The Egyptian Journal of Hospital Medicine (Oct 2012) Vol., 49: 539– 554

EFFECT OF CAMEL MILK ON ALLOXAN-INDUCED DIABETIC RATS

Eman G. E. Helal*, Samia M. Abd-Elwahab, and Anwaar Alkamel Mohammad *****
Departments of Zoology –Faculty of Science-Al-Azhar University (Physiology* and Histology **)
and Department of Medical Science- Faculty of dentistry Sinai University***

Abstract

Back ground: Diabetes is a chronic metabolic disease which affects large number of population all over the world. Such disease is associated with many complications which may leads finally to patient's mortality. Camel milk supplementation reduces the insulin requirement in Type I diabetic patients. So this study was planned to evaluate the effect of camel milk as hypoglycemic agent.

Material and method: Thirty male adult albino rats were used to investigate the effect of camel milk (CM) treating diabetic rats. Rats were divided into three equal groups, control, diabetic non treated and diabetic CM treated groups. After thirty days of treatment all rats of each group were sacrificed. The body weight of each rat was determined at the beginning and the end of each period. Blood glucose, serum insulin, lipid and protein profiles, liver and kidney functions, blood picture and liver glycogen were determined for each rat at the end of each period. Pancreatic samples were obtained and processed for microscopic and quantitative evaluation after staining the prepared sections with both Heamatoxylin and eosin as well as special stain for demonstration of the different pancreatic cells in the Islet of Langerhans.

Results: The obtained results showed that the induced diabetes was diagnosed by laboratory assessment to body weight loss, hyperglycemia, and hypoinsulinemia, significant increase in liver and kidney functions, lipid and protein profiles and decreased liver glycogen content. While, CM treatment led to a significant improvement in all these parameter except liver function. Microscopically there was definite vaculation, degeneration, karyolysis and pyknosis of beta pancreatic cells in diabetic group while the other pancreatic cells were not affected (alpha and delta cells). The use of CM treatment of this study greatly improves such cellular changes.

Conclusion: it was recommended that the use of the CM as a hypoglycemic agent may be of good results besides repeating such study with the use of variable doses may be helpful in better evaluation for the required doses.

Key words: Alloxan- hypoglycemia- Diabetic- Pancreas.

Introduction

The incidences of diabetes mellitus have been increased worldwide (Onkamo et al., 1999). Prevention and early treatment are important because diabetes interrupts normal development in children and carries the threat of severe complication in the more active period of life (Dahlquist, 1999). Its primary treatment is insulin replacement, however, at present, entire physiological insulin replacement cannot be achieved in clinical practice and metabolic disturbances cannot be normalized. Oral therapy is still the best treatment but in some countries like ours in

Egypt, needle phobia and cost of treatment forces diabetic patients to adopt alternative treatments. In this connection we have heard many folklore stories which describe the use of camel milk in treatment of diabetes mellitus. There is also an account in memories of Emperor Jahangir (1579 -1627 AD) about usefulness and acceptability of CM (Rogers, 1989).

Camel milk is known for its medicinal properties, which are widely exploited for human health, as in several countries from theex-Soviet Union (Kenzhebulat et al., 2000) and developing countries (Mal et al., 2006). Camel milk is considered to have anti-cancer (Magjeed, 2005), hypo-allergic (Shabo et al., 2005) and anti-diabetic properties (Agrawal et al., 2003).

It is found that one of the camel milk proteins has many characteristics similar to insulin (Beg et al, 1986b) and it does not form coagulum in acidic environment (Wangoh, 1993). This lack of coagulum formation allows the camel milk to pass rapidly through stomach together with the specific insulin-like protein and remains available for absorption in the intestine. Radioimmunoassay of camel milk has revealed high concentration of insulin i.e. 52 units/liter (Singh, 2001). The concentration of insulin in human milk is also significantly higher (60.23 ± 41.05 micro u/ml) (Shehadeh et al., 2001), but probably because of coagulation in stomach it is not available for absorption in the intestine.

Camel milk consumption and lifestyle have definite influence on prevalence of diabetes. Hence, adopting such life pattern may play protective role in preventing diabetes to some extent (Agrawal et al., 2007). So that, in the present study aims to clarify the role of the CM in treating diabetic rats.

Material and methods

Material:

A-Animals:

Thirty adult male albino rats of local strain with body weight (b. wt.) ranging between (120-140 gm) were divided into three equal groups:

- Group I (control group),
- Group II (diabetic group), were given s.c alloxan (120 mg / kg b. wt.) in order to induce diabetes mellitus.
- Group III (CM treated group), were given alloxan to induce diabetes then given camel milk (0.1 ml/100 gm b. wt.) orally once daily for one month.

B-Drugs and chemicals:

Alloxan (powder from B.D.H chemical LTD, England) dissolved in acetate buffer (pH 5.5) prepared immediately before use.Camel milk, brought from Sinai Peninsula in Egypt.

Methods:

- **Induction of diabetes mellitus**: By giving sub continues freshly prepared alloxan solution 120 mg / kg dissolved in 0.5ml acetate buffer (pH 5.5) to an overnight fasting of the animals according to (Malaisse, 1982). After 48 hours blood

glucose level was determined by glocometer. Rats with blood glucose level ranging from 180 to 250 were considered diabetic (Dunn et al., 1943).

- **Camel Milk tereatment:** Diabetic rats treated with camel milk (0.1 ml/ 100 gm b.wt. given orally by oral tube).

- **Preparation of serum and determ-ination of various parameters:** At the end of the experiment, all animals were weighted and blood samples collected from the orbital plexus using heparinized capillary tubes according to the method of Schermer (1967). Blood was collected either on EDTA for hematological studies or in clean centrifuge tubes to separate the serum by centrifugation for 10 min. at 5000 rpm, and the supernatant serum was immediately separated for biochemical analysis. Animals were then killed and samples were collected for histological examination. 1gm of liver was taken for glycogen determination (Joseph, 1955). Samples from the pancreases were also taken, stained with Hematoxylin and Eosin (HX & E) and modified aldhyde fuchsin (Halami, 1952) for histological study.

Student (t) test was used to compare between groups, P< 0.05 was considered significant (Snedecor and Cochron, 1980).

Results

As shown in Tables (1&2), alloxan led to significant decrease in body weight, liver glycogen and serum insulin with significant increase in blood glucose level (P< 0.01) as compared to the control group. CM treatment led to a significant decrease in liver glycogen and serum insulin levels when compared to the control group, while glucose level and HOMA-IR were significantly increased (P< 0.01) but less than the percentage ratio of diabetic group and body weight showed no significant change.

As indicated in table (3), there was a high significant increase in serum T-cholesterol, Triglyceride, LDL-C and vLDL-C and significant (P<0.01) decrease in HDL-C and HDL-C/LDL-C in diabetic group when compared with the control group. In Camel-milk treated group, no significant changes in these parameters were recorded when compared with control group except (HDL-C/LDL-C), which showed a significant increase (P<0.05).

As shown in Table (4), a significant decrease (P<0.01) in total protein, albumin and A/G ratio, and a significant increase (P<0.05) in globulin level were detected in diabetic group when compared to the control group. On the other hand, Camel milk treatment ameliorated these disorders to the normal value.

As indicated in Tables (5and 6), the current study showed a highly significant increase (P<0.01) in ALT and AST levels in the diabetic and camel milk treated groups when compared to the control one. At the same time, serum urea showed a high significant increase in both diabetic

and camel-milk treated groups. Creatinine levels showed no significant change in camel-milk treated group but significant increase ($P<0.05$) in the diabetic group.

In Table (7), the results showed no significant change in RBCs count and Hct in diabetic rats, but significant decrease ($P<0.05$) in the camel milk treated rats. WBCs and lymphocyte counts showed no significant change between treated and diabetic rats, except lymphocyte which showed highly significant increase in diabetic group.

Histological examination of the pancreatic tissues from the control group stained with Hx & E showed normal pancreatic islets. Modified aldhyde fuchsin stain showed the three main types of cells of the pancreatic islets (alpha, beta and delta cells). B-cells were more abundant, occupying the central portion of the islet and contained numerous granules. Alpha and delta cells occupied the periphery of the islets. Delta cells were usually adjacent to alpha cells and were somewhat larger in size. Alpha cells were granular and polygonal with central spherical nuclei (Plate 1&2). Alloxan administration led to shrinkage of the pancreatic islets. The cytoplasm of the cells was vacuolated with pyknotic nuclei, and many necrotic cells were seen and others showed hydropic degeneration (Plate 1&2). Pancreatic tissues from CM treated rats showed nearly normal architecture of the pancreatic islets. Most of the cytoplasm became granulated, less vacuoles appeared in β-cells and nuclei became normal (Plate 1&2). As shown in table (8), a significant decrease ($p<0.01$) in number of A-cell, B-cell and D-cell of the islets in diabetic group. While, CM treated group showed no significant change in both A-cell and D-cell number but showed significant decrease ($p<0.01$) in B-cell number.

