фунгистатикалық тиімділікке ие екендігі анықталды, ал Клотримазол препараты тиімдірек болды. *T. benhamiae* №19 және №20 штамдары амфотерицин мен флуконазол препараттарына жоғары төзімділікке ие. Автор мысықтардың Дерматофитозының жаңа қоздырғышы *Trichophyton benhamiae* саңырауқұлаққа қарсы препараттарға сезімталдығына талдау жүргізу кезінде нәтижелердің сенімділігін алу үшін АГВ бейтарап ортасын қолдану ұсынылады.

Кілт сөздер: дерматофиттер, трихофитоз, *Trichophyton benhamiae*, мысықтар, фунгицидтік препараттар, сезімталдық, изолят.

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SENSITIVITY ANALYSIS OF A NEW PATHOGEN OF CAT DERMATOPHYTIA *TRICHOPHYTON BENHAMIAE* TO FUNGICIDAL DRUGS

Abstract.

Trichophyton benhamiae is a zoophilic dermatophyte known as one of the new pathogens of animal and human dermatophytosis.

Awareness of the nature of susceptibility and resistance to common anti-dermatophytic drugs is an important step towards taking appropriate therapeutic measures and reducing the duration of the disease and adjusting the costs of the therapeutic process. To date, no large-scale studies of the activity of antifungal agents in vitro against *T. benhamiae* isolates have been conducted. Isolates *T. benhamiae* №. 19 and *T. benhamiae* № 20 were tested against five common antifungal agents: clotrimazole, ketoconazole, fluconazole, amphotericin, nystatin, on different nutrient media: Saburo medium and AGV medium. As a result of the study, it was found that clotrimazole and ketoconazole drugs had high fungistatic efficacy on *T. benhamiae* strains №. 19 and № 20, and clotrimazole was more effective. *T. benhamiae* strains № 19 and № 20 are highly resistant to the drugs amphotericin and fluconazole. The author recommended to obtain the reliability of the results when analyzing the sensitivity of a new pathogen of cat dermatophytosis *Trichophyton benhamiae* to antifungal drugs, it is recommended to use a neutral AGV medium.

Key words: dermatophytes, trichophytia, *Trichophyton benhamiae*, cats, fungicides, sensitivity, isolate.

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CLINICAL AND HEMATOLOGICAL INDICATORS OF *DIFFERENTIAL DIAGNOSIS OF LIVER DISEASES IN DOGS*

Abstract.

The task of work was to study the differential diagnosis of spontaneous cases of hepatodystrophy and cirrhosis in dogs based on the results of the study of the clinical condition, changes in hemocytopoiesis and biochemical blood parameters,since liver pathology in dogs is quite common and leads to the loss of health of pets.

Dogs suffering from hepatodystrophy were depressed, vomiting, diarrhea were observed, some animals had slight hepatomegaly and pain in the liver area. Violation of the protein-synthesizing function of the liver was manifested by hypoalbuminemia, pigmentary – by an increase in the amount of total bilirubin and the appearance of conjugated. The urine–forming function of the liver and the filtering function of the kidneys also underwent changes. The activity of AST, ALT, GHT, LDH probably increased, the activity of XE decreased, that is.

Clinically, in dogs with cirrhosis, depression of the general condition, decreased appetite, and in some cases, jaundice of the conjunctiva were observed, but ascites and bradycardia were the most typical. One of the important general clinical indicators is the hemoglobin content, which was reduced in liver cirrhosis. If with hepatodystrophy the content of total protein increases or remains unchanged within the maximum norm, then it decreases in dogs with liver cirrhosis. The amount of albumin in liver cirrhosis was half as much as in hepatodystrophy, and in no case did it exceed 23.2 g/l, and in hepatodystrophy was at least 27.5 g/l.

If the activity of AST, ALT, and LDH increased by 1.5–2.2 times in hepatodystrophy, it did not exceed the normal range in liver cirrhosis. Changes in GHT activity are more informative: in cirrhosis it increased threefold, and in hepatodystrophy – twice. The most informative are changes in XE. Its activity in dogs with cirrhosis is sharply reduced and in no case did it exceed 21.4 μkat/l, while in hepatodystrophy it was at least 23.8 μkat/l.

Therefore, for the differentiation of hepatodystrophy and cirrhosis, the following indicators have the greatest diagnostic value: in cirrhosis – abdominal ascites, hypoproteinemia, a significant decrease in albumin content and XE activity, and in hepatodystrophy – normo- or hyperproteinemia, slight hypoalbuminemia and increased activity of cellular enzymes (AST, ALT , LDH).

Key words: dogs, liver diseases, hepatodystrophy, cirrhosis, diagnostic methods, erythrocytes, leukocytes, indicator enzymes, albumins, bilirubin.

Introduction.

Thanks to the experience of scientists over many years, as well as the development of modern technologies, the diagnosis of internal diseases has become easier and the study of their pathogenesis has deepened. Despite the large number of biochemical, instrumental and other methods of research, the practitioner of veterinary medicine still has certain difficulties in diagnosing a sick animal, since a significant number of internal non-infectious diseases do not have pathognomonic symptoms, and the same symptoms are found in different diseases. Therefore, the recognition of diseases and their differential diagnosis require further study.

Liver diseases are common among small pets. According to researchers, liver pathology in the structure of internal diseases ranges from 5 to 50.8 % [1, p. 20; 2, p. 35; 3, p. 640]. Hepatitis, hepatodystrophy, liver abscesses, cirrhosis, cholecystitis and gallstone disease are diagnosed. According to J. H. Poldervaart et al. (2009) [4, p. 77], the structure of liver diseases in dogs is: hepatitis – 18 %, blood stasis – 9.1 %; primary tumors – 3.8; portosystemic shunts – 5.7; liver cyst – 5.6; fibrosis – 4.1; lipidosis – 3.9; cirrhosis – 2.3; metastasizing tumors – 13.9; other liver diseases – 33.6 %. According to the results of laboratory studies, hepatodystrophy is diagnosed in 50.8 % of dogs of service breeds [2, p. 37], according to other data – 30–40 %, and liver cirrhosis – in 34.7 % [5, p. 60; 6, p. 43; 7, p. 213; 8, p. 90].

In the conditions of large cities, liver damage is caused by feeding dogs insufficient in terms of energy and essential amino acids (methionine) and vitamins (tocopherol), often by excessive feeding, stress. In addition to alimentary etiology, hepatitis of infectious etiology is quite common in dogs [9, p. 1020; 10, p. 988; 11, p. 1335; 12, p. 838].

Liver pathology in dogs can occur secondary to kidney diseases, heart failure, disorders of protein, fat or carbohydrate metabolism, anemia, hypovitaminosis, gastroenteritis, infectious (leptospirosis, parvovirus enteritis, plague, etc.) [12, p. 839] and invasive (toxocariasis, hookworm, coccidiosis, babesiosis) diseases [13, p. 185; 14, p. 616; 15, p. 144; 16, p. 80; 17, p. 122].

Liver pathology (hepatitis or hepatodystrophy) that occurs secondarily should be taken into account in the complex pathogenetic therapy of sick dogs. With a protracted course and chronic intoxication, hepatitis and hepatodystrophy are complicated by the development of liver cirrhosis, and the driving mechanisms for this are blood and bile stasis, dystrophy and necrosis of hepatocytes [16, p. 82; 17, p. 125; 18, p. 190]. Changes due to cirrhosis are irreversible, therefore it is important and relevant to differentiate this disease from others – hepatodystrophy or hepatitis. For this purpose, we studied dogs with spontaneous cases of hepatodystrophy and cirrhosis. After the clinical examination, a laboratory blood test was performed.

Materials and methods.

The work was carried out in the clinic of Bila Tserkva National Agrarian University in the Research Institute of Internal Diseases of Animals. Spontaneous hepatodystrophy was studied in 12 dogs, and liver cirrhosis in 8. The age of the dogs was 2–4 years. Basically, these were German and Caucasian shepherds. After clinical examination of the dogs, hemocytopoiesis indicators were determined in the blood serum in the laboratory by generally accepted methods, as well as the content of bilirubin (according to Iendrashyk), total protein (refractometrically), protein fractions (nephelometrically), transaminase activity (by the Reitman and Frenkel method), alkaline phosphatase – LF (set reagents of the company "SIMKO Ltd"), gamma glutamyltransferase – GHT (according to Szazs), lactate dehydrogenase – LDH (by the method of Savel, Tovarek) and cholinesterase (photometrically using the substrate acetylcholine chloride).

Results and discussion.

In dogs suffering from spontaneous hepatodystrophy, we did not observe any special changes in the clinical condition. However, all of them were depressed, had impaired appetite, anemic conjunctiva, vomiting, diarrhea, hepatomegaly and pain in the liver area were observed in 2 animals.

Indicators of hemocytopoiesis were within physiological fluctuations, although anemia (hemoglobin was 95.0 g/l) and leukocytosis (number of leukocytes – 13.6 g/l) were observed in one dog, which indicates a protective response of "white blood" cells to intoxication .

