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Evaluating Clinicopathological and Ultrasonographic Studies on Treatment of Feline Hepatic Lipidosis

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ABSTRACT— In the present study, a total of 30 cats were divided into: "12" clinically healthy cats (control group), and "18" clinically diseased cats (diseased group). Clinically diseased cats were subjected to our suggested therapeutic regime which includes: Liver albumin plus®, Neuroton®, Amri-K® and fluid therapy. Treatment continued up to one month according to the severity of the case .Clinical, ultrasonographic and clinicopathological (erythrogram, leukogram, coagulation monitoring tests and blood biochemistry) examinations were achieved. Clinical examination of diseased group showed panorama of clinical signs including cachexia, vomiting, diarrhea, lethargy, hepato-encephalopathic signs (muscle fasciculation, tremors and convulsions) with hemorrhagic diathesis and icterus. Palpation revealed enlarged liver extended beyond the last right rib. Ten days after the starting of treatment, disappearance of all clinical signs except icterus was observed. Ultrasonography of diseased group showed diffuse hyperechogenicity of hepatic parenchyma, decreased visualization of intrahepatic blood vessels and enlargement of liver. Hepatic parenchyma was isoechoic or hyperechoic when compared with falciform or omental fats. Liver was also hyperechoic with respect to the spleen and renal cortices. Ultrasonography of treated group revealed returning of hepatic parenchyma to moderately echoic in addition to clearance of echogenic portal vein walls. Normocytic normochromic anemia in association with stress leukocytosis, prolongations of PT and APPT, and mild thrombocytopenia were recorded in diseased group. Blood biochemistry results of diseased group showed hepatic dysfunctions in the form of significant increases in hepatic enzyme activities, bilirubin concentration and lipogram parameters in addition to hypoproteinemia, hypoalbuminemia, insignificant changes in globulins concentration with significant decrease of A/G ratio along with significant decreases in BUN and creatinine concentrations. Hypoglycemia, hypokalemia and hypophosphatemia were also recorded. Treated group results showed significant improvements in all hematological and biochemical parameters toward control values. These significant clinicopathological improvements of treated group along with the observed improvement in ultrasonographic findings indicate the effectiveness of our suggested therapeutic regime in the treatment of Feline hepatic lipidosis.

Key words: Feline Hepatic Lipidosis, Clinical pathology, Ultrasonography.

INTRODUCTION

Hepatic lipidosis is known as "fatty liver disease" or "lipid mobilization syndrome". Feline hepatic lipidosis (FHL) is a common cholestatic disease affecting cats. It is considered the consequence of prolonged anorexia and subsequent dramatic lipolysis **(Center, 2005)**. It has been reported that, female cats (queens) are more affected than male cats (tom cats) **(Ettinger and Feldman, 2000)**. An explanation for an occasionally

proposed sex predisposition in FHL has never been convincingly described. FHL is characterized by excessive accumulation of triglycerides in hepatocytes. Although the exact mechanisms remain elusive, there is clearly imbalance between the influx of fatty acids (FA) derived from peripheral fat stores and *de novo* synthesis of FA in the liver on the one hand, and the rate of hepatic FA oxidation and the efflux of hepatic triglycerides via very-low density lipoprotein cholesterol (VLDL-c) on the other hand **(Armstrong and Blanchard, 2009)**. Hepatic lipidosis may be primary (idiopathic) or secondary to another disease **(Ettinger and Feldman, 2000)**. Primary or idiopathic hepatic lipidosis is most commonly recognized in obese indoor cats following a period of anorexia or stress. It results from the accumulation of large amounts of lipid in hepatocytes, altering hepatocytes morphology and producing acute hepatopathy associated with severe intrahepatic cholestasis and hepatic dysfunction. Mortality rate of this disease is high unless it is treated aggressively. Secondary hepatic lipidosis is a neuro-endocrine response to other diseases therefore less closely associated with obesity and it may be seen in normal or even thin cats **(Tilley and Smith, 2007)**. Most cases of primary hepatic lipidosis occur in middle-aged cats with no apparent breed predisposition. Clinical signs may appear to be non-specific at first including severe persistent anorexia with lethargy. Cats may lose weight and have unkempt appearance. Jaundice may or may not occur. Intensive treatment of cats is required as the disease has a high mortality if not managed aggressively. In cases of secondary lipidosis, the underlying cause of disease should be treated **(Nelson and Couto, 2009)**. Successful recovery of cats with hepatic lipidosis initially requires correction of fluid and electrolyte abnormalities but the cornerstone of therapy is enteral nutritional support concentrating on meeting protein and caloric needs **(Armstrong, 2014)**. The purpose of the present study was to evaluate the effectiveness of our suggested therapeutic regime in the treatment of feline hepatic lipidosis based on clinicopathological and ultrasonographic findings before and after treatment.

