGT and LDH when compared with fatty liver group but didn't have effect on serum enzymes when compared with control group.

Table (5) showed a significant increase ($P< 0.05$) in serum urea and creatinine in fatty liver group when compared with control group.

It was found that rats treated with curcuma after induction of fatty liver showed a significant decrease in serum urea and creatinine when compared with control group, while serum urea and creatinine showed a high significant reduction when compared with fatty liver group.

On the other hand, giving curcuma before induction of fatty liver ameliorated the effect oxy tetracycline on serum urea and creatinine.

Histopathological results:

The liver of control rats was formed of the classic hepatic lobules. Each lobule showed radially arranged hepatocytes forming cords around the central vein. Hepatocytes appeared polygonal in shape with rounded vesicular nuclei. Blood sinusoids were seen separating the cords of the liver cells and lined by flattened endothelial cells and von Kupffer cells (Plate 1A).

Examination of liver sections of rats injected with oxy tetracycline for three consecutive days showed apparent enlargement of hepatocytes up to ballooning. Cells all over the hepatic lobule were seen to contain mainly macro vacuoles dispersed throughout the cytoplasm (Plate 1B). Some cells had around one large vacuole filling the whole cell with thin rim of cytoplasm around and pushing the nucleus to one side. Other cells contained smaller vacuoles with either central or eccentric nuclei (Plate 1B).

After treatment with oxy tetracycline for three consecutive days and curcuma for one month, liver lobular morphology similar to the control rats (Plate 1C).

One month after oxy tetracycline injection, liver sections showed many fat vacuoles and ballooned hepatocytes; apoptosis was abundant (Plate 1D).

Treatment with curcuma for 15 days before induction of fatty liver with oxy tetracycline, most of the hepatocytes had vesicular nuclei and mildly vacuolated granular cytoplasm (Plate 1E) when compared to fatty liver groups (Plate 1, B and D).

Table (1): Effect of curcuma on serum glucose in treated and recovery groups.

		Treated			Protective group		
		Control	Fatty liver	Curcuma	Control	Fatty liver	Curcuma
Glucose (mg/dl)	Mean	74.1	95.0	73.4	74.9	96.1	88.1
	± SE	1.48	1.22	1.6	1.4	1.3	1.4
	P1		**	N.S		**	**
	P2			**			**

Each value is the mean of 5 animals ± SE.

N.S: non significant.

*, **: is the significant difference when compared at $P< 0.05$ and $P < 0.01$ respectively.

P1: in comparison with control group.

P2: in comparison with fatty liver group.

Table (2): Effect of curcuma on serum total lipids, Triglycerides, cholesterol, LDL and HDL in treated and recovery groups.

		Treated			Protective group		
		Control	Fatty liver	Curcuma	Control	Fatty liver	Curcuma
Total Lipids (mg/dl)	Mean	309	411.4	313.7	306.2	408.8	389.4
	± SE	2.7	2.1	1.7	2.1	1.6	1.6
	P1		**	N.S		**	**
	P2			**			**
Triglycerides (mg/dl)	Mean	42.4	90.4	43.4	41.3	89.5	76.3
	± SE	1.9	1.4	1.7	1.1	1.3	1.6
	P1		**	N.S		**	**
	P2			**			**
Cholesterol (mg/dl)	Mean	95.8	104.5	95.1	94.6	106.1	98.3
	± SE	1.5	1.5	2.1	1.4	1.8	1.7
	P1		**	N.S		**	N.S
	P2			**			*
LDL (mg/dl)	Mean	40.6	48.02	40.1	38.5	48.1	42.9
	± SE	2.88	1.4	1.4	1.3	1.4	1.4
	P1		*	N.S		**	*
	P2			**			*
HDL (mg/dl)	Mean	46.7	38.4	46.0	47.8	39.9	40.1
	± SE	1.6	1.2	1.6	1.3	1.4	0.93
	P1		**	N.S		**	**
	P2			**			N.S

Each value is the mean of 5 animals ± SE.
N.S: non significant.
*, **: is the significant difference when compared at P< 0.05 and P < 0.01 respectively.
P1: in comparison with control group.
P2: in comparison with fatty liver group.

