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Clinical and Diagnostic Studies on Feline Cholestasis and the Significance of mi-RNA-122 as a Biomarker

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Abstract

THE PRESENT STUDY included 719 cats admitted to the teaching veterinary hospital, the faculty of veterinary medicine, Cairo University, and private clinics. All cats were from different breeds, sexes, and ages. This number was divided into 488 diseased and 231 apparently healthy cats. 128 (26.2%) cases were recorded with different hepatopathies from the diseased number. Feline cholestasis was detected in 32 cases (25%). The present study gathered 52 cats (32 cases with cholestasis and 20 healthy cats). Comprehensive clinical, ultrasonographic, and Hemato-biochemical investigations besides hepatocyte-derived miRNA-122 evaluation were conducted on all animals. Feline cholestasis was mostly detected in mixed breed cats older than 3 years, more in tom cats than queens. The most recorded clinical signs were icteric mucous membranes, yellow skin, anorexia, cranial abdominal pain, vomiting, and diarrhea. The recorded causes of cholestasis were biliary sludge, cholecystitis, hepatic lipidosis, and cholangitis. Descriptive ultrasonographic findings were also included. Regarding cats with cholestasis, Physical examination showed significant elevation in respiration and pulse rates. The hematological evaluation showed significant anemia with elevation of absolute neutrophilic count. Serum biochemistry revealed significant elevation in all hepatic function enzymes and bilirubin. Also, a significant decrease was recorded in potassium levels. Hepatic biomarker (miRNA-122) fold expression recorded a significant increase in diseased cats indicating a close association between cholestasis and hepato-biliary cellular injury. Mi-RNA-122 was a specific and early predictor of inflammatory reactions of varying degrees. This made it of major clinical significance in the diagnosis of cholestasis in cats.

Keywords: Feline cholestasis, Incidence, Ultrasonography, Hemato-biochemistry, Mi-RNA-122.

Introduction

The feline liver was known to be the largest internal organ occupying about 4% of the cat's total body weight. The right medial lobe came in contact with the right portion of the gallbladder (GB), The quadrate lobe was centrally located and encircled the GB [1, 2]. The cat gall bladder ultrasonography appeared to differ in conformance, encircled by the echogenic wall and adjacent to the right medial lobe laterally and the quadrate lobe medially [3].

Cat liver has six lobes and is situated in the intrathoracic part of the abdominal cavity caudal to the diaphragm [4]. In Domestic Cat gall bladder in Egypt, three main forms are gathered: single gall bladder, duplex fundus, and bilobed gall bladder. The cat bile duct was relatively long, passed through the hepatoduodenal ligament integrated with the pancreatic duct, and penetrated the duodenal wall to open on the major duodenal papilla encircled by the hepatopancreatic ampulla 2-2.5 cm distal to the pylorus [3].

Feline liver hematopoietic role onsets at embryonic life, then after birth, other functions were modified including drug metabolism, detoxification, and production of plasma proteins, albumin, and blood clotting factors. The liver shares a major role in regulating lipids and glucose besides conjugation and eliminating harmful substances and toxins. The bile of cats is composed of cholesterol and non-sulfated bile acids, sharing in digestion and maintenance of digestive functions [2, 5].

Feline hepatopathies are a common problem and possess challenging criteria for veterinary practitioners. Etiology, clinical signs, and prognosis of hepatopathies in cats were commonly related to the biliary tree and gallbladder as the biliary system in feline species was considered the primary target for infectious and non-infectious causes [6, 7]. Non-infectious causes of hepatopathies were more

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prevalent than primary infectious causes [8]. The most common causes of feline hepatic disorders might be either biliary diseases like Cholangitis-cholangiohepatitis complex, cholestasis, and triaditis or infiltrative as hepatic lipidosis [9].

Cholestasis is a serious medical problem connected to biliary illness and is considered a pathophysiological condition linked to biliary system malfunction. It was classified into extra-hepatic and intra-hepatic cholestasis [2]. Extra-hepatic biliary tract obstruction (EHBDO) was defined as the obstruction of the extra-hepatic biliary system getting into the duodenum, following extra luminal compression or intraluminal obstruction of the common bile duct [10]. The most frequent causes of EHBDO were biliary sludge and stones which were linked to cholangitis and diminished gallbladder contractility. Other causes included gastrointestinal disease, pancreatitis, biliary neoplasia, gallbladder mucocele, and foreign bodies were recorded [11]. Intra-hepatic cholestasis was commonly caused by narrowing or compressed bile ducts in the hepatic parenchyma. Hepatic lipidosis might result in a marked increase in liver weight, function disability, and intra-hepatic cholestasis [12, 13].

Clinical presentation of feline hepatopathies was commonly varied and might be overlapped in some cases. The most common clinical signs of cats with cholestasis were jaundice, anorexia, depression, vomiting, and abdominal distention. Cranial abdominal pain may be recorded but it might be masked. Also, Acholic or pale stool was detected if there was a full biliary tract obstruction [2, 14].

The diagnostic protocol depends on numerous steps. They included many diagnostic tools such as hematological examination, serum biochemistry, stool analysis, and abdominal imaging evaluation. The serum biochemical profile was specific for evaluating the hepatocytes and biliary system functions. This profile included serum liver enzymes, albumin, urea, bilirubin, cholesterol, and glucose levels. Serum biochemical is helpful but may not prove the degree of inflammation associated with cholestasis. A novel specific and sensitive diagnostic biomarker for feline cholestasis diagnosis was the estimation of serum concentrations of hepatic derived micro-RNA-122. It was considered a useful specific organ damage biomarker due to its considerable facilitating assessment, stability in biological fluids, and tissue specificity [15, 16].

The main objectives of the present study were the detection of the incidence and common causes of feline cholestasis at teaching veterinary hospital, faculty of veterinary medicine, Cairo University, Egypt. Also, estimation of hemato-biochemical status and evaluation of hepatocyte-derived miRNA-122 as a novel biomarker for the detection of hepatic damage degree and dysfunction in cases with feline cholestasis.

Material and Methods

Animals

The present study gathered 719 cats admitted to the teaching veterinary hospital, faculty of veterinary medicine, Cairo University, and private clinics belonging to Giza governorate (from Jul.2023 to Jul.2024). All cats were from different common breeds, sex and ages. This number was divided into 488 diseased and 231 apparently healthy cats. Of the diseased number, 128 (26.2%) cases suffered from different hepatopathies. Feline cholestasis was detected in 32 cases (25%). The present study was conducted on 52 cats and thorough clinical examination, ultrasonography, and hemato-biochemical investigation was applied to all cases. Based on the examination of cats, they were classified into a diseased group (n=32) included cats diagnosed with cholestasis, and apparently healthy control group (n=20). Healthy cats were admitted for annual health checkups and had normal hemato-biochemical and ultrasonographic examinations.

Clinical examination

All admitted cases were recorded in the registration sheet for date, age, breed, and the main complaint mentioned by owners. Complete clinical examination including case history (medical, dietary, vaccination, management, and environmental) was taken. Vital signs were assessed including rectal temperature, pulse, respiration, superficial lymph nodes, and mucous membranes. Different body systems and organs were carefully investigated [17].

Ultrasonographic examination

Ultrasonography was done for the evaluation of hepatic parenchyma, blood vessels, biliary system, gallbladder, and abdominal organs by using the E-Saote® ultrasonographic device with a micro-convex transducer (5-10 MHz) according to [18].

Hemato-biochemical analysis and hepatocyte-derived miRNA-122 evaluation

Whole blood samples were obtained from cephalic or jugular veins of all cats in the present study. Samples were taken EDTA tubes for estimation of hematological parameters including packed cell volume (PCV), hemoglobin concentration, red blood cell count (RBCs), red blood cell indices including mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC). Total white blood cells count (WBCs) and differential leukocytic counts including absolute numbers of neutrophils, lymphocytes, monocytes, eosinophils, and basophils were also evaluated. Plain tubes were used for serum separation and used for the quantification of total protein, albumin, globulin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline

phosphatase (ALP), gamma-glutamyl-transferase (GGT), total, direct bilirubin, BUN, creatinine, amylase, lipase, sodium, potassium, chloride, total cholesterol, triglycerides, and glucose using specific test kits according to manufacturer instructions (Spectrum Diagnostic Co., MDSS, GmbH, Hannover, Germany). Serum samples were stored at −80ºC till hepatocyte-derived miRNA-122 was analyzed. RNA from samples was extracted using RNeasy Mini Kit (Catalogue no.74104, QIAGEN, Germany) according to manufacturer instructions [19, 20]. Quantitative type polymerase chain reaction (PCR) for hepatocyte-derived miRNA-122 estimation was done using Quantitect SYBR Green PCR Kit (QIAGEN, Germany). Primers and probes used in SYBR Green Real-time PCR are shown in Table 1 and cyclic conditions for SYBR Green Real-time PCR are designed in Table 2. Amplification curves and cycle threshold (CT) values were determined by the Stratagene MX3005P software [21]. Estimation of gene expression variation in different samples and the cycle threshold of each sample was compared with that of the apparently healthy control group.

Fecal analysis

A stool sample of each case was collected and examined soon on admission. Gross appearance, direct smear, and concentration floatation techniques were applied for the presence of adult worms, protozoa oocytes, and parasitic eggs [22].

Statistical analysis

Recorded data among hematology and serum biochemistry were presented as mean ± standard error. The comparison between the control and the diseased group was made using the statistic program SPSS version 16.0 using a t-test. $p \leq 0.05$ was considered statistically significant [23].

Results

Hepatopathies are considered a common feline disorder in Egypt. The incidence of feline hepatopathies and cholestasis were recorded at a teaching veterinary pet animal hospital, faculty of veterinary medicine, Cairo University, Egypt for one year study (Jul. 2023-Jul. 2024). Hepatopathies were recorded in 128 cases (26.2%) of total diseased cases (488) admitted to the hospital. Feline cholestasis was detected in 32 cases (25%) out of 128 cases suffered from different causes of hepatopathies (Table 3).

Regarding the risk factors associated with feline cholestasis, it was mostly recorded in mixed breeds, especially at the age of more than 3 years. Disease was recorded more in tom cats than queens while most cases were seen during spring months. Cats with free access to feed all day were at risk of cholestasis more than the scheduled daily feed program. Regarding feed type, cholestasis incidence was higher in cats that depend on fresh feed (wet home-made feedstuffs) than mixed feed type (Table 4).

The most recorded clinical signs were severe jaundice, yellow discoloration of oral mucous membrane (Fig. 1a), episcleral (Fig. 1b), and skin (Fig. 1c). Anorexia, weight loss, and pale acholic stool were detected signs. Cranial abdominal pain was evident in certain cases while vomiting and diarrhea were also associated with clinical signs that may vary in occurrence and severity. The most common recorded causes of feline cholestasis were cholecystitis (12 cases), cholangitis (10 cases), hepatic lipidosis (7 cases), and biliary sludge (3 cases).

Abdominal ultrasonographic examination was applied on diseased cases at the time of admission. The gall bladder appeared large with hyper-echogenic biliary sludge attached to the wall and did not show movability by changing the animal's position (Fig. 2a, b). Also, hepatic parenchyma showed a slight hypo-echogenic pattern indicating a slight inflammatory reaction (Fig. 2a). Cholecystitis was another ultrasonographic finding that appeared as thickened hyper-echogenic gall bladder wall with edge shadow artifact and diffused hyper-echogenicity indicating chronic inflammatory reaction (Fig.2c).

All admitted cases with feline cholestasis were subjected to complete physical and hemato-biochemical examinations compared with apparently healthy cats. In terms of physical examination, statistical analysis showed significant ($P \leq 0.001$) increase in both respiration and pulse rates while there was no significant change regarding rectal temperature (Table 5).

Regarding hematological evaluation shown in (Table 6), statistical analysis showed a significant decrease in PCV, RBCs count ($P \leq 0.05$), and hemoglobin concentration ($P \leq 0.01$) in affected cases. Absolute neutrophil count showed a significant ($P \leq 0.01$) increase in diseased cats compared to control healthy cats while absolute basophil count showed a significant ($P \leq 0.001$) decrease in affected cats. Other parameters had no significant changes toward healthy cats.