The number of erythrocytes in dogs with signs of hepatodystrophy had a tendency to decrease, which indicates suppression of bone marrow function due to intoxication, however, the significant volume of each erythrocyte makes it possible to rationally supply tissues with oxygen, as their total respiratory surface to some extent compensates for the tendency to oligocythemia. Hematopoiesis in dogs must meet the high demands of the body to provide it with oxygen both at rest and during periods of excessive physical exertion [7, p. 213].

A fairly high level of hemoglobin in the blood of dogs with a moderate number of erythrocytes is an indicator that its concentration in each erythrocyte is significantly higher, compared to animals of other species. HGE in sick dogs averaged 26.3±1.4 pg and did not differ from the indicator in healthy dogs. Decrease by 13.8 % ($p<0.05$) of hemoglobin content and by 4.3 % ($p<0.05$) of hematocrit value in the studied animals indicates the initial stages of anemia during intoxication. The average volume of erythrocytes did not differ significantly from the value of clinically healthy dogs.

In the biochemical study of blood serum, the average content of total protein had a tendency to increase, although it was within the normal range.

In dogs suffering from hepatodystrophy, the evaluation of the protein synthesizing function of the liver showed a decrease of 14.9 % ($p<0.05$) in the amount of albumin in the blood serum, and this is a typical indicator of its pathology. The share of albumins in the total amount of protein

decreased by 8.8 %, and it amounted to 40.0±1.9% ($p<0.05$). The tendency to hyperproteinemia can be explained by the increase in globulins. A decrease in the albumin-globulin ratio by 30.2 % ($p<0.001$), compared to clinically healthy dogs, causes the opposite direction of changes in albumins and globulins.

Another specific function of the liver, in addition to the synthesis of albumins, is participation in the exchange of bilirubin. Impaired liver pigment function in canine hepatodystrophy patients is indicated by the fact that the average total bilirubin content was probably 3.4 times higher than in clinically healthy individuals ($p<0.01$), although it did not exceed the maximum limit of physiological fluctuations, which , according to the literature [7, p. 212], is 5.4 μmol/l. Conjugated bilirubin was also found in blood serum in the amount of 1.1±0.1 μmol/l, which is 28.9 % of the total. This is due to the fact that the excretion of conjugated bilirubin into the lumen of bile capillaries is delayed due to dystrophic changes in hepatocytes, as their energy capabilities are insufficient for this. Therefore, bilirubin bound to glucuronic acid enters the bloodstream, which is an indicator of intrahepatic cholestasis.

The amount of urea in blood serum with hepatosis in dogs was reduced by 24.5 %, but its average value did not exceed the normal range.

Damage to the liver in patients with hepatosis in dogs causes a violation of the functional state of the kidneys, in particular, a decrease in their filtration function, as indicated by a 1.6-fold increase in the content of creatinine in the blood serum ($p<0.05$). Some dogs had creatinine more than 200.0 μmol/l. Determining the concentration of creatinine in the blood is especially important in dogs, because they, compared to other domestic animals, most often suffer from various nephropathies [2, p. 35; 16, p. 82]. Therefore, a decrease in the level of urea and an increase in creatinine indicate a violation of the functional state of the liver and kidneys, from which we can conclude about the development of hepatorenal syndrome in dogs, when the kidneys are affected along with the liver.

For the diagnosis of hepatodystrophy, the methods of determining the activity of liver-indicating enzymes in the blood serum of dogs turned out to be the most informative and indicative. Thus, the activity of aspartate aminotransferase (AST) was increased by 2.0 times ($p<0.001$), and alanine aminotransferase (ALT) by 2.2 times ($p<0.001$), compared to clinically healthy dogs, and this indicates the elimination of enzymes in blood with cellular destruction of hepatocytes.

Regarding the determination of the ratio of AST and ALT activity (De-Ritis coefficient), its decrease may be due to the destruction of the cell membrane. ALT is found only in the cytoplasm, and its increase indicates damage to subcellular organelles – mitochondria, where aspartic transferase is localized (in addition to the cytoplasm). This indicates severe damage to hepatocytes. In dogs with hepatodystrophy, the De-Ritis coefficient was 0.82±0.02, i.e. it was reduced by 8.9 %, compared to clinically healthy dogs.

The activity of the cytosolic enzyme – lactate dehydrogenase (LDH) increased 1.5 times ($p<0.05$) compared to clinically healthy dogs. This indicates cytolysis syndrome, and gamma-glutamyltransferase (GHT) – 2.0 times ($p<0.01$). This is a consequence of the development of intrahepatic cholestasis. The activity of alkaline phosphatase (AL) did not change.

In the diagnosis of liver pathology, the activity of cholinesterase (CHE) is important. In conditions of parenchymal damage, the synthesis of XE decreases, so its activity in the blood was reduced by 1.6 times, compared to clinically healthy dogs ($p<0.01$).

Therefore, dogs with hepatodystrophy developed a syndrome of cytolysis and cholestasis (bile secretion disorder with congestion), which is evidenced by an increase in the activity of indicator enzymes and the presence of conjugated bilirubin in the blood serum. Minor hypoalbuminemia, hypoazotemia, and creatinineemia were also observed.

Liver cirrhosis is the final stage of chronic hepatitis.

Clinically, in dogs with signs of cirrhosis, depression of the general condition, decreased appetite, bradycardia (63.8±0.4 beats/min), anemia of the mucous membranes were observed, in

some – jaundice of the conjunctiva, itching, body temperature was within the normal range, feces masses had a yellow-gray color, the volume of the abdomen was increased. During the puncture, straw-colored fluid without sediment was released from the abdomen. So, a typical symptom of cirrhosis was ascites.

Hematopoiesis in dogs was characterized by a probable ($p<0.05$) decrease in the number of erythrocytes by 18.5 %, and oligocythemia was observed in three dogs and the number of erythrocytes in them was only 4.2±0.03 T/l.

The group average number of leukocytes in dogs with signs of cirrhosis tended to increase. Oligochromemia was noted in three animals – hemoglobin was 93.3±0.3 g/l. It developed more intensively than oligocythemia, so the average content of hemoglobin in each erythrocyte in dogs with signs of cirrhosis decreased by 15.7 %. The average amount of hemoglobin in dogs with liver cirrhosis was lower than the average norm ($p<0.001$) and that which was established by us and is also given in the literature [19, p. 40; 20, p. 114].

The hematocrit value in general in the group was significantly reduced (by 9.3 %), which was caused not only by oligocythemia, but also by a pronounced tendency to decrease ($p<0.05$) average volume of erythrocytes. The combination of oligocythemia with a tendency to microcytosis causes a decrease in the total respiratory surface of erythrocytes by 21.2 %. Therefore, in dogs with cirrhosis of the liver, a whole complex of hematopoiesis disorders develops: hemoglobin synthesis, erythrocytopoiesis is inhibited, erythrocyte saturation with hemoglobin decreases, the total respiratory surface of erythrocytes decreases, which in turn causes a negative impact on metabolism, in particular glycolysis, function and structure various internal organs, including the liver.

In the blood serum of dogs with signs of cirrhosis, the content of total protein was reduced by 1.3 times ($p<0.01$). This indicates severe dystrophic processes in the liver and a significant violation of its protein synthesis function. This is also indicated by a significantly reduced content of albumins in the total amount of protein in the blood serum of sick dogs, the amount of which did not exceed 20 g/l. With the development of hypoproteinemia, the share of albumins was reduced to 26.1±0.9 % of the total protein. The albumin-globulin ratio decreased from 0.96±0.05 in clinically healthy dogs to 0.35±0.02 in patients with cirrhosis (by 2.7 times; $p<0.001$).

A decrease in the content of albumins in the blood leads to a decrease in oncotic pressure, which is one of the reasons for the development of ascites [20, p. 112].

In dogs with cirrhosis, the amount of total bilirubin increased by 5.8 times ($p<0.001$), compared to clinically healthy dogs, which indicates a much more pronounced bilirubinemia than in hepatodystrophy. An objective interpretation of these changes is possible only taking into account the content of conjugated bilirubin (3.7±0.08 mmol/l) and its share in the total amount of pigment, where it was 56.9 %. Therefore, the main cause of bilirubinemia is a violation of the elimination of conjugated bilirubin in the lumen of bile capillaries due to a decrease in the elimination capabilities of hepatocytes and their damage due to cirrhosis. One of the reasons for the accumulation of conjugated bilirubin in the blood can be cholestasis, which occurs as a result of the growth of connective tissue. This causes an increase in the pressure in the bile capillaries and makes it difficult to eliminate it against the concentration gradient. The accumulation of conjugated bilirubin in the blood is the cause of the jaundiced color of the conjunctiva.

Significant hypoazotemia was observed in dogs with signs of cirrhosis, which indicates a decrease in the urea-forming function of the liver. The content of urea was reduced by 1.8 times ($p<0.01$). The synthesis of urea is associated with the expenditure of a significant amount of energy (3 molecules of ATP are spent to obtain 1 molecule). When hepatocytes are damaged, when the formation of ATP is sharply reduced, the synthesis of urea is disturbed.

However, the compensatory capabilities of the liver for urea synthesis are quite significant, so its production decreases only with severe damage to hepatocytes [20, p. 115], which is observed in dogs with cirrhosis. It is obvious that ammonia, which is not completely neutralized in the liver,

enters the blood and penetrates into the cerebrospinal fluid, which causes the development of hepato-encephalic syndrome, which in sick dogs is manifested by depression and anorexia.

Violations of hematopoiesis and chronic intoxication lead to a violation of the filtering function of the kidney glomeruli, which is manifested in 25 % of dogs by an increase in the concentration of creatinine, so its average content was increased by 1.6 times ($p<0.05$), compared to clinically healthy.

When determining the activity of indicator enzymes for the liver, it was noted that AST and ALT were within the normal range, although in three dogs the enzyme activity was increased: AST – up to 442.5±36.7 ncat/l ($p<0.01$), ALT – 436.2±30.8 ncat/l ($p<0.05$). A slight increase in the activity of cytolytic enzymes in only three dogs could not affect the overall results, so their average activity by group did not change. The absence of their hyperfermentemia against the background of deep disorders of the albumin- and urea-synthesizing and bilirubin-secreting functions of the liver is a consequence of the replacement of parenchymal cells of the organ by connective tissue.

Severe damage to hepatocytes is indicated by a probable increase of 13.9 % in the De-Ritis coefficient ($p<0.05$), as this is a sign of increased activity of the mitochondrial fraction of AST. LDH activity was increased by 1.7 times, LF remained unchanged, and GGT increased by 3.0 times.

We also determined the activity of one of the secretory enzymes – cholinesterase (CHE), which is synthesized in hepatocytes, so it is an objective criterion of their function. The activity of XE was reduced by three times compared to clinically healthy animals ($p<0.001$). The degree of decrease in enzyme activity corresponds to the severity and prevalence of hepatocyte damage, that is, significant changes in the structure of the liver in the studied patients can be asserted.

So, in cirrhosis, ascites, oligochromemia, oligocythemia, significant bilirubinemia, hypoproteinemia, hypoalbuminemia, hypoazotemia, creatininemia, an increase in GGT and a decrease in XE activity were found, while the activity of cellular enzymes (AST, ALT, LDH, LF) remained unchanged.

Conclusions.

1. When evaluating the clinical condition of dogs with signs of hepatodystrophy, they noted depression, decreased appetite, anemic conjunctiva, the liver was slightly enlarged, and the area of percussion was painful. With cirrhosis, depression, decreased appetite, visible mucous membranes are anemic, in some animals the conjunctiva is slightly jaundiced. The development of cholestasis is manifested by skin itching, combing, eczema. A typical symptom is abdominal dropsy.

2. The evaluation of biochemical indicators of blood serum showed that in hepatodystrophy the content of total protein had a tendency to increase, and in cirrhosis this indicator was probably ($p<0.01$) reduced by 22.7 %. The number of albumins for liver cirrhosis is half as much as for hepatodystrophy. Both diseases increase the content of total and conjugated bilirubin in blood serum. With hepatodystrophy, the activity of AST, ALT, and LDH increased by 1.5–2.2 times, and with cirrhosis, it did not exceed the normal range. The activity of GHT changed in both diseases, but in liver cirrhosis, it increased threefold. XE activity in liver cirrhosis is sharply reduced.

References

1. DeMarle, K.B, Webster, CR.L., Penninck, D, & Ferrer,L. (2021). Approach to the Diagnosis of Hepatocutaneous Syndrome in Dogs: A Retrospective Study and Literature Review. J Am Anim Hosp Assoc. Jan 1; 57 (1): 15–25. doi: 10.5326/JAAHA-MS-7072.

2. Dykyi, O.A., Holovakha, V.I., Fasolia, V.P., & Soloviova, L.M. (2000). Informatyvnist okremykh pokaznykiv dlia diahnostyky patolohii pechinky i nyrok u sobak [Informativeness of individual indicators for diagnosis of liver and kidney pathology in dogs]. Visnyk Bilotserkivskoho derzhavnoho ahrarnoho universytetu. Bila Tserkva, vv. 11. pp. 32–37. (in Ukrainian)

3. Dos Santos, J.P., Lucina, S.B., da Costa, B.N., Olaguivel, KL.C., Tuleski, GL.R., & Sousa, M.G. (2021). Assessment of heart rate turbulence in dogs with myxomatous mitral valve disease. Open Vet J. Oct–Dec; 11 (4): 635–644. doi: 10.5455/OVJ.2021.v11.i4.13.

4. Poldervaart, J.H., Favier, R.P., Penning L.C. et al. (2009). Primary hepatitis in dogs: a retrospective review (2002–2006). J. Vet. Intern. Med. Vol. 23 (1). pp. 72–80.

5. Lokes, P.I., & Lokes-Krupka, T.P. (2014). Dyferentsiina diahnostyka khvorob pechinky u sviiskykh sobak i kotiv. [Differential diagnosis of liver diseases in domestic dogs and cats]. Visnyk Poltavskoi derzhavnoi ahrarnoi akademii. Poltava. no 1. pp. 58–61. (in Ukrainian)

6. Lucina, S.B., Sarraff, A.P., Wolf, M., Silva, VB.C., Sousa, M.G, & Froes, T.R. (2021). Congenital Heart Disease in Dogs: A Retrospective Study of 95 Cases. Top Companion Anim Med. Jun; 43: 100505. doi: 10.1016/j.tcam.2020.10050.

7. Malikova, A.I. (2020). Morfolohichni ta biokhimichni pokaznyky krovi sobak, khvorykh na hepatodystrofiiu. [Morphological and biochemical blood parameters of dogs with hepatodystrophy]. Stan ta perspektyvy vyrobnytstva, pererobky i vykorystannia produktsii tvarynnytstva. pp. 212–214. (in Ukrainian)

8. Michael, A.E., Case, J.B., Massari, F., Giuffrida, M.A., Mayhew, P.D., Carvajal, J.L., Regier, P. J., Runge, J.J., & Singh, A. (2021). Feasibility of laparoscopic liver lobectomy in dogs. Vet Surg. Jul; 50 Suppl 1: O89–O98. doi: 10.1111/vsu.13566.

9. Neo, S., Takemura-Uchiyama, I., Uchiyama, J., Murakami, H., Shima, A., Kayanuma, H., Yokoyama, T., Takagi, S., Kanai, E., & Hisasue, M. (2022). Screening of bacterial DNA in bile sampled from healthy dogs and dogs suffering from liver- or gallbladder-associated disease. J Vet Med Sci. Jul 25; 84 (7): 1019–1022. doi: 10.1292/jvms.22-0090.

10. O'Kell, A.L., Gallagher, A.E., & Cooke, K. L. (2022). Gastroduodenal ulceration in dogs with liver disease. J Vet Intern Med. May; 36 (3): 986–992. doi: 10.1111/jvim.16413.

11. Pena-Ramos, J., Barker, L., Saiz, R., Walker, D.J, Tappin, S., Hare, CH.Z., Roberts, M. L., Williams, T.L., & Bexfield, N. (2021). Resting and postprandial serum bile acid concentrations in dogs with liver disease. J Vet Intern Med. May; 35 (3): 1333–1341. doi: 10.1111/jvim.16134.

12. Rahman, S.A., Khor, K.H., Khairani-Bejo, S., Lau, S.F., Mazlan, M., Roslan, A., & Goh, S. H. (2021). Detection and characterization of Leptospira spp. in dogs diagnosed with kidney and/or liver disease in Selangor, Malaysia. J Vet Diagn Invest. Sep; 33 (5): 834–843. doi: 10.1177/10406387211024575.

13. Rybachuk, Zh.V. et al. (2020). Zmina deiakykh diahnostychnykh pokaznykiv funktsionalnoho stanu pechinky u sobak u razi vykorystannia FPD «Imunobakteryn-D». [Changes in some diagnostic indicators of the functional state of the liver in dogs in case of use of the "Immunobacterin-D" FPD]. Visnyk Poltavskoi derzhavnoi ahrarnoi akademii. no 1. pp. 182–188. (in Ukrainian)

14. Saunders, A.B. (2021). Key considerations in the approach to congenital heart disease in dogs and cats. J Small Anim Pract. Aug; 62 (8): 613–623. doi: 10.1111/jsap.13360.

15. Soloviova, L.M., Holovakha, V.I., & Utechenko, M.V. (2001). Kliniko-biokhimichni ta histolohichni zminy pechinky u sobak pry toksychnii hepatodystrofii. [Clinical, biochemical and histological changes of the liver in dogs with toxic hepatodystrophy]. Visnyk Bilotserkivskoho derzhavnoho ahrarnoho universytetu. vv. 18. Bila Tserkva. pp. 141–147. (in Ukrainian)

16. Timoshenko, O.P., Snopenko, O.S., Kibkalo, D.V., Korenev, M.I., & Maslak, Yu.V. (2021). Diahnostychna znachymist vymiriuvannia «kutykuliarnoho indeksu» u sobak za patolohii pechinky ta nyrok [Diagnostic value of "cuticular index" measuring in dogs with liver and kidney pathology]. Veterinary Science, Technologies of Animal Husbandry and Nature Management, 8, 78–84, DOI:10.31890/vttp.2021.08.11. (in Ukrainian)

17. Wilkinson, A., Panciera, D., DeMonaco, S., Boes, K., Leib, M., Clapp, K., Ruth, J., Cecere, T., & McClendon, D. (2022). Platelet function in dogs with chronic liver disease. J Small Anim Pract. Feb; 63 (2) : 120–127. doi: 10.1111/jsap.13342.

18. Soloviova, L.M. (2002). Efektyvnist likuvannia toksychnoi hepatodystrofii u sobak. [Effectiveness of treatment of toxic hepatodystrophy in dogs]. Visnyk Bilotserkivskoho derzhavnoho ahrarnoho universytetu. vv. 23. Bila Tserkva. pp. 187–193. (in Ukrainian)

19. Korenieva, Zh.B., Chebotarova, H. M., Holovanova, A.I., Lototskyi, V.I., & Kernova, M. P. (2018). Monitorynh porushennia funktsii pechinky u dribnykh tvaryn v suchasnykh ekolohichnykh umovakh. [Monitoring of liver dysfunction in small animals in modern environmental conditions]. Naukovyi visnyk veterynarnoi medytsyny. 1. pp. 38–44. (in Ukrainian)

20. Levchenko, V.I., Holovakha, V.I., Dykyi, O.A. & Soloviova, L.M. (2000). Deiaki aspekty patohenezu hepatodystrofii u sobak. [Some aspects of the pathogenesis of hepatodystrophy in dogs]. Visnyk Bilotserkivskoho derzhavnoho ahrarnoho universytetu. vv. 13 (2). Bila Tserkva. pp. 110–116. (in Ukrainian)

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ИТТЕРДІҢ БАУЫР АУРУЛАРЫНЫҢ ДИФФЕРЕНЦИАЛДЫҚ ДИАГНОЗЫНЫҢ КЛИНИКАЛЫҚ-ГЕМАТОЛОГИЯЛЫҚ КӨРСЕТКІШТЕРІ

Аңдатпа

Жұмыстың мақсаты клиникалық жағдайды, гемоцитопоэздегі өзгерістерді және қанның биохимиялық көрсеткіштерін зерттеу нәтижелері бойынша иттердегі гепатодистрофияның және цирроздың спонтанды жағдайларының дифференциалды диагностикасын зерттеу болды, өйткені иттерде бауыр патологиясы өте кең таралған және әкеледі. үй жануарларының денсаулығының жоғалуына.

Гепатодистрофиясы бар иттер депрессияға ұшырады, құсу, диарея байқалды, кейбір жануарларда - аздап гепатомегалия және бауырда нәзіктік. Бауырдың ақуыз синтездеу функциясының бұзылуы гипоальбуминемиямен, пигментті - жалпы билирубин мөлшерінің жоғарылауымен және конъюгацияланған билирубиннің пайда болуымен көрінді. Бауырдың зәр шығару қызметі және бүйректің сүзу қызметі де өзгерістерге ұшырады. ACT, ALT, GGT, LDH белсенділігі айтарлықтай жоғарылады, ChE белсенділігі төмендеді, яғни функционалды бауыр жеткіліксіздігі, цитолиз және холестаз синдромдары дамыды.

Клиникалық түрде циррозы бар иттерде жалпы жағдайының депрессиясы, тәбетінің төмендеуі, ал кейбір иттерде конъюнктивалық сарқырама байқалды, бірақ асцит пен брадикардия ең тән болды. Асцит бауыр циррозының дамуының соңғы кезеңі екені анық, сондықтан бұл ауруларды саралауға болатын жеке қан параметрлерін салыстыру қажет. Маңызды жалпы клиникалық көрсеткіштердің бірі – бауыр циррозында төмендеген гемоглобиннің мөлшері. Егер гепатодистрофия кезінде жалпы ақуыз мөлшері максималды норма шегінде жоғарыласа немесе өзгеріссіз қалса, бауыр циррозы бар иттерде ол төмендейді. Бауыр циррозында альбумин мөлшері гепатодистрофиядағыдан екі есе болды және ешбір жағдайда 23,2 г/л-ден аспады, ал гепатодистрофияда кемінде 27,5 г/л болды.

Егер гепатодистрофияда АСТ, АЛТ және LDH белсенділігі 1,5-2,2 есе артса, бауыр циррозында ол қалыпты шектен шықпады. GGT белсенділігінің өзгеруі ақпараттылығы жоғары: циррозда үш есе, ал гепатодистрофияда екі есе өсті. ChE өзгерістері ең ақпаратты болып табылады. Циррозы бар иттерде оның белсенділігі күрт төмендейді және ешбір жағдайда 21,4 мккат/л-ден аспайды, ал гепатодистрофияда ол кемінде 23,8 мккат/л болды.

Сондықтан гепатодистрофия мен циррозды дифференциациялау үшін келесі көрсеткіштер ең үлкен диагностикалық мәнге ие: циррозда - абдоминальды тамшылар, гипопротеинемия, альбумин мөлшерінің және ChE белсенділігінің айтарлықтай төмендеуі, ал гепатодистрофияда - нормо- немесе гиперпротеинемия, гипоальбуминнің шамалы жоғарылауы. жасушалық ферменттердің белсенділігі (AST, ALT, LDH).

Кілт сөздер: иттер, бауыр аурулары, гепатодистрофия, цирроз, диагностикалық әдістер, эритроциттер, лейкоциттер, индикаторлық ферменттер, альбуминдер, билирубин.

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КЛИНИЧЕСКИЕ И ГЕМАТОЛОГИЧЕСКИЕ ПОКАЗАТЕЛИ ДИФФЕРЕНЦИАЛЬНОЙ ДИАГНОСТИКИ БОЛЕЗНЕЙ ПЕЧЕНИ У СОБАК

Аннотация

Задачей работы было изучение дифференциальной диагностики спонтанных случаев гепатодистрофии и цирроза у собак по результатам исследования клинического состояния, изменений гемоцитопоэза и биохимических показателей крови, поскольку патология печени у собак достаточно распространена и приводит к потере здоровья домашних любимцев.

Собаки, больные гепатодистрофией, были подавлены, наблюдались рвота, диарея, у отдельных животных – незначительная гепатомегалия и болезненность в области печени. Нарушение белоксинтезирующей функции печени проявлялось гипоальбуминемией, пигментной – увеличением количества общего билирубина и появлением конъюгированного. Мочевыделительная функция печени и фильтрационная – почек также претерпели изменения. Достоверно повысилась активность АСТ, АЛТ, ГГТ, ЛДГ, снизилась активность ХЭ, то есть, развивались синдромы функциональной недостаточности печени, цитолиза и холестаза.

Клинически у собак, больных циррозом, наблюдали угнетение общего состояния, снижение аппетита, у некоторых – желтушность конъюнктивы, однако наиболее типичными были асцит и брадикардия. Очевидно, что асцит является финальной стадией развития цирроза печени, поэтому необходимо сравнить отдельные показатели крови, по которым можно дифференцировать эти заболевания. Одним из важных общеклинических показателей является содержание гемоглобина, который при циррозе печени был понижен. Если при гепатодистрофии содержание общего белка повышается или остается без изменений в пределах максимальной нормы, то у собак, больных циррозом печени, он снижается. Количество альбуминов при циррозе печени было вдвое меньше, чем при гепатодистрофии, и ни в коем случае не превышало 23,2 г/л, а при гепатодистрофии было не менее 27,5 г/л.

Если при гепатодистрофии активность АСТ, АЛТ и ЛДГ повышалась в 1,5–2,2 раза, при циррозе печени не выходила за пределы нормы. Изменения активности ГГТ более информативны: при циррозе она повышалась в три раза, а при гепатодистрофии – в 2 раза. Наиболее информативными являются изменения ХЭ. Активность ее у собак, больных циррозом, резко снижена и ни в коем случае не превышала 21,4 мккат/л, тогда как при гепатодистрофии была не менее 23,8 мккат/л.

Следовательно, для дифференциации гепатодистрофии и цирроза наибольшее диагностическое значение имеют следующие показатели: при циррозе – брюшная водянка, гипопротеинемия, значительное уменьшение содержания альбуминов и активности ХЭ, а при гепатодистрофии – нормо- или гиперпротеинемия, незначительная гипоальбуминемия и повышение активности клеточных ферментов (*АСТ, АЛТ, ЛДГ).*

Ключевые слова: собаки, болезни печени, гепатодистрофия, цирроз, методы диагностики, эритроциты, лейкоциты, индикаторные ферменты, альбумины, билирубин.