Table (3): Effect of curcuma on serum total proteins, albumin, globulin and A/G ratio in treated and recovery groups.

		Treated			Protective group		
		Control	Fatty liver	Curcuma	Control	Fatty liver	Curcuma
Total Protein (g/dl)	Mean	7.5	5.7	7.3	7.6	5.3	5.4
	± SE	0.08	0.09	0.1	0.1	0.08	0.07
	P1		**	N.S		**	**
	P2			**			N.S
Albumin (g/dl)	Mean	4.4	3.8	4.1	4.5	3.6	3.5
	± SE	0.1	0.08	0.09	0.1	0.08	0.06
	P1		**	N.S		**	**
	P2			*			N.S
Globulin (g/dl)	Mean	3.1	1.9	3.2	3.1	1.7	1.9
	± SE	0.08	0.08	0.07	0.06	0.08	0.05
	P1		**	N.S		**	**
	P2			**			N.S
A/G ratio	Mean	1.4	2.1	1.3	1.5	2.1	1.9
	± SE	0.07	0.1	0.04	0.05	0.14	0.08
	P1		**	N.S		**	**
	P2			**			N.S

Each value is the mean of 5 animals ± SE.
N.S: non significant.
*, **: is the significant difference when compared at P< 0.05 and P < 0.01 respectively.
P1: in comparison with control group.
P2: in comparison with fatty liver group.

Table (4): Effect of curcuma on serum aspirate amino transferase (AST), alanine amino transferase (ALT), gamma glutamyl transferse (GGT) and lactate dehydrogenase (LDH) in treated and recovery groups.

		Treated			Protective group		
		Control	Fatty liver	Curcuma	Control	Fatty liver	Curcuma
AST (U/ml)	Mean	94.7	124.7	94.1	95.1	126.1	120.2
	± SE	1.1	1.38	1.16	1.02	1.64	1.24
	P1		**	N.S		**	**
	P2			**			*
ALT (U/ml)	Mean	49.3	94.7	55.9	49.6	96.5	92.8
	± SE	1.71	1.55	1.7	1.63	1.56	1.94
	P1		**	N.S		**	**
	P2			**			N.S
GGT (iu/L)	Mean	10.04	21.34	10.3	10.7	21.12	16.04
	± SE	0.33	0.73	0.22	0.26	0.69	0.37
	P1		**	N.S		**	**
	P2			**			**
LDH (U/L)	Mean	179	523	201	185	505	322
	± SE	2.75	3.48	2.77	2.15	4.18	3.14
	P1		**	**		**	**
	P2			**			**

Table (5): Effect of curcuma on serum urea and creatinine in treated and recovery groups.

		Treated			Protective group		
		Control	Fatty liver	Curcuma	Control	Fatty liver	Curcuma
Serum Urea (mg/dl)	Mean	37	203	46	28	204	109
	± SE	2.6	8.6	1.9	1.2	6.3	4.6
	P1		**	N.S		**	**
	P2			**			**
Serum Creatinine (mg/dl)	Mean	0.71	2.1	0.86	0.63	1.7	0.91
	± SE	0.02	0.1	0.06	0.02	0.1	0.05
	P1		**	N.S		**	**
	P2			**			**