MATERIALS AND METHODS

Study Design

The present study was carried out on 30 cats (Siamese and Persian, 1.8-7.3 years-old) admitted to the clinic of Faculty of Veterinary Medicine, Cairo University and to a private veterinary clinic in Giza governorate in the period from March, 2013 to February, 2015. On the basis of clinical examination, cats were divided into: "12" clinically healthy cats (control group), and "18" clinically diseased cats ("6" cats were severely ill with signs of vomiting, icterus and weight loss, "2" cats showed signs of convulsions, tremors, vomiting, icterus and weight loss, and "10" cats showed signs of lethargy, dullness and icterus). Our treatment was applied on clinically diseased cats.

Clinical Examination

Respiratory rate, pulse rate and rectal temperature of all cats were recorded. Examination of cat mucous membranes and superficial lymph nodes was done. All cats were thoroughly investigated and clinically examined by abdominal palpation and tactile percussion according to the method described by **Kelly (1984)**.

Ultrasonography

Ultrasonography was performed after 12 hours fasting. Examined cats were positioned in dorsal recumbency. Cranial ventral abdomen was clipped and sheaved then covered with coupling gel. Transverse and

longitudinal scans were taken using Pie-Medical Scanner (Maastricht, Netherlands) and sector transducer with alternating frequency of 5.0-7.5 MHz according to the method described by **Nyland *et al.*, (1989)** .

Therapeutic Regime

Diseased cats were subjected to massive daily treatment as follows; oral administration of one tablet from hepato-potentiating drug commercially identified as Liver albumin plus® (Sigma pharmaceutical company), intramuscular (I/M) injection of one Neuroton® ampule (Amoun pharmaceutical company), I/M injection of one Amri-K® ampule (Amria Egypt company), and fluid therapy in the form of dextrose 5% solution (Nasr pharmaceutical company). Treatment continued up to one month according to the severity of the case. Nutritional treatment included dietary supplement of high carbohydrate, low fat and high quality protein diet was applied.

Clinicopathological Examinations

Blood samples from the anterior median vein of each cat were collected at the beginning of the study from the control and diseased groups and from the treated group at the end of treatment period. Obtained blood sample was divided into three parts. First part was anticoagulated by EDTA and used for evaluating hemogram. Second part was collected in sodium citrate-containing tube; its plasma was taken after centrifugation and kept deep frozen until analysis of prothrombin time (PT) and activated partial thromboplastin time (APTT). PT and APTT along with platelets count considered as coagulation monitoring tests according to **Feldman *et al.*, (2000)**. Third part was collected in a clean centrifuge tube for serum separation, clear non hemolysed supernatant serum was harvested for biochemical studies.

Hematological and Biochemical Studies

Hematological Studies

Erythrocyte (RBCs) and total leukocyte (TLC) counts, packed cell volume (PCV), hemoglobin (Hb) concentration and differential leukocytic count (DLC) on Field stained blood smears were performed according to **Feldman *et al.*, (2000)**.

Biochemical Studies

Serum samples were prepared to assay the following biochemical studies; alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP) and gamma-glutamyl transferase (GGT) activities according to **Reitman and Frankel (1957)**, **Tietz (1986)**, **Dumas and Biggs (1972)** and **Szasz (1976)**, respectively. Total and direct bilirubin concentrations were done as described by **Doumas *et al.*, (1973)**. Total cholesterol and high density lipoprotein cholesterol (HDL-c) concentrations were determined according to **Allain *et al.*, (1974)** and **Warnick *et al.*, (1983)**, respectively. Calculated low density lipoprotein cholesterol (LDL-c) and measured very low density lipoprotein cholesterol (VLDL-c) concentrations were carried according to **Friedewald *et al.*, (1972)**. Total triglycerides concentration was determined according to **Wahlefeld (1974)**. Total proteins and albumin concentrations were measured according to **Weichselbaun, (1946)** and **Dumas and Biggs (1972)**, respectively while globulins concentration was calculated by subtracting value of albumin from value of total proteins. A/G ratio was obtained by subdividing values of albumin by those of globulins. BUN, creatinine, glucose, potassium and inorganic phosphorus concentrations were determined according to **Tabacco *et al.*, (1979)**, **Fabiny and Ertingshausen (1971)**, **Trinder (1959)**, **Scott *et al.*, (1999)** and **Goodwin (1970)**, respectively.