In the same context, biochemical parameters were illustrated in (Table 7). Statistical results among hepatic function tests showed significant ($P \leq 0.001$) elevation in the levels of aspartate aminotransferase, alkaline phosphatase, γ-glutamyl transferase, total and direct bilirubin in diseased cats compared with healthy cats. Significant elevation ($P \leq 0.01$) was recorded for alanine aminotransferase (ALT) in diseased cats. Pancreatic functions including amylase and lipase showed a significant ($P \leq 0.001$) decrease in diseased cats. Mineral profile results showed a significant ($P \leq 0.05$) decrease in serum potassium levels in affected cats. Hepatic biomarker (miRNA-122) fold expression revealed a high significant

($P \leq 0.001$) increase in cats with cholestasis compared with apparently healthy control group. No significant changes were recorded for other biochemical parameters.

Discussion

Feline hepatopathies are considered major and common disorders facing small animal practitioners. Cats commonly had hepatobiliary disorder or hepatic lipidosis more than parenchymal disease [6]. The present study showed the incidence of feline hepatopathies for a year study (from Jul. 2023 to Jul. 2024) was 26.2% from diseased conditions admitted to teaching veterinary pet animal hospital, faculty of Veterinary Medicine, Cairo University, Egypt. There is no available data about the incidence of hepatopathies among cats in Egypt presented till now. A study reported an occurrence of 7.02% regarding cats in Brazil [24]. The difference from our study may be attributed to geographical distribution, feed type, stress conditions, and observation of symptoms as they may be clear or masked. In concurred with the present study regarding feline cholestasis incidence, another study recorded an etiological prevalence of cholestasis as cholecystitis (23%) and hepatic lipidosis (2%) [25]. In terms of risk factors, the present study showed that mixed-breed cats were more prone to cholestasis followed by Persian breed. In contrary, domestic shorthair cats were the common affected breed [26]. The affected breeds as Siamese, domestic shorthair, domestic longhair, Ragdoll, Bengal, Himalayan, and Scottish Fold [27]. The variation in the incidence is explained by the difference in the distribution of cat breeds in Egypt and other countries. In the same context of the present findings, cats with cholestasis reported a median age of more than 3 years that may reach about 10, 11, and 12 years [27, 28]. The most recorded causes of feline cholestasis were cholangitis and cholecystitis mimicked the findings of a recent study [2]. The most recorded clinical symptoms of cholestasis were jaundice, acholic stool, anorexia, weight loss, vomiting, and diarrhea[6]. In agreement with a recent study [29], physical examination results showed a significant elevation in respiration and pulse rates. Ultrasonographic findings indicated a distended gall bladder with hyper-echogenic debris material attached to its wall in cases with biliary sludge [10, 27]. Over-thickening hyper-echogenic gall bladder wall with edge shadow artifact and diffused hyper-echogenicity of hepatic parenchyma were associated with cholecystitis. The present results regarding hematology revealed a significant decrease in PCV, hemoglobin, and RBCs count indicating anemia with elevation of WBCs and neutrophilic count in cases with cholestasis[30]. Serum biochemical changes in cases of cholestasis included hyperbilirubinemia associated with a gradual rise of ALP, ALT, and GGT indicating cellular injury of the liver and biliary tract lining epithelium, they were recorded by different studies [2, 6, 31]. Serum potassium level showed a significant decrease which may be due to severe vomiting or diarrhea associated with cholestasis. The present work regarding miRNA-122 biomarker showed high significant elevation cholestasis affrected cats compared with healthy cats. These findings came to an agreement with the recent findings [32]. These results prove that cholestasis can lead to cellular injury of liver cells and biliary system lining epithelium causing their inflammation augmented with rise in leukocytic and neutrophilic counts.