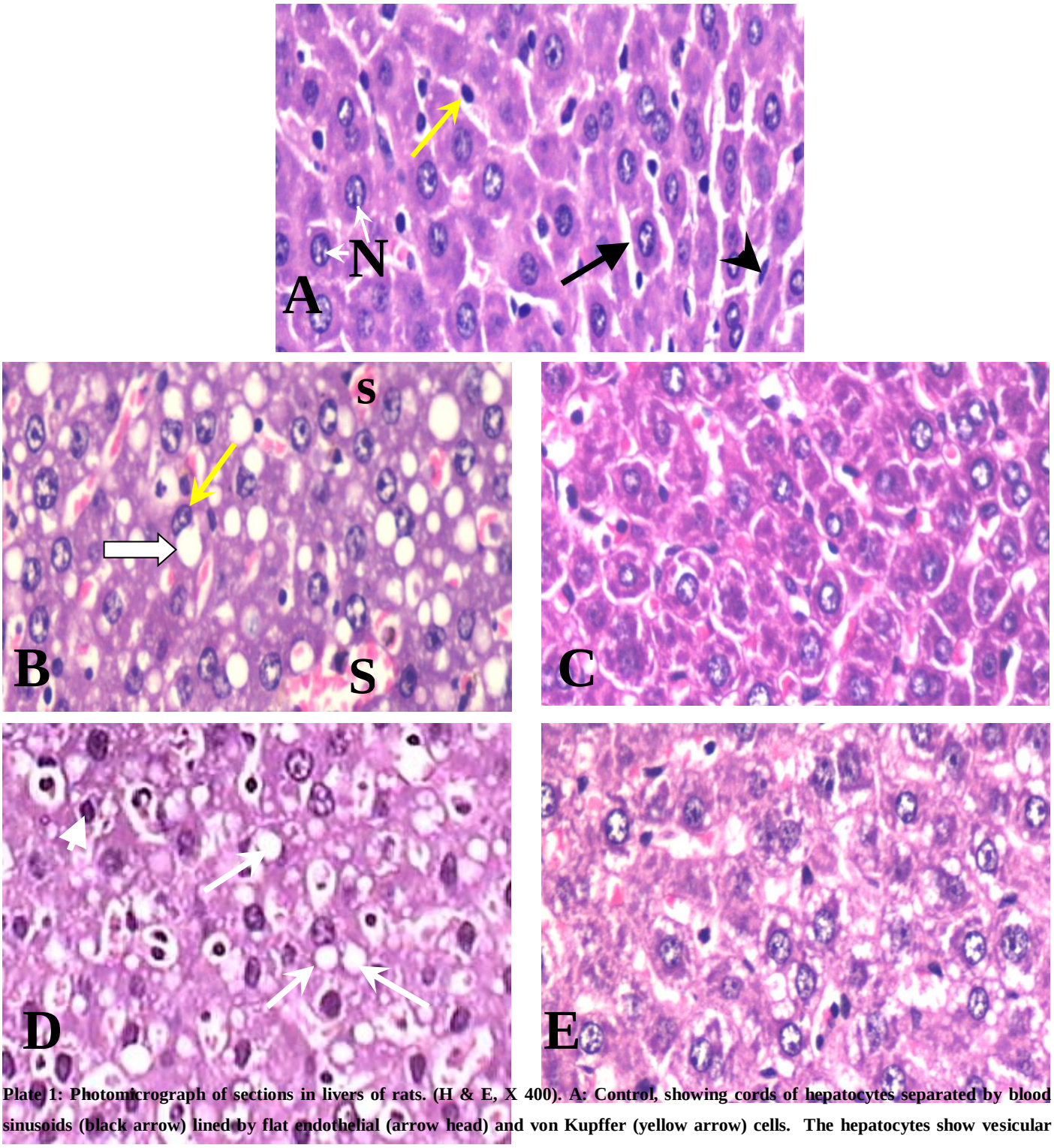

Plate 1: Photomicrograph of sections in livers of rats. (H & E, X 400). A: Control, showing cords of hepatocytes separated by blood sinusoids (black arrow) lined by flat endothelial (arrow head) and von Kupffer (yellow arrow) cells. The hepatocytes show vesicular nuclei (N) and granular cytoplasm. B: After treatment with Oxy tetracycline for three consecutive days, displaying fatty liver as indicated by many fat vacuoles (white arrow), which occupy almost all the cytoplasm of hepatocytes, displacing nucleus (yellow arrow) to periphery. Dilated congested blood sinusoids (S) are also seen. C: After treatment with Oxy tetracycline for three consecutive days and curcuma for one month, showing normal architecture. No vacuoles were detected all over the lobule D: After treatment with Oxy tetracycline for three consecutive days and then recovery after one month, showing many fatty vacuoles (arrows), ballooned hepatocytes and apoptosis (arrow head). E: After treatment with curcuma for 15 days and then injection with oxy tetracycline for three consecutive days, mildly vacuolated granular cytoplasm was apparent.