The above mentioned biochemical parameters were assayed using reagent kits supplied by StanBio Laboratories incorporation, USA.

Statistical Analysis

Analysis of the data was performed by ANOVA (Analysis Of Variance) test using the computer software Statistical Package for Social Sciences (SPSS) for Windows (version 10.0) according to the method described by **Irwan (1996)**.

RESULTS

Clinical Findings before Treatment

Clinically healthy cats (control group) were characterized by absence of any clinical signs of hepatobiliary disorders. Clinically diseased cats showed panorama of clinical signs including cachexia, vomiting, diarrhea, lethargy, hepato-encephalopathic signs (muscle fasciculation, tremors and convulsions) with hemorrhagic diathesis. Palpation revealed enlarged liver extended beyond the last right rib. Significant increases in respiratory and pulse rates between control and diseased groups were recorded.(Table, 1)

Clinical Findings after Treatment

Ten days after the starting of treatment, disappearance of all clinical signs except icterus was recorded. Six cases which were severely ill with signs of vomiting, icterus and weight loss treated efficiently and started to eat one week after treatment. Two cases which showed signs of convulsions, tremors, vomiting, icterus and weight loss, controlled by the end of experiment (one month). Ten cases with signs of lethargy, dullness and icterus responded rapidly to treatment within 3-7 days. Significant decreases in respiratory and pulse rates between diseased and treated groups were recorded. (Table, 2)

Ultrasonographic Findings

Ultrasonographic findings of control group showed isoechoic hepatic parenchyma and hyperechoic portal vein walls, while that for diseased group showed diffuse hyperechogenicity of hepatic parenchyma, decreased visualization of intrahepatic blood vessels and enlargement of liver. Hepatic parenchyma was isoechoic or hyperechoic when compared with falciform or omental fats. Liver was also hyperechoic with respect to the spleen and renal cortices. There was increased attenuation of ultrasound waves by hepatic parenchyma. Ultrasonographic findings of treated group after a period of 10 days to 1 month showed, hepatic parenchyma was returned to moderately echoic in addition to clearance of echogenic portal vein walls. (Figures, 1 &2).

Clinicopathological Results

Erythrogram

Mean values of erythrogram including PCV %, Hb concentration, RBCs count, mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration (MCHC) of different groups are illustrated in tables 3 &4.

Erythrogram results of diseased group when compared with that of control group revealed presence of normocytic normochromic anemia which determined by significant decreases in PCV%, Hb concentration and RBCs count, and insignificant changes in MCV and MCHC values. Poikilocytosis and Heinz bodies were observed during microscopic examination of stained blood films.

Leukogram

Mean values of leukogram including TLC, neutrophil, lymphocyte, eosinophil and monocyte counts of different groups are illustrated in tables 3 &4.

Leukogram results of diseased group when compared with that of control group showed typical picture of stress leukocytosis including neutrophilia, lymphopenia, monocytopenia and eosinopenia.

Coagulation Profile

Mean values of coagulation profile including PT, APTT and platelets count of different groups are illustrated in tables 3 &4.

Results of diseased group when compared with that of control group showed prolongations of PT and APPT, and mild thrombocytopenia.

Biochemical Evaluation

Statistical analysis of different biochemical parameters of different groups is illustrated in tables 5 &6.

Biochemical results of diseased group when compared with that of control group revealed, the activities of hepatic enzymes (AST, ALT, ALP and GGT) and the concentration of bilirubin were significantly increased. Lipogram results showed significant increases in total cholesterol, HDL-c, LDL-c, VLDL-c and total triglycerides concentrations. Protein profile results showed hypoproteinemia, hypoalbuminemia, insignificant changes in globulins concentration with significant decrease of A/G ratio. BUN and creatinine concentrations were significantly decreased. Hypoglycemia, hypokalemia and hypophosphatemia were recorded.