Table (1) Percentage of body weight change in control, diabetic and camel milk treated male albino rats.

	Control	**Diabetic**	**Camel milk**
Body weight change (%)	21.89±0.93	13.26**±1.03	23.6 $^{n.s}$±0.86
%		-37.87 %	+7.81%

Data expressed as:
Mean ± standard error,
% = percentage of change,
**= highly significant,
n.s. = non significant,
(+) = Increased from control,
(-) = Decreased from control.

Table (2) Serum glucose (mg/dl) and insulin (pg/ml) levels, glycogen content and HOMA test in the liver (mg/dl) in normal, diabetic and camel milk treatment on male albino rats.

	Control	Diabetic	Camel milk
Glucose (mg/dl)	121.8±1.11	286.4** ±2.35	187.4** ±1.12
%		+135.14%	+53.86%
Insulin (µu/l)	4.06±0.04	3.01** ±0.04	2.96** ±0.04
%		-25.86 %	-27.09 %
Glycogen content (mg/dl)	10.78±0.24	2.45** ±0.16	4.83** ±0.28
%		-77.27 %	-55.2%
HOMA-IR	1.22±0.01	2.13** ±0.03	1.37** ±0.02
%		+74.59 %	+12.30%

Data expressed as:
Mean ± standard error,
% = percentage of change,
**= highly significant,
n.s. = non significant,
(+) = Increased from control,
(-) = Decreased from control.

Table (3) Serum cholesterol (mg/dl), triglycerides (mg/dl), HDL-cholesterol (mg/dl), LDL-cholesterol (mg/dl) and vLDL-cholesterol (mg/dl) levels in normal, diabetic and camel milk treatment on male albino rats.

	Control	Diabetic	Camel milk
T. Lipid	303.8±2.08	436.2**±1.35	290.8**±2.29
%		43.58%	-4.28%
T. cholesterol	109.4±1.96	199 **±1.87	104 n.s±1.87
%		81.9%	-4.94%
Triglycerides	85±1.37	92.2**±1.02	83 n.s±1.55
%		8.47%	-2.35 %
(HDL-C)	25.2±0.73	39.2**±0.96	27.6 n.s±0.93
%		55.56%	+9.52 %
(LDL-C)	67.2±2.34	77.36**±1.61	61.6 n.s±2.12
%		15.12%	-8.33 %
HDL/ LDL	0.38± 0.02	0.51 **± 0.02	0.47*± 0.03
%		34.21%	+23.69%
(VLDL-C)	17+0.28	28.44**±0.2	16.6 n.s±0.31
%		67.29%	-2.35 %

Data expressed as:
Mean + standard error,
% = percentage of change,
****= highly significant,**
n.s. = non significant,
(+) = Increased from control,
(-) = Decreased from control.

Table (4) Serum total protein (g/dl), albumin (g/dl), globulin (g/dl) concentration and A/G ratio in normal, diabetic and camel milk treated male albino rats.

	Control	Diabetic	Camel milk
Total protein	6.34+0.11	5.88*+0.09	6.22 ns+0.06
%		-7.26 %	-1.89%
Albumin	3.54+0.13	2.84**+0.07	3.26 ns+0.12
%		-19.77 %	-7.91 %
Globulin	2.8+0.19	3.04 ns+0.07	2.98 ns+0.12
%		+8.57 %	+6.43 %
A/G ratio	1.29+0.13	0.93*+0.04	1.03ns + 0.05
%		-27.91 %	-20.15 %

Data expressed as:
Mean ± standard error,
% = percentage of change,
**= highly significant,
n.s. = non significant,
(+) = Increased from control,
(-) = Decreased from control.

Table (5) Serum ALT and AST activities in control, diabetic and camel milk treatment on male albino rats.

	Control	Diabetic	Camel milk
ALT (U/ml)	7.6+0.51	9.8**+0.37	9.8**+0.37
%		+28.9 %	+28.94%
AST (U/ml)	6.6+0.93	11.2**+0.37	11.4**+0.4
%		+ 69.69 %	+72.72 %

Data expressed as:
mean ± standard error,
% = percentage of change,
**= highly significant,
n.s. = non significant,
(+) = Increased from control,
(-) = Decreased from control,

Table (6) Creatinine and Urea activities in control, diabetic and camel milk treatment on male albino rats.