Discussion

The present study was conducted to evaluate the beneficial effect of curcuma on fatty liver status induced by oxy tetracycline.

Mechanisms by which oxytetracycline induce steatosis include:

a) Inhibition of β oxidation of free fatty liver acids and lipoprotein secretion from the liver (Freneaux *et al*., 1988, Letteron *et al*., 2003; Amarcher and Martin 1997). b) Increase cholesterol and triglyceride biosynthesis. c) Increase in free radical levels and decrease the antioxidant enzyme levels (Asha *et al*., 2007).

In the present study results, oxytetracycline induced hyperglycemia in fatty liver rats treating with curcuma turning glucose level back to normal value. The improvement in glycemic status may be due to anti-inflammatory effects of curcuma where it leads to a decrease in NF-KB (nuclear factor kappa-light-chain-enhancer of activated B cells) activity in liver tissue and a decrease in macrophage infiltration, which can explain the anti-diabetogenic effects seen in adipose tissue following the ingestion of curcumin (Yamuchi *et al*., 2001).

The results revealed that, the hyperlipidemic effect of oxytetracycline ameliorated with the treatment of rats with curcuma. Curcuma may lower lipid peroxidation by maintaining the activity of antioxidant enzymes (superoxide dismutase, catalase and glutathione peroxidase). These enzymes play an important role in the regulation of lipid peroxidation (Harris, 1992).

Curcuma can scavenge oxygen free radicals such as superoxide anions and hydroxyl radicals which play an important role in inhibition of lipid peroxidation (Choudhary *et al*., 1999).

Therefore, curcuma can lower lipid peroxidation by influencing a number of important factors that regulate lipid oxidation. Also fat is more easily digested with the use of curcuma because it stimulates the flow of bile (Pulla and Lokesh, 1994). Arafa (2005) reported that, the effect of curcumin on cholesterol could be due to an effect on cholesterol absorption, degradation or elimination, but not due to an antioxidant mechanism. Also curcumin reveals a messaging molecule that communicates with genes in liver cells, directing them to increase the production of mRNA that direct the creation of receptors for LDL cholesterol. With more LDL receptors, liver cells are able to clear more LDL cholesterol from the body (Jain *et al*., 2006).

In this study, oxytetracycline injection of rats resulted in high significant increase in activities of serum ALT, AST, GGT and LDH, this is in agreement with Asha *et al*. (2007) who reported that, this significant increase is due to rise in free radical levels and decrease in the antioxidant enzyme levels. However, rats which were treated with curcuma after induction of fatty liver showed a decrease in these activities. This meant that, curcuma ameliorated the oxidative hazardous effect of oxytetracycline and prevented the fibrotic processes and subsequent liver damage induced by oxytetracycline administration (Eun- Jeon *et al*., 2000).

The pretreatment with curcuma before induction of fatty liver reduced the levels of total lipids, cholesterol, triglycerides and LDL and increased HDL but these levels remained higher than normal values

Group of rats which were treated with curcuma showed alleviation of the effect of oxy tetracycline on liver enzymes in addition to promotion of the regeneration of hepatic cells, as shown by histological examination. The results of biochemical analysis were in the same direction. This may be attributed to the ability of curcuma to promote the hepatocyte membrane integrity ((Shabon, 2008).

Administration of curcuma prior to fatty liver induction ameliorated oxy tetracycline effects on liver enzymes but not returned back to the normal values. This ability of curcuma to protect the liver from inflammatory condition may be due to its anti-inflammatory effect through inhibition of expression of cyclo-oxygenase-2 (Shakibaei *et al*., 2007), or by elevation the antioxidant agents in the body, or the scavenging reactive oxygen species and nitrogen oxide and enhancing antioxidant defense by increasing reduced glutathione level (Kaur *et al*., 2006). The protective effect of curcuma was also proven by histopathological examination of the liver.