By comparing all the previously recorded clinicopathological findings including hematological and biochemical parameters of treated group with that of the diseased group, significant improvement in treated group values (toward control values) was recorded as shown in tables 4 &6.

Table (1): General clinical examination of clinically healthy (control) and clinically hepatic lipidosis diseased group

Parameters	Control group	Diseased group
Respiratory rate	28.00 ± 3.56	33.32 ± 3.12
Pulse rate	81.18 ± 3.68	90.27 ± 4.16
Rectal temperature	38.40 ± 0.22	38.90 ± 0.27
Mucous membranes	Very faint rosy red	Icteric
Superficial lymph nodes	Free	Free

FHL represents “Feline Hepatic Lipidosis.”

Table (2): General clinical examination of clinically FHL diseased and treated groups

Parameters	Diseased group	Treated group
Respiratory rate	33.32 ± 3.12	29.90 ± 2.92
Pulse rate	90.27 ± 4.16	87.19 ± 4.89
Rectal temperature	38.90 ± 0.27	38.60 ± 0.30
Mucous membranes	Icteric	Icteric
Superficial lymph nodes	Free	Free

Table (3): Hematological parameters of clinically healthy (control) and clinically FHL diseased groups (mean values ± SD).

Parameters	Control group	Diseased group
PCV (%)	41.87±1.29	31.75±1.58*
Hb (g/dl)	13.77±1.42	8.87±1.09*
RBCs ($\times 10^6$/µl)	7.35±0.58	4.35±0.39*
MCV(fl)	65.03±4.13	64.60±5.58
MCHC (g %)	32.54±2.04	29.27±1.99
TLC ($\times 10^3$/µl)	9.96 ±0.80	11.03 ±0.97*
Neutrophil count ($\times 10^3$/µl)	6.54 ±0.33	8.41 ±0.23*
Lymphocyte count ($\times 10^3$/µl)	3.45 ±0.19	2.40 ±0.17*
Monocyte count ($\times 10^3$/µl)	0.43 ±0.03	0.33 ±0.02*
Eosinophil count ($\times 10^3$/µl)	0.54±0.03	0.43 ±0.04*
Platelets count ($\times 10^3$/µl)	210.00±7.03	199.00±7.85*
PT (Sec.)	16.00±0.09	21.00±0.18*
APPT (Sec.)	18.00±0.72	26.32±0.83*

*Significant difference at P value ≤ 0.05.

Table (4): Hematological parameters of clinically FHL diseased and treated groups (mean values ± SD).

Parameters	Diseased group	Treated group
PCV (%)	31.75±1.58	36.98±1.64*
Hb (g/dl)	8.87±1.09	10.79±1.18*
RBCs ($\times 10^6$/µl)	4.35±0.39	5.88±0.45*
MCV(fl)	64.60±5.58	64.95±5.33
MCHC (g %)	29.27±1.99	30.01±2.11
TLC ($\times 10^3$/µl)	11.03 ±0.97	10.37 ±0.38*
Neutrophil count ($\times 10^3$/µl)	8.41 ±0.23	7.03 ±0.23*
Lymphocyte count ($\times 10^3$/µl)	2.40 ±0.17	3.00±0.17*
Monocyte count ($\times 10^3$/µl)	0.33 ±0.02	0.40 ±0.02*
Eosinophil count ($\times 10^3$/µl)	0.43 ±0.04	0.51 ±0.04*
Platelets count ($\times 10^3$/µl)	199.00±7.85	203.23±7.11*
PT (Sec.)	21.00±0.18	18.00±0.18*
APPT (Sec.)	26.32±0.83	22.32±0.67*

*Significant difference at P value ≤ 0.05.

Table (5): Biochemical parameters of clinically healthy (control) and clinically FHL diseased groups (mean values ± SD).