	Control	Diabetic	Camel milk
Creatinine (mg/dl)	0.62+0.04	0.82**+0.03	0.68 n.s+0.04
%		+32.25 %	+9.67%
Urea(mg/dl)	18.4+0.5	28.4**+0.5	22.6**+0.9
%		+ 54.35 %	+22.83 %

Data expressed as:
Mean ± standard error,
% = percentage of change,
*= highly significant,
n.s. = non significant,
(+) = Increased from control,
(-) = Decreased from control,

Table (7) RBCs and WBCs count, Hct value and percentage of lymphocyte and granulocyte in control, diabetic and camel milk treatment on male albino rats.

	Control	Diabetic	Camel milk
RBCs count	8.94+0.29	8.35 n.s+0.26	7.92*+0.23
%		-6.59 %	-11.41%
Hct	42.92+1.52	44.2n.s+1.15	37.19*+1.19
%		+ 2.98 %	-13.35%
WBCs	9+0.35	8.06n.s+0.4	8.58n.s+0.38
%		-10.45 %	-4.67 %
Lymph	90.02+1.42	82.58**+1.62	85.46n.s+1.72
%		-8.26 %	-5.07 %

Data expressed as:
Mean ± standard error,
% = percentage of change,
**= highly significant,
n.s. = non significant,
(+) = Increased from control,
(-) = Decreased from control.

Table (8) Number of A-cell, B-cell and D-cell in control, diabetic and camel milk treatment on male albino rats.

	Control	Diabetic	Camel milk
A-cell count	3.67+0.33	1.67**+0.33	3 n.s+0.58
%		-54.5 %	-18.25%
B-cell count	91.67+0.88	57.33**+0.88	64.33**+0.88
%		-37.46 %	-29.82%
D-cell count	4.67+0.33	2.33**+0.33	4.67n.s+0.33
%		-50.1 %	0 %

Data expressed as:
Mean ± standard error,
% = percentage of change,
**= highly significant,
n.s. = non significant,
(+) = Increased from control,
(-) = Decreased from control.