Serum total protein, albumin and globulin were decreased in fatty liver group, while A/G was increased. This decrease could be related to hepatic dysfunction and decreased protein synthesis. Also it may be due to either damage

of vital biological processes or to changes in permeability of liver, kidney and other tissue cells leading to leakage of protein *via* the kidney (Roushdy *et al*., 1989).

Administration of curcuma after fatty liver induction was tolerated the hypoproteinemic effect of oxy tetracycline due to its beneficial effect on liver and kidney functions. While administration of curcuma before fatty liver induction improves protein profile but didn't return it back to normal values. Treating rats with oxy tetracycline caused kidney dysfunction which appeared through high increase in serum urea and creatinine, while after treating rats with curcuma, renal function markers returned back to normal values which may be due to the protective effect of curcumin against renal injury by suppressing oxidative stress, increasing kidney GSH content and gluthation peroxidase activity (Venkatesan, 2000).

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تأثير الكركم على الكبد الدهنى المستحدث بواسطة أكسى تتراسيكلين في الجرذان

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الكبد الدهني يعد الآن من أكثر الأمراض شيوعاً فى مصر وخصوصاً فى متوسطي الأعمار وأيضاً كبار السن ، ويفسر سبب الإصابة بهذا المرض بأنه نتاج طبيعي لطبيعة الطعام المصري المليئ بالشحوم والدهنيات وخصوصاً مع الميل للكسل والخمول وعدم القيام بأي نوع من أنواع التمرينات الرياضية المهمة .
ولقد ظهر فى الآونة الأخيرة إستخدام الأعشاب والنباتات الطبية للعلاج وتحسين بعض الحالات المرضية وقد استهدفت هذه الدراسة إيضاح الدور الوقائى لنبات الكركم ضد الأخطار الناتجة عن الكبد الدهنى على بعض المعايير البيو كيميائية والمظاهر الهستولوجية وقسمت هذه الحيوانات إلى مجموعتين :

1-مجموعة أعطيت الكركم بعد إحداث الكبد الدهنى وقسمت إلى ثلاثة مجموعات :

أ-مجموعة ضابطة .
ب- مجموعة مصابة بالكبد الدهنى (120مجم/كجم أكسى تتراسيكلين) لمدة 3 أيام متتالية ثم تم ذبحها بعد 30 يوما.(مجموعة إستشفائية)
ج-مجموعة مصابة بالكبد الدهنى لمدة 3 أيام ثم عولجت بالكركم (0.4 % من الغذاء) لمدة 30 يوما ثم ذبحت.

2-مجموعة أعطيت الكركم بعد إحداث الكبد الدهنى وقسمت إلى ثلاثة مجموعات :

أ-مجموعة ضابطة .
ب- مجموعة مصابة بالكبد الدهنى (120مجم/كجم أكسى تتراسيكلين) لمدة 3 أيام متتالية ثم تم ذبحها .
ج-مجموعة أعطيت الكركم (0.4 % من الغذاء) لمدة 15 يوما ثم أصيبت بالكبد الدهنى لمدة 3 أيام ثم ذبحت.

ولقد أوضحت نتائج هذا البحث أن معاملة الجرذان بالأكسى تتراسيكلين لمدة 3 أيام متتالية له آثار سلبية عديدة تتمثل فى زيادة معدل كلا من: نسبة السكر فى الدم- وظائف الكلى- وظائف الكبد وإنزيمات القلب- البروتين الكلى- الألبيومين ومجموعة الدهون والكوليسترول والدهون الثلاثية بالدم ، كما تشتمل أيضا على النقص الشديد فى HDL-Cholesterol وصاحب كل ذلك وجود عدد كبير من التغيرات النسيجية فى كبد الجرذان البيضاء، أما المعاملة بنبات الكركم فقد أدت إلى ظهور تحسن ملحوظ فى المعايير البيوكيميائية والهستولوجية، وقد لوحظ أن المجموعة الأولى قد أعطت نتائج أفضل من المجموعة الثانية ولهذا ينصح باستخدام الكركم فى طعام مرضى الكبد الدهنى أومن لهم تاريخ عائلى لهذا المرض .