Parameters	Control group	Diseased group
ALT (IU/L)	62.69 ± 3.2	122.10 ± 7.14*
AST (IU/L)	63.48 ± 2.6	139.12 ± 11.9*
ALP (IU/L)	31.76 ± 0.24	68.94 ± 8.6*
GGT (IU/L)	18.07 ± 0.33	37.98 ± 2.4*
Total bilirubin (mg/dl)	0.76 ± 0.15	1.43 ±0.18*
Direct bilirubin (mg/dl)	0.31 ± 0.12	0.63 ±0.06*
Indirect bilirubin (mg/dl)	0.45 ± 0.10	0.80 ±0.05*
Total cholesterol (mg/dl)	162.98 ± 9.86	266.15 ± 13.66*
HDL-c (mg/dl)	65.98 ± 3.72	78.19 ± 4.13*
LDL-c (mg/dl)	86.19 ± 5.75	165.18 ± 8.44*
VLDL-c (mg/dl)	10.79 ± 0.91	23.17 ± 1.18*
Total triglycerides (mg/dl)	53.97 ± 3.35	115.20 ± 10.59*
T. proteins (g/dl)	8.16 ± 0.72	7.51 ± 0.19*
Albumin (g/dl)	4.13 ± 0.12	3.37 ± 0.77*
Globulins (g/dl)	4.02 ± 0.29	4.12 ± 0.31
A/G	1.03 ± 0.21	0.82 ± 0.14*
BUN (mg/dl)	22.27 ± 0.19	16.80 ±1.6*
Creatinine (mg/dl)	0.86 ± 0.05	0.71 ± 0.04*
Glucose (mg/dl)	89.19 ± 0.8	69.85 ± 1.8*
Potassium (mEq/L)	4.90± 0.11	2.51± 0.14*
Inorganic phosphorus (mg/dl)	5.10± 0.30	2.97± 0.28*

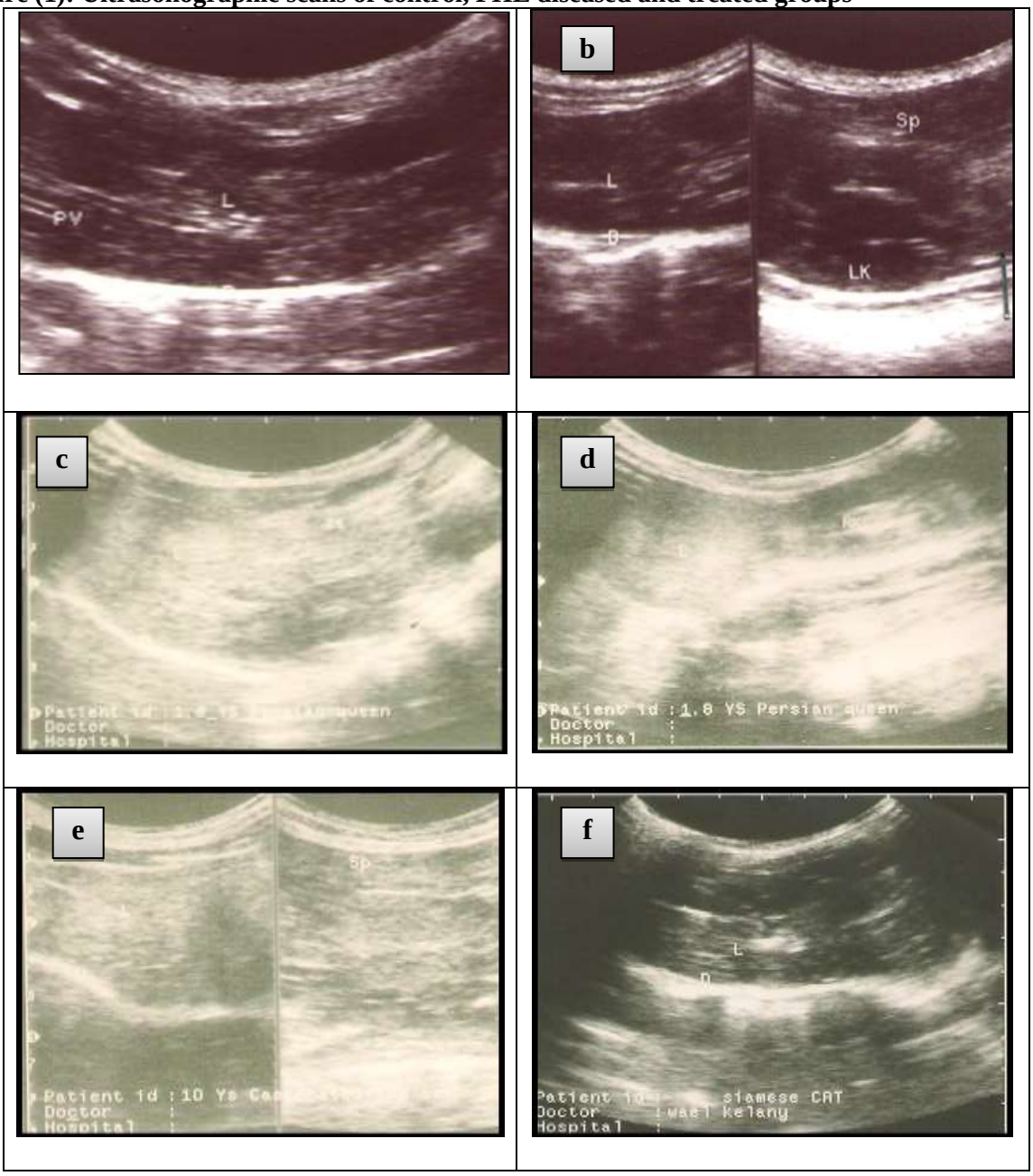
*Significant difference at P value ≤ 0.05.