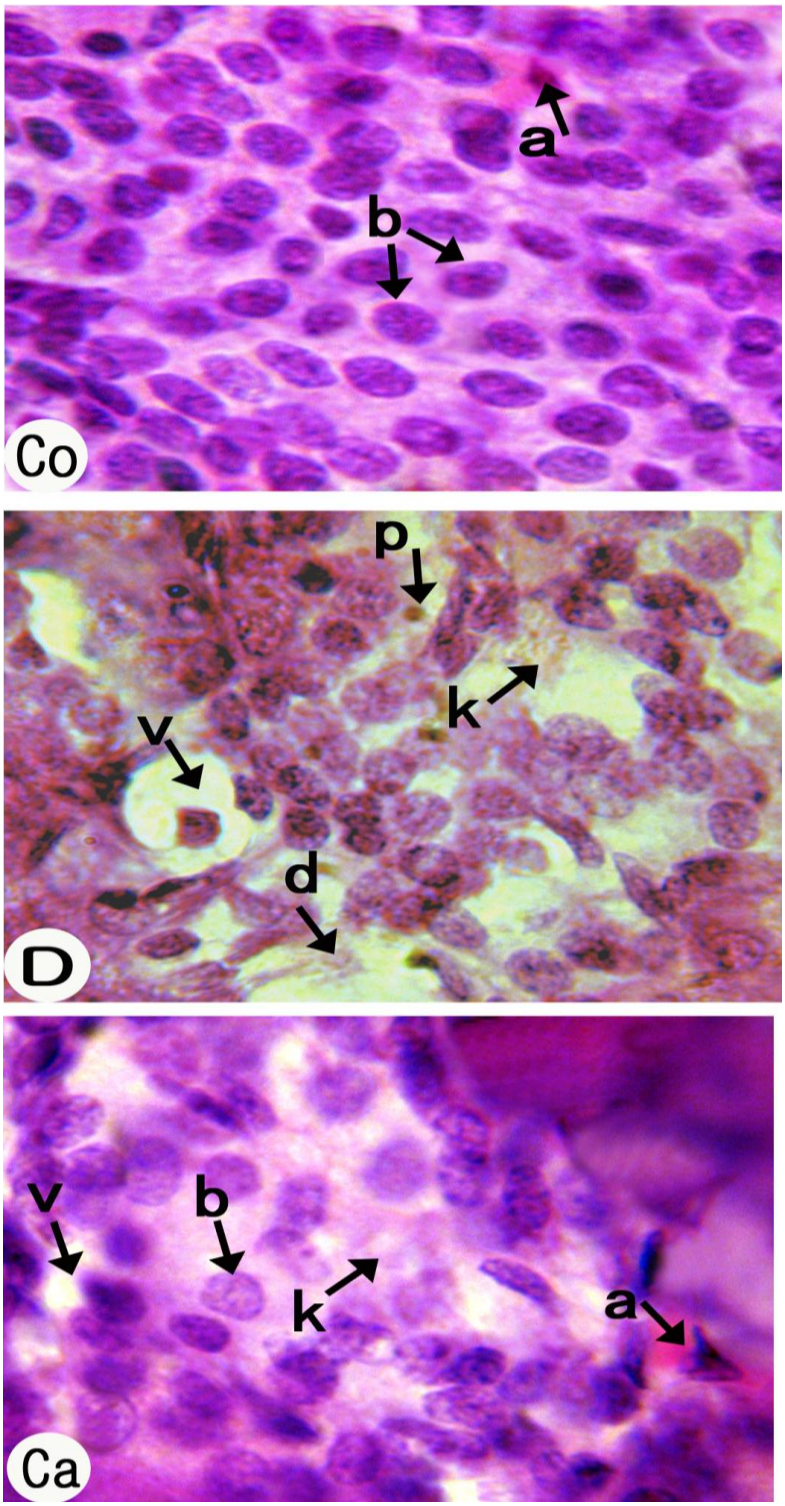

Plate (1): **(Hx & E X 1000)**

Histopathological changes in the pancreas of Control (Co.), Diabetic (D) and Camel milk (Ca) treated rats

Where,

a; normal alpha cell
b; normal beta cell
v; vacuolated beta cell
d; degenerated beta cell
P; pyknotic beta cell
k; beta cell karyorrhexis