Conclusion

The present study sheds light on feline cholestasis as a common cat hepatopathy. The recorded feline hepatopathy was about 26 % of diseased cats admitted to teaching veterinary pet animal hospital, faculty of veterinary medicine, Cairo University, Egypt. Feline cholestasis shared 25% of hepatopathies. Its occurrence was most recorded in mixed breeds, at ages over 3 years, tom cats, and during the spring season. Also, free feed access and fresh-type feed followed the same manner. From examination and laboratory results, feline cholestasis led to hepatocellular injury expressed by concomitant elevation of liver enzymes, bilirubin, and miRNA-122 levels which were specific and early predictors of cellular injury and inflammatory reactions of varying degrees. This made it of valuable clinical significance in the diagnosis of cholestasis.

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Conflict of interest: None

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Author`s contribution: OMA collected data and samples and performed the physical, hemato-biochemical examination, HA designed the entire protocol of our study and described the results, and discussed them, MEA performed the ultrasonographic examination and collected data. All authors drafted the manuscript, reviewed it, and approved the last version of the manuscript.

Ethical of approval

All study procedures were approved by the Institutional Animal Care and Use Committee, Faculty of veterinary medicine, Cairo University with approval number (Vet-CU-IACUC No. 18042024907)

TABLE 1. **Primers and probes used in SYBR-Green real time PCR.**

Gene	Primer nucleotide sequence (5'-3')	References
U6 (housekeeping)	GCTTCGGCAGCACATATACTAAAAT	**[19]**
MiRNA-122	CGCTTCACGAATTTGCGTGTCAT Gcgagcacagaattaatacgac Tggagtgtgacaatggtgtttg	**[20]**

TABLE 2. Cyclic conditions for SYBR-Green real-time PCR according to Quantitect® SYBR-Green PCR Kit. PCR:

Gene	RT.	1ry D.	Amplification (40 cycles)			Dissociation curve (1 cycle)		
			2ry D.	A. Optics on	E.	2ry D.	A.	Final D.
U6 (housekeeping)	50˚C 30 min.	94˚C 15 min.	94˚C 15 sec.	60˚C 30 sec.	72˚C 30 sec.	94˚C 1 min.	60˚C 1 min.	94˚C 1 min.
MiRNA-122				55˚C 30 sec.			55˚C 1 min.	

Polymerase chain reaction, RT.: reverse transcription, A: annealing, E: extension, D: denaturation.

TABLE 3. Incidence of feline cholestasis in cases admitted to the teaching veterinary hospital, faculty of veterinary medicine, Cairo University for one year period from June 2023 to June 2024.

Group	Apparently healthy cats	Diseased cats		
Sub-group	-	**Hepatopathies**		**Other health problems**
Common causes	-	Feline cholestasis	Other causes of hepatopathies	-
No. (%)	231(32.1%)	32 (25%)	96 (75%) 128 (26.2%)	360 (73.8%)
Total diseased no.	-	488 (67.8%)		
Total no.		719 (100%)		

TABLE 4. Risk factors associated with feline cholestasis through one year study period (Jul. 2023-Jul. 2024).

Risk factor	Category	Cholestasis 32 cases (100%)	
		Number of cases	Percentage
Breed	Persian	14	43.7 %
	Mixed breeds	15	46.8 %
	Siamese	1	3.1 %
	Himalayan	2	6.2 %
Age	Less than 2 years	9	28.1 %
	2-3 years	11	34.3 %
	More than 3 years	12	37.5 %
Sex	Tom cats	18	56.2 %
	Queens	14	43.7 %
Season	Summer	3	9.3 %
	Autumn	10	31.2 %
	Winter	8	25 %
	Spring	11	34.3 %
Feeding program	Free access	30	93.7 %
	Scheduled	2	6.2 %
Diet type	Dry food	9	28.1 %
	Soft canned food	0	0 %
	Fresh food (wet food)	12	37.5 %
	Mixed type food	11	34.3 %

TABLE 5. Physical examination parameters in cats affected with cholestasis.

Physical parameters	Control group	Feline cholestasis
Respiration rate (Time/min)	47.7± 2.44	87 ± 2.87***
Pulse rate (Pulse/min)	156 ± 2.22	189 ± 2.82***
Rectal temperature (°C)	38.6 ± 0.07	38.7 ± 0.12

***P≤0.001

TABLE 6. Hematological parameters in cats with cholestasis compared with apparently healthy cats.

Hematological parameters	Control group	Feline cholestasis
PCV (%)	38.3 ± 1.18	33.8 ± 1.62*
Hb (g/dl)	13.6 ± 0.56	11.8 ± 0.55*
RBCs count (×10^6 /µl)	8.9 ± 0.33	7.43 ± 0.34**
MCV (Fl)	43 ± 0.78	45.7 ± 1.23
MCH (Pg/l)	15.2 ± 0.42	16.1 ± 0.51
MCHC (%)	35.3 ± 0.88	35.6 ± 1.09
WBCs count (×10^3 cell/ µl)	10.6 ± 0.90	14.7 ± 1.60
Absolute neutrophils (×10^3 cell/ µl)	5.9 ± 0.45	8.6 ± 0.66**
Absolute lymphocytes (×10^3 cell/ µl)	3.3 ± 0.48	5.1 ± 1.08
Absolute monocytes (×10^3 cell/ µl)	0.35 ± 0.12	0.46 ± 0.11
Absolute eosinophils (×10^3 cell/ µl)	0.61 ± 0.13	0.37 ± 0.06
Absolute basophils (×10^3 cell/ µl)	0.23 ± 0.04	0.03 ± 0.01***
Platelet count (×10^3 cell/ µl)	343.6 ± 19.0	336.9 ± 31.2

*P≤0.05, **P≤0.01 and ***P≤0.001

TABLE 7. Serum biochemical parameters and hepatocyte-derived biomarker (miRNA-122) expression in cats suffered from cholestasis compared with healthy cats.

Parameter		Control group	Feline cholestasis
Serum parameters	Total proteins (g/dl)	7.74 ± 0.51	7.4 ± 0.41
	Albumin (g/dl)	3.2 ± 0.14	3.2 ± 0.18
	Globulins (g/dl)	4.6 ± 0.41	4.1 ± 0.30
	A/G ratio	0.83 ± 0.11	0.97 ± 0.13
	AST-GOT (IU/L)	40.1 ± 4.00	79.2 ± 8.22***
	ALT-GPT (IU/L)	67 ± 5.94	173.2 ± 29.5**
	ALP (IU/L)	41.5 ± 4.83	174.4 ± 26.2***
	GGT (IU/L)	3.4 ± 0.66	51.6 ± 11.4***
	T. bilirubin (mg/dl)	0.18 ± 0.02	1.6 ± 0.27***
	D. bilirubin (mg/dl)	0.08 ± 0.01	1 ± 0.17***
	BUN (mg/dl)	26.8 ± 2.88	40.7 ± 5.49
	Creatinine (mg/dl)	1.09 ± 0.05	1.8 ± 0.46
	Amylase (IU/L)	836.6 ± 50.5	557 ± 37.9***
	Lipase (IU/L)	461.9 ± 45.3	290 ± 17.8***
	Sodium (mEq/L)	135 ± 5.10	140.5 ± 5.2
	Potassium (mEq/L)	3.9 ± 0.36	2.9 ± 0.25*
	Chloride (mEq/L)	98.2 ± 4.19	95.5 ± 4.11
	Triglycerides (mg/dl)	66.3 ± 7.37	55.1 ± 4.58
	T. cholesterol (mg/dl)	134 ± 8.88	146.3 ± 15.25
	Glucose (mg/dl)	115.6 ± 3.06	108.2 ± 3.94
Hepatic biomarker miRNA-122	miRNA-122 (Fold expression)	1 ± 0.00	2.3 ± 0.19***

*P≤0.05, **P≤0.01 and ***P≤0.001

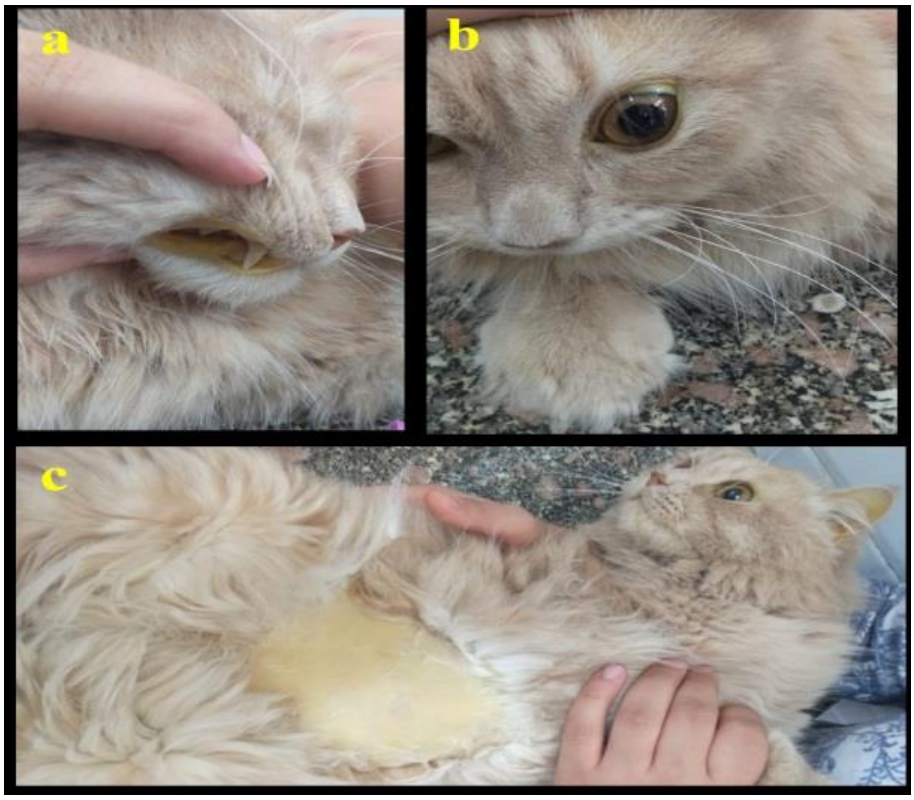

Fig. 1. Clinical signs associated with feline cholestasis. a: severe yellow discoloration of oral mucous membrane in four years old tom cat with cholestasis. b: icteric episcleral and conjunctiva mucous membrane. c: yellowish discoloration of skin (jaundice).

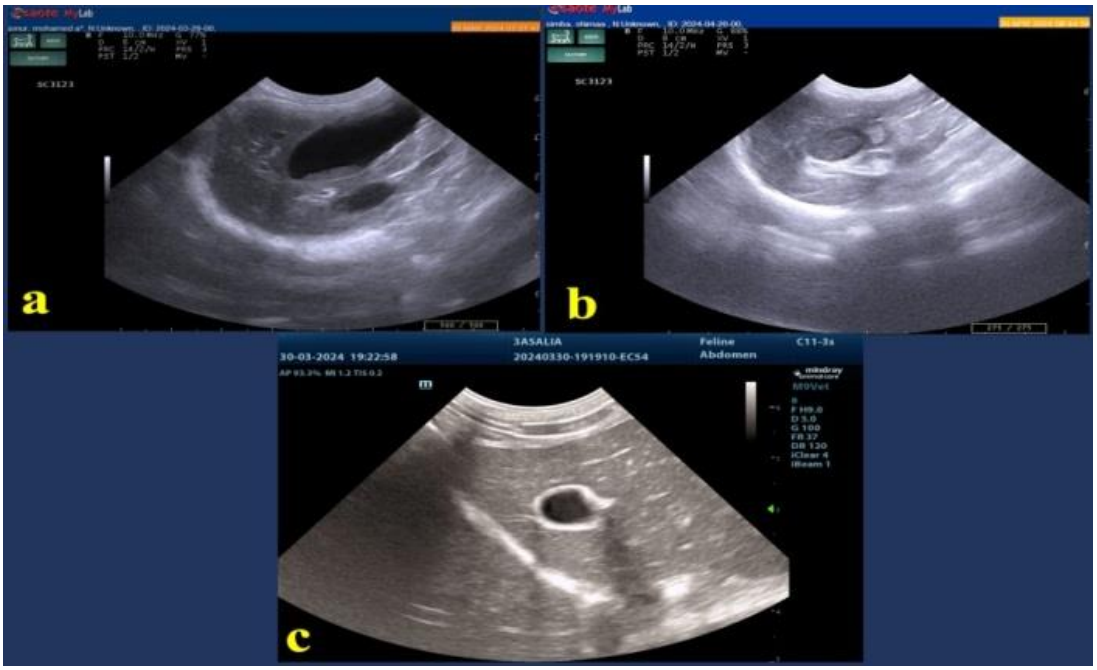

Fig. 2. Ultrasonographic views of liver in case of feline cholestasis. a: showed hyper-echogenic biliary sludge with hypo-echogenic hepatic parenchyma in a three years old mixed breed tom cat. b: hyper-echogenic severe degree of biliary sludge in 4 years old Persian tom cat. c: severe cholecystitis with thickened hyper-echogenic gall bladder wall in 3 years old mixed breed tom cat.

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دراسات سريرية وتشخيصية على الركود الصفراوي في القطط وأهمية الميكرو أرإن إيه 122 كمؤشر حيوي

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الملخص

شملت الدراسة الحالية 719 قطة تم إدخالها إلى المستشفى البيطري التعليمي، كلية الطب البيطري، جامعة القاهرة، والعيادات الخاصة. كانت جميع القطط من سلالات وأجناس وأعمار مختلفة. تم تقسيم هذا العدد إلى 488 قطة مريضة و231 قطة سليمة ظاهريًا. تم تسجيل 128 حالة (26.2%) بحالات مرضية مختلفة من العدد المصاب. تم الكشف عن الركود الصفراوي القططي في 32 حالة (25٪). جمعت الدراسة الحالية 52 قطة (32 حالة مصابة بالركود الصفراوي و20 قطة سليمة). أجريت فحوصات سريرية وموجات فوق صوتية وكيميائية حيوية شاملة بالإضافة إلى تقييم miRNA-122 المشتق من خلايا الكبد على جميع الحيوانات. تم اكتشاف الركود الصفراوي في القطط في الغالب في القطط المختلطة التي يزيد عمرها عن 3 سنوات، وأكثر في قطط الذكور مقارنة بالإناث. وكانت العلامات السريرية الأكثر تسجيلاً هي اصفرار الأغشية المخاطية، واصفرار الجلد، وفقدان الشهية، وآلام البطن القحفية، والقيء، والإسهال. وكانت الأسباب المسجلة للركود الصفراوي هي الحمأة الصفراوية، والتهاب المرارة، والاعتلال الدهني الكبدي، والتهاب الأقنية الصفراوية. وأدرجت أيضا نتائج وصفية بالموجات فوق الصوتية. بالنسبة للقطط المصابة بالركود الصفراوي، أظهر الفحص البدني ارتفاعًا ملحوظًا في معدلات التنفس والنبض. أظهر تقييم أمراض الدم فقر دم كبير مع ارتفاع العدد المطلق للعدلات. كشفت الكيمياء الحيوية في الدم عن ارتفاع كبير في جميع إنزيمات وظائف الكبد والبيليروبين. كما تم تسجيل انخفاض ملحوظ في مستويات البوتاسيوم. سجل التعبير الحيوي الكبدي (miRNA-122) زيادة كبيرة في القطط المريضة مما يشير إلى وجود علاقة وثيقة بين الركود الصفراوي والإصابة الخلوية الكبدية الصفراوية. كان Mi-RNA-122 مؤشرًا محددًا ومبكرًا للتفاعلات الالتهابية بدرجات متفاوتة. وهذا ما جعله ذا أهمية سريرية كبيرة في تشخيص الركود الصفراوي في القطط.

الكلمات الدالة: الركود صفراوي في القطط، نسب الإصابة، التصوير بالموجات فوق الصوتية، الكيمياء الحيوية للدم، ميكرو أرإن إيه 122.