Table (6): Biochemical parameters of clinically FHL diseased and treated groups (mean values ± SD).

Parameters	Diseased group	Treated group
ALT (IU/L)	122.10 ± 7.14	99.09± 6.18*
AST (IU/L)	139.12 ± 11.9	102.14± 9.93*
ALP (IU/L)	68.94 ± 8.6	49.01± 6.9*
GGT (IU/L)	37.98 ± 2.4	25.88± 1.94*
Total bilirubin (mg/dl)	1.43 ±0.18	0.98±0.16*
Direct bilirubin (mg/dl)	0.63 ±0.06	0.45±0.15*
Indirect bilirubin (mg/dl)	0.80 ±0.05	0.53±0.04*
Total cholesterol (mg/dl)	266.15 ± 13.66	189.41± 11.35*
HDL-c (mg/dl)	78.19 ± 4.13	69.21± 3.22*
LDL-c (mg/dl)	165.18 ± 8.44	101.14± 6.18*
VLDL-c (mg/dl)	23.17 ± 1.18	19.01± 1.07*
Total triglycerides (mg/dl)	115.20 ± 10.59	95.91± 8.74*
T. proteins (g/dl)	7.51 ± 0.19	8.11± 0.11*
Albumin (g/dl)	3.37 ± 0.77	4.01± 0.91*
Globulins (g/dl)	4.12 ± 0.31	4.08± 0.22
A/G	0.82 ± 0.14	0.98± 0.11*
BUN (mg/dl)	16.80 ±1.6	19.88±0.99*
Creatinine (mg/dl)	0.71 ± 0.04	0.82± 0.02*
Glucose (mg/dl)	69.85 ± 1.8	79.62± 0.98*
Potassium (mEq/L)	2.51± 0.14	3.92± 0.09*
Inorganic phosphorus (mg/dl)	2.97± 0.28	4.31± 0.19*

*Significant difference at P value ≤ 0.05.

Figure (1): Ultrasonographic scans of control, FHL diseased and treated groups



(a): Normal hepatic scan of control group (Persian queen, 2.6 years-old) showed isoechoic hepatic parenchyma and hyperechoic portal vein walls.

(b): Normal double B-scan of liver, left kidney and spleen of control group (Siamese queen, 7.3 years-old) showed increased echogenicity of spleen than hepatic parenchyma and increased echogenicity of hepatic parenchyma than left renal cortex.

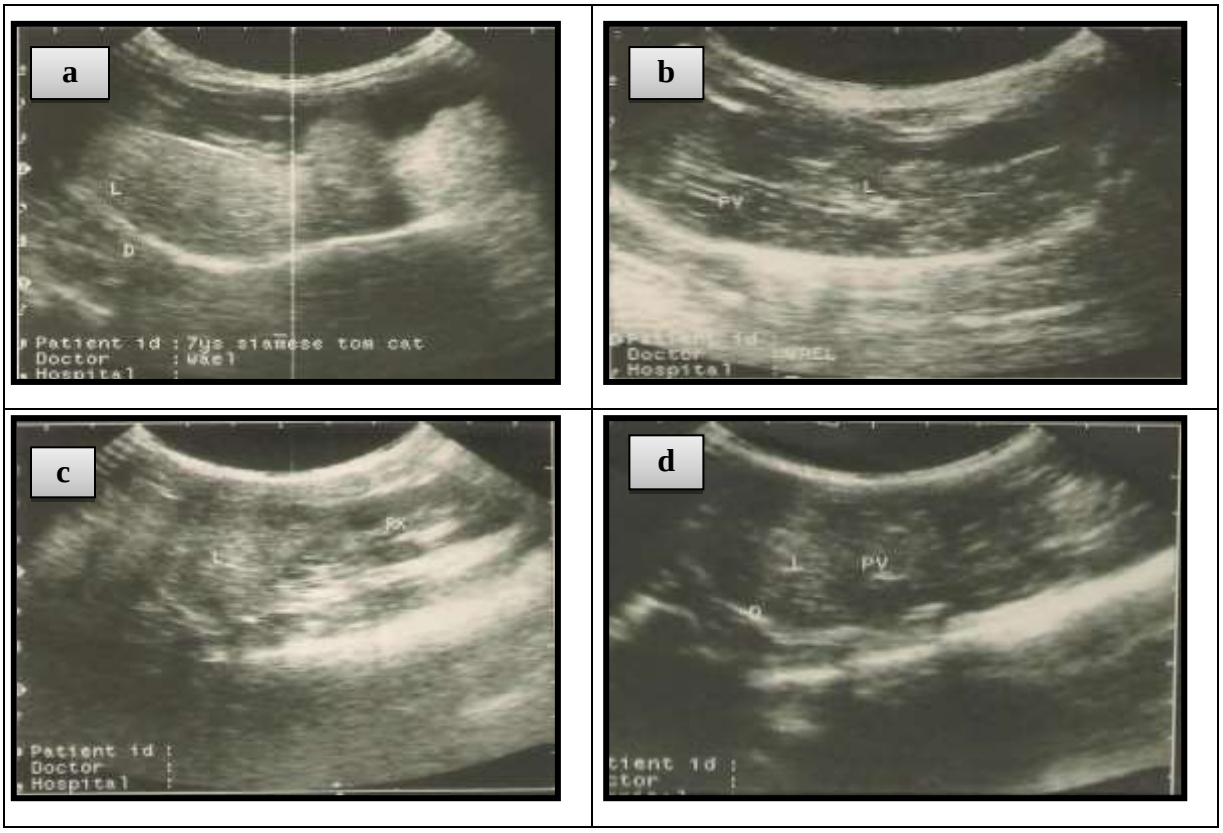
(c): Hepatic scan of diseased group (Persian queen, 1.8 years-old) showed marked increase in echogenicity with absence of echogenic walls of portal veins.

(d): Hepatic scan of treated group (the same Persian queen of "c") after 14 days of treatment displayed decreased echogenicity, but the liver parenchyma still moderately hyperechoic.

(e): Sagittal double B-scan of diseased group liver and spleen (Siamese tom cat, 7 years-old) showed the liver parenchyma is little bit more echoic than spleen with disclearance of echogenic portal vein walls.

(f): Hepatic scan of treated group (the same Siamese tom cat of "e") after 10 days of revealed moderately echoic liver parenchyma with clearance of portal veins.

Figure (2): Ultrasonographic scans of control, FHL diseased and treated groups



(a): Hepatic scan of diseased group (Siamese tom cat, 7 years-old) showing presence of diffuse hepatic hyperechogenicity with ascetic fluid, and clearance of liver sublobes (disappearance of echogenic portal vein walls).

(b): Hepatic scan of treated group (the same Siamese tom cat of "a") after 22 days of treatment revealed decreased echodenicity with clearance of echogenic portal vein walls.

(c): Sagittal scan of diseased group liver and right kidney (Persian queen, 2 years-old) displayed diffuse hyperechoic liver parenchyma (fatty liver) with disclearance of echogenic portal vein walls. Liver parenchyma showed marked echogenicity than renal cortex.

(d): Hepatic scan of treated group (the same Persian queen of "c") after one month of treatment revealed moderately echoic liver parenchyma with clear echogenic portal vein walls.

DISCUSSION

Feline hepatic lipidosis "feline fatty liver syndrome" is one of the most common forms of liver disease of cats. Liver main functions include protein synthesis, production of chemicals necessary for digestion, and detoxification of the body. Liver also plays an important role in metabolism, emulsification of fats, production of coagulation factors, and in decomposition of red blood cells. Liver is of such importance to the body, carrying out so many complex functions, that there is no way to compensate the loss of the liver when it fails. Normally, when a body is undernourished or starved, the body automatically moves fat from its reserves to the liver to be converted into lipoproteins for energy. Cat bodies physiologically are not able to convert large stores of fat, so when a cat is in starvation mode, the fat that is released to the liver is not processed efficiently, resulting in fatty and low functioning liver. As the fat accumulates in the liver it becomes swollen and if not treated promptly, hepatic lipidosis can lead to various complications and eventually death **(Brenner *et al.*, 2011)**.

Thirty cats ("12" clinically healthy and "18" clinically hepatic lipidosis diseased cats) referred between 2013 and 2015 were included in this study. The most common complaints of clinically diseased cats which reported by the owner on initial questionnaire were lethargy, history of weight loss and anorexia. Few cats were scored as obese with the majority considered to be underweight or very thin. This finding is in contrast with that of previous reports of obesity being a predisposing risk factor for FHL **(Armstrong, 2014)**. Less frequent complaints were reduced appetite, vomiting, neurological signs and jaundice.

Ultrasonography is the most useful modality available for hepatic imaging and also permits evaluation of other abdominal organs to detect abnormalities associated with diseases that accompany hepatic lipidosis **(Brenner *et al.*, 2011)**. In the present study ultrasonographic examination of diseased group showed an enlarged liver and hyperechoic parenchyma, these findings are in agreement with **Armstrong and Blanchard (2009)** who recorded that, the hepatomegaly is commonly seen in cases of feline hepatic lipidosis. Ultrasonography of treated group revealed the returning of hepatic parenchyma to moderately echoic in addition to clearance of echogenic portal vein walls.

Hematological results including presence of normocytic, normochromic anemia and stress leukocytosis are in agreement with that reported by **Couto (2009)**. Coagulation profile of diseased group revealed prolongations of PT "measures the activities of extrinsic and common pathway factors" and APTT "an indicator of the function of coagulation factors in the intrinsic and common pathways", and thrombocytopenia. Similar results were recorded by **Center (2005)**. Liver plays an important role in the production of blood coagulation factors, so that the recorded abnormalities in coagulation profile are related to its affection **(Brenner *et al.*, 2011)** which supported by the observed changes in hepatic function tests and ultrasonography of liver. Response of diseased group to vitamin K supplementation during our treatment, suggests vitamin K deficiency. Intrahepatic cholestasis leading to reduced enterohepatic circulation of bile acids resulting in reduced absorption of fat-soluble vitamins (vitamin K) is the suspected mechanism associated with FHL.

Observed biochemical abnormalities in diseased group are typical of an intrahepatic cholestatic disorder including: increased bilirubin concentration and increased enzyme

activities of ALP, ALT, AST and GGT. These results are in agreement with those obtained by **Brown *et al.*, (2000**). Increased hepatic enzyme activity levels along with the present hypoglycemia, hypoalbuminemia, and low BUN are indicators of significant altered hepatic dysfunction which was confirmed by the observed ultrasonographic hepatic findings. Recoded hypoalbuminemia is often seen in hepatic disease and indicate the lower production either by impaired liver functions or decreased protein uptake **(Nelson *et al.*, 2004)**. Gastrointestinal signs in FHL cats reported in this study, in combination with decreased intake (reduced appetite/anorexia) are considered the common causes of the recorded hypokalemia and hypophosphatemia **(Center *et al.*, 2015** and **Dibartola *et al.*, 2004)**. Previous study done by **Megumi *et al.*, (2015)** showed similar electrolyte changes.

Conclusion and recommendations:

The present study concluded the effectiveness of our suggested therapeutic regime in the treatment of FHL depending on the observed significant improvement in all clinical hematological and biochemical parameters along with the significant improvement in hepatic ultrasonographic findings after treatment. The study recommends the use of our therapeutic regime for a longer period varied according to the severity of the case until complete curing of all cats.

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