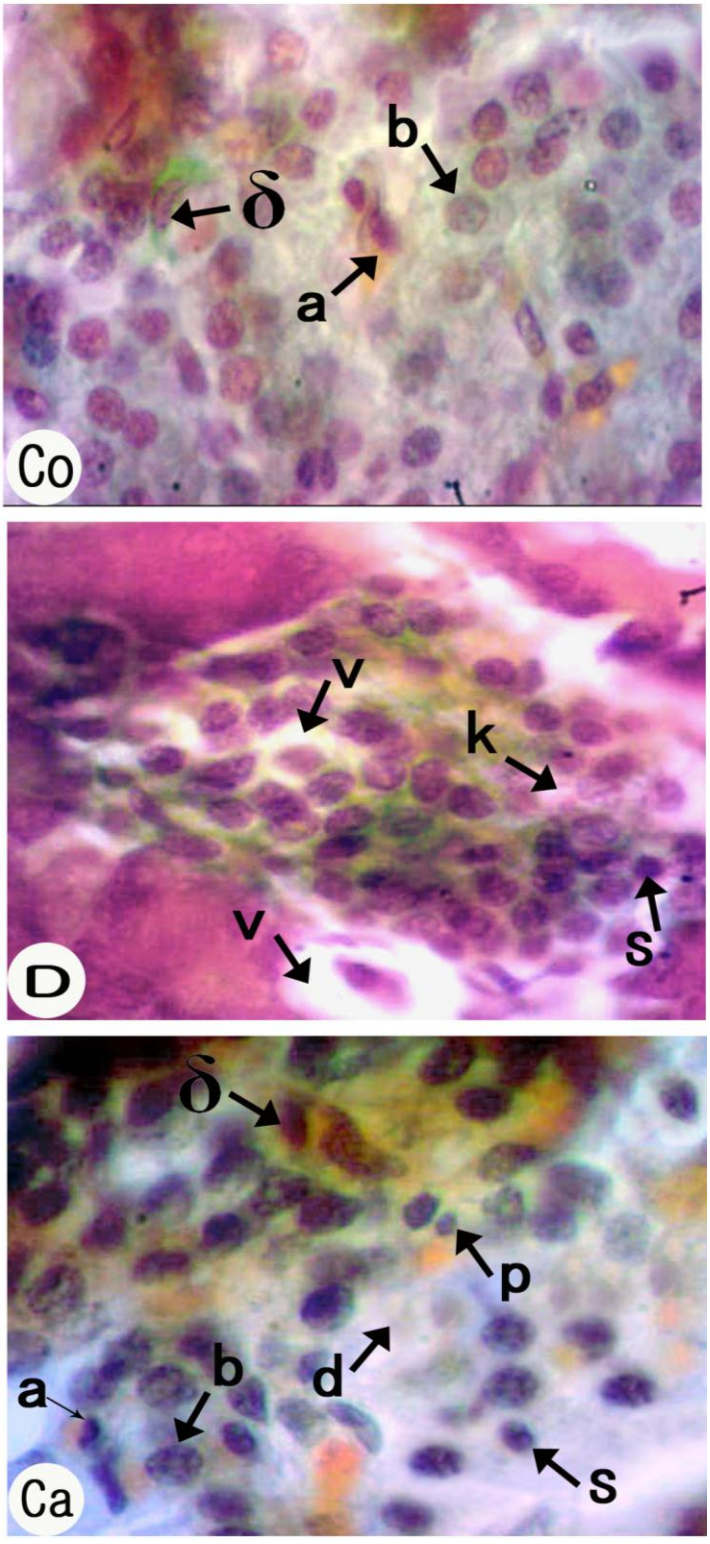

Plate (2): **(Modified aldehyde Fuchsin stain X 1000)**

Histopathological changes in the pancreas of Control (Co.), Diabetic (D) and Camel milk (Ca) treated rats

Where,

a; normal alpha cell
b; normal beta cell
k; beta cell karyorrhexis
P; pyknotic beta cell
v; vacuolated beta cell
δ; normal delta cell
d; degenerated beta cell
S; shrinkage of nuclear beta cell

Discussion

The diabetic patients need alternative therapies to control all the pathological aspects of diabetes and the high cost and poor availability of current therapies in developing countries (Ashraf et al., 2009).

Camel milk is considered to have anti-diabetic properties (Agrawal et al., 2003). It's therefore speculated that CM could help supplement insulin shots for Type I diabetics, in addition to its clear potential for diabetes prevention (Agrawal et al., 2009). So, this study is a step to evaluate and follow up the effect of CM as a hypoglycemic agent or has a role in amelioration the insulin resistance.

The present results, revealed loss in body weight gain in diabetic rats with compared with control rats. This loss may be due to an excessive amount of glucose and an insufficient amount of insulin in the bloodstream. This triggers the release of triglycerides form adipose tissue and catabolism of amino acids in muscle tissue. A loss of both fat and lean mass, leading to a significant reduction in total body weight gain and may be a result to fluid loss especially in untreated diabetic patient. (Morley et al., 2006)

We observed a significant improvement in body weight gain after camel milk treatment. The positive effects in weight gain may be because of good nutritional value of camel milk. This important may be due to stimulation effect of CM on most aspects of carbohydrate metabolism, including rapid up take of glucose by the cells, increased rate of absorption from the gastrointestinal tract and even increased insulin secretion with its resultant secondary effects on carbohydrate metabolism. (Guyton and Hall, 2000 and Stamfield, 2011), or anabolic effect of camel milk and strengthening the gastrointestinal tract by increasing both the rate of secretion of the digestive juices and the motility of the gastrointestinal tract (Guyton and Hall, 2000)

Serve hyperglycemia in diabetic rats recorded in the present work can be considered as a direct reflex to the marked hypoinsulinemia caused by the selective destructive cytotoxic effect of alloxan on the β cells of the pancreas which has a direct effect on their membrane permeability by causing failure of ionic pumps and increased cell sizes. It also inhibits intercellular energy generation; insulin secretion causes sudden activation of quiescent cell for a high level of protein synthesis and produced rapid and massive beta cell death which leading to a decrement in β cells number (Majno and Joris, 1999)

The destructive effect of alloxan on β-cells may be also attributed to the ability to inhibit enzymes of tricarboxylic acid cycle and Ca+2 dependants dehydrogenises in β-cell mitochondrion, causing ATP deficiency, cessation of insulin production and cell necrosis (Shafrir, 2003).

The results also showed β-cells with vacuolated cytoplasm in the diabetic group. Vacuolation of the islet in the most

prominent with lesion associated with functional islet abnormality and development of hyperglycemia (Bolaffi et al., 1986 and Kessler et al., 1999). Also, the vaculation may be due to the diabetogenic action of alloxan which induced highly reactive oxygen radicals, which one cytotoxic to β-cells (Fischer and Homburger, 1980).

According to Yamaoto et al. (1981) and Ronald (1988) the fragmentation of nuclear DNA of pancreatic β- cells seems to be important for the development of diabetes and supposed to be resulted from the accumulation of superoxide or hydroxyl radicals in the β-cells.

CM treatment led to a significant increase in glucose level ($p<0.01$) but less than the percentage ratio of diabetic group. This action is due to the presence of insulin or insulin like protein in it. Its therapeutic efficacy may be due to lack of coagulum formation of milk in acidic media. Pozzilli et al. (2000) indicate that addition of 5 mg of oral insulin does not modify the course of the disease in the first year after diagnosis and probably does not statistically affect the humeral immune response against insulin. It is important to note that a certain level of scientific testing on camel milk has been already attempted and documented, particularly, insulin levels in camel milk and this scientific wisdom can be a remarkable achievement for diabetic patients (Agrawal et al. 2002). Agrawal et al. (2005) found that one of the camel milk proteins has many characteristics similar to insulin and it does not form coagulum in acidic environment. This lack of coagulum formation allows the camel milk to pass rapidly through the stomach together with the specific insulin like protein or insulin and remains available for absorption in intestine, the important observation of this study was the significant reduction in insulin doses to obtain glycemic control at the end of 1 year in patients taking camel milk. It is suggested that camel milk has antidiabetic activity possibly because of: insulin like activity, regulatory and immune-modulatory function of on β-cells, there is a good amount of lysozyme, lactoferrin, lactoperoxidase, immunoglobulin G and secretary immunoglobulin A in camel milk. It is found that amino acid sequence of some of the camel milk proteins is rich in half-cystine, which has superficial similarity with insulin family of peptides.

The elevation of serum AST and ALT activity in the present work may be attributed to the excessive release of such enzymes from the damaged liver cells into the blood circulation. Where, there is an inverse relationship between the liver activity and the level of enzymes in serum (Awadallah and El-Dessouky, 1977). And may be consistent with their greater need for gluconeogenesis substrates or may reflect damage of the hepatic cells due to hepatotoxic effect of alloxan (Helal, 2000 and Youssef and Osman, 2002).

The present study showed that serum total proteins, albumin concentrations and

A/G ratio were significant decrease in alloxan diabetic rats, while globulin concentration showed no significant change when compared with those of non-diabetic ones. Helal (2000) and Abdel-Moneim (2002) found marked decrease in serum total proteins and albumin in diabetic animals. This decrease in total serum protein content of diabetic rats may be due to the decreased amino acids uptake (Garber, 1980) greatly decreased concentration of a variety of essential amino acids (Brosnan et al., 1984), increased conversion rate of glycogenic amino acids to CO2 and H2O (Mortimore and Mandon, 1970), reduction in protein synthesis which in turn may be due to a decrease in the amount and availability of mRNA (Peavy et al., 1985 and Wool et al., 1986) and a reduction in ribosomal protein synthesis as a result of insulin deficiency (Jefferson et al., 1983).

Otherwise, treatment of alloxan diabetic rats with the CM produced no significant change in serum total proteins, albumin and globulin concentrations and A/G when compared with control group. This improvement in proteins profiles is in harmony with increased serum insulin level by treatment; this explanation was in agreement with Flaim et al. (1985) who showed that the decrease of serum total proteins and albumin in diabetic animals was restored to control rates by insulin treatment. Insulin injection accelerates amino acids transport through uptake of amino acids by cells (Werner, 1983) and angmenting incorporation of certain amino acids into proteins (Granner, 1988).

The present results elucidated that total lipids, triglycerides, total cholesterol, LDL-cholesterol and vLDL-cholesterol were increased significantly in serum of diabetic rats as compared with nondiabetic ones. In agreement with these results, Battell et al. (1998), and Abdel-Moneim et al. (2002) found marked increase of serum triglycerides, cholesterol and LDL-cholesterol levels in diabetic animals. This defective of removal was found to be due to the decrease in lipoprotein lipase (LPL) activity secondary to insulin deficiency (Minnich and Zilversmit, 1989).

The elevated level of serum triglycerides in diabetic animals of the present study may be attributed to decreased clearance and increased production of the major transporters of endogenously synthesized triglycerides (Betteridge, 1986; Howord, 1987 and Rawi et al., 1998). Also, the expansion of cholesterol pool in diabetes might be explained by a higher input into system through an acceleration of intestinal cholesterol synthesis (Feingold et al., 1985; O'Meara et al., 1990 a and Mathe, 1995) or an increment of the rate of intestinal cholesterol absorption (Nervi et al., 1974 ; Feingold et al., 1985 and Mathe, 1995.LDL-cholesterol in serum of diabetic rats showed a significant increase. This abnormality certainly plays a role in the increased risk of cardiovascular disease. Increased LDL-cholesterol may be due to

overproduction of v LDL by the liver or decreased removal of vLDL and LDL from the circulation (Tsustsumi et al., 1995).

HDL-cholesterol concentration showed a very highly significant decrease after induction of diabetes by alloxan. This result agrees well with that of Lassko et al. (1986) and Osman and Kandil (1991) who demonstrated marked decrease of HDL-cholesterol in serum of IDDM patients and alloxan diabetic rats

Otherwise, alloxan diabetic rats treated with the C.M showed a decline in the total lipids as well as cholesterol and triglyceride levels when compared with percentage of diabetic rats. These observations indicate that the treatment with C.M were ameliorated these toxic effects generally and turn back all lipids profile to normal values (Levy, 1977).

The significant increase of serum urea level may be related to the impairment of renal function following congestive heart failure. Varley (1976) decided that serum Creatinine often raise in type 2 diabetes due to renal arterial disease and/or cardiac failure rather than to diabetic nephropathy (Guyton and Hall, 2000). And also, this may be resulted from failure of the body to excrete the metabolic end products of proteins (Guyton and Hall, 2000). Where, proteins metabolic rate increased in diabetic as a result of gluconeogenesis increasing rate.

The improvement of serum urea and Creatinine levels in rats treated with CM may be reflected its effect on improved kidney function and stop the destructive effect of alloxan, which in turn may decrease the excessive loss of albumin in urine of diabetic rats.

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تأثير لبن الجمال على الجرذان المصابة بالسكر التجريبي

ايمان جمال الدين عزت هلال* ، سامية محمد عبد الوهاب ** ، أنوار الكامل محمد ***

قسم علم الحيوان- كلية العلوم- جامعة الأزهر(فسيولوجي*وهستولوجي**) وقسم العلوم الطبيه بكليه طب الاسنان –جامعه سيناء***

يعتبر مرض السكري من الأمراض الأيضية المزمنة والذي يصيب ويؤثر في اعداد كبيرة من البشر في انحاء العالم المختلفة. ويصاحب هذا المرض مضاعفات كثيرة والتي قد تؤدي في النهاية لموت المريض . وقد اظهرت بعض الدراسات ان استعمال لبن الجمال كمكمل او كإضافة في الغذاء يقلل من احتياج مرضي السكر من نوع الإنسولين، ولهذا كان الغرض من تصميم هذة الدراسة هو لتقييم تأثير لبن الجمال كعامل مخفض لمستوى السكر في الدم.

و فى هذه الدراسة قد تم استعمال ثلاثون من الجرذان الذكور البالغة لدراسة تأثير لبن الجمال على الفئران المصابة بالسكر، وتم توزيع هذة الجرذان الى ثلاثة مجموعات متساوية العدد: مجموعة ضابطه(Control) ومجموعة مصابة بالسكر ولا تتلقى علاج، ومجموعة مصابة وتعامل بلبن الجمال، وتم وزن كل فأر في بداية التجربة وايضا في نهاية التجربة اى بعد ثلاثون يوما من المعاملة .

وفي نهاية الثلاثون يوما من المعاملة تم ذبح جميع الجرذان في كل المجموعات وتم اخذ عينات من الدم و الكبد والبنكرياس من كل فأر. ثم تم تقدير جلوكوز الدم ومستوى الإنسولين و وظائف الكبد والكلى والدهون والبروتينات في السيرم وبعض القياسات فى الدم وكمية الجليكوجين في كبد كل جرذ وكذلك الدراسة الهستلوجية للبنكرياس .

وقد لوحظ ان المعاملة بلبن الجمال أدى الى تحسين ذو دلالة إحصائية في التقليل من حالات فقدان وزن الجسم وارتفاع الجلوكوز ونقص الانسولين فى الدم ونقص جليكوجين الكبد والتي تنتج عادة في الجرذان المصابة بمرض السكر المستحدث بمادة الألوكسان .كما لوحظ ان المعاملة بلبن الجمال ادى ايضا الى تحسن ملحوظ في التغيرات الهستوباثولوجية لخلايا بيتا في جزر لانجرهانز.