UDC 636

HEPATIC STEATOSIS OF DAIRY COWS

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The article describes widely spread disease – hepatic steatosis of dairy cows in a full expanded form necessary for research by students and investigation by specialists of zoo veterinarian profile. The given description for the first time represents: exact diagnosis of disease and form of its manifestation; primary factors and emersion conditions and development of disease process; symptoms of manifestation and pathological-anatomical alterations; diagnosis and prognosis. The topicality of the given work is that authors in section "disease treatment" give the description of a new developed and implemented method of treatment and precaution of hepatosis of an animal with a help of injection hepatoprotector medication.

Key words: hepar diseases, hepatic steatosis, cow, injection hepatoprotector, precaution, treatment.

Hepatic steatosis (lipidosis, adipose degeneration) – is a disease characterized by changes of trophism and morphology of hepatocytes in consequence of energy exchange breakdown in an organism and infiltration of hepar tissues by lipids [30, 33, 35]. Along the current of disease one can distinguish sharp, subacute and chronic form. According to clinic-anatomical hepar state, genetically conditioned (or dependent) adipose degeneration manifests by atrophy or hypertrophy [33, 35]. Sharp adaptation breakdown of lipid-carbohydrate (energy) exchange, which clinically manifests directly in the last days of calving or in the first seven days after calving, appears to be the reason of sharp hepar adipose infiltration of genetically conditioned genesis. Fleshy and/or high yielder animals are more often amenable to emersion of hapar lipidosis in the beginning of lactation [8, 38, 51], so far as in the result of more intense lipid tissue mobilization and slow recovery of energy consumption functions from fore-stomaches [13, 14, 23, 24, 67], fat pools of the bodies are more wasted [19, 26, 38]. However, not all the animals which are undergone to this impact have hepatic steatosis, but it depends on adaptative-defence mechanisms of an organism [29, 34, 45, 54].

Cow hepatic steatosis (genetically dependent adipose degeneration, lipidosis), as a rule is a secondary process, it can be observed on the back of fore-stomach hypotonia predelivery, abomasums and bowels [39, 55]. Appliance of hormonal agents for expulsion of afterbirth, for cow productive semination and/or leaper of heifers in the age of 18^{th} months and also intense fattening of replacements refer to the next aggravating reasons, in combination with genetic pathology and hepar liability to dystrophy and also low adaptive capacity [52]. Up to the present moment nosogenesis has been poorly investigated, only one thing was evident that impaired fat metabolism contributed to disease emersion. Impaired fat metabolism in the hepatocytes and dystrophic changes development in the organ under the influence of higher mobilization from the lipid fat depos, which stand over in hepar in the composition of triacylglycerols are at the bottom of the given nosogenesis [14, 35, 65]. Therewith gluconeogenesis, lipogenesis, oxidizing enzyme activity, hepar barrier function, plasma protein synthesis are broken down [17, 35].

Dystrophic changes of liver pulp on the initial step of process development are accompanied by hepatocytes intumescense, disorder of segment balk structure and general hepatomegalia. Under these changes biliary tracts and portal vessels are compressed. Loss of stricken cells is accompanied by knot regeneration of survive segment remainders and accretion connective tissue elements on their place. Accretive knot regenerates squeeze the surrounding tissue, especially thin-walled veins, capillaries, lymphatic clefts and bile ducts that leads to blood supply reduction of unstruck cells. Therewith, emersion of communicating partitions between portal fields and the centre of segments increases. Hereafter, if the process takes a progressive character in consequence of disintegration

and resolution of parenchymatous cells great number, the size of the hepar decreases and symptoms of hepar fatty atrophy develop. In hard cases the cells which suffered from disintegration, can gradually be replaced by fibrinous connective tissue than so called hepatic cirrhosis develops [35].

Under hypertrophic alteration hepar significantly increases in the result of intense processes of connective tissue neoplasms not only inside segments, but also out of them. In the result of organ hypertrophy blood flow slows, bradyhemarrhea appears in the system of janitrix that leads to emersion of catarrhal process in biliary tracts and the digestive tract. Passive congestion in portal system of hepar and mesenterium vessels induces changes of physico-colloidal and buffer blood condition that leads to increase of permeability and fragility of circumferential blood-vessels. Destruction of a number of hepatocytes leads to disorder of protein synthesizing function of hepar. In the result of which a number of albumins, plasmozyme, fibrinogen and a lot of other ferment decreases in the blood serum, but gamma-globulin fraction increases. Interruption of hepar neutralizing function leads to intoxication growth in the organism and reduction of organism autarcesis to different infections. Parenchymal or obstructive jaundice develops due to hepatism and autointoxication.

In such cases emersion of dystrophic damages of hepar and also the other oragns and tissues complicates course of concomitant diseases and can become a reason of death. Nosogenesis is connected with lipidic metabolism tension in hepar of cows before calving and in the early phase of lactation, that leads to lipids accumulation in hepar in consequence of excessive mobilization of fat reserves. The volume of fat pools and ability to use them under the conditions of lactation dominant at that is considered as basic nosotropic steatosis factor and development of genetically conditioned hepar adipose degeneration of cows.

Intense selection of dairy cattle for increasing of milk yields led to the fact that genetic productivity potential at an early stage of lactation exceeds the ability of an animal to consume a fair number of fodders that forms the state of negative energetic balance [25]. If in the period of deep pregnancy consumption of energy and flexible elements of cows are determined by the speed of fetal growth then in fresh period – are determined by biosynthesis activity and component milk secretion from one side and inadequate exogenous income of nutrient substances and energy –from the other side [8, 15, 28, 47]. Herewith, intense use of fat and protein reserves for originating deficit covering is accompanied by so called “premilking” of the animals and a row of significant shifts in lipidic exchange [4, 5, 6, 57, 59, 68, 69, 77], defining the following productivity and state of health of high-producing cows [15, 28, 47, 60]. Excessive deposition of lipids in hepar parenchyma cells (predominantly triacylglycerols) disturbs its functions, such as gluconeogenesis, synthesis of animal starch, oxidation of fatty acids and etc., and predisposes the animals to a row of diseases – first of all to ketosis [8, 13, 14, 16, 17, 19, 21, 26]. Physiologic-genetic disposition of high-producing cows to pathologic mobilization of own organism supplies for milk production, in its turn leads not only to excessive fat, but in the consequence to protein and massive hepatic necrosis [17, 18, 19, 20, 25, 26, 27, 47, 52].

The latest researches of VFA (volatile fatty acids) concentration lability behavior in blood and in farding bag as the basic energy sources for ruminants depending on its motor function showed that in the end of pregnancy and at the beginning of lactation, functional ability of farding bag was significantly decreased [30, 36, 39], but condition indexes of cow body live weight had negative dynamics in the beginning of lactation [8, 48]. This circumstance gave explanation to the basic physiological reason of intermissions in the farding bag activity – the leading fore-stomach of ruminants. Physiological matter of this phenomenon lies in the fact that in the latest duration of gestation not only fetus intense growth in weight and size occurs, but also there is a lot of waters in the cow gravid uterus, which in combination influence on farding bag with physical compression and therefore restrict its sound movements [30, 39]. Nowadays, on the basis of these data one can make reasoned conclusion that energy gap in early period of lactation is connected not with lack of consumed fodder, but with insufficient (slow) function of farding bag, which begins to manifest two months before calving [36, 39]. Therewith in the beginning of high-producing cows lactation by means of tissue reserves almost half of energy consumption on milk components formation is provided according to some sources, more than 300g of protein and to 1000g of fat per 24 hours [19,

25, 26, 58]. According to the other observations high cow milk yield was provided except fodder energy by the way of mobilization of 2 kg of body fat per a day, that is, metabolic processes in fat tissue in this period of lactation are pointed to fatty acids mobilization [21, 47, 53, 70]. According to a variety of information [56, 71, 79], cows waste up to 60 kg of tissue lipids for the early period of lactation. Therewith reserve lipids intense mobilization besides positive effect under highly expressed degree also has its negative sides: it can restrain fodder consuming, depress fat synthesizing function of milk gland, and predispose cows to ketosis disease. Furthermore at this period energy efficiency using of reserve lipids is higher than fodder energy [15, 25, 28, 47]. Consequently, quantity and using of cow body fat appear to be critical variables for optimization of milk productivity, food consumption, reproduction and health [20, 33].

In its turn, in connection with this, basic hepar functions suffer: biliation and biliary excretion, synthesis of protein, urea and animal starch, toxin deactivation and etc. This disease is also conditioned by alteration of farding bag microorganisms species composition and serves as a reason of insuffucient or excessive cellulose digestion, protein decomposition and self-poisoning by decay products. The given disease is accompanied by decrease of live weight, milk yields [14], debility of joints, alteration of nervous and heart-vascular systems condition, indigestion [19, 25, 26, 64], metabolism [52] and leads to the general toxicosis.

Under the conditions of chronic disease hepatic cirrhosis is possible along with dystrophic alterations in hepar. Hepatic steatosis (or genetically conditioned form of adipose degeneration) is determined with a help of percussion [62], by increased boundaries of hepatic obtrusion. Diagnostic measurements of percussion boundaries according to Aliev are realized by horizontal line. At the same time it should be noted that hepatic alterations proceed with concomitant variations of forestomaches diastalsis (determined by auscultation established procedure), extreme emaciation (under visual check), and metabolic disease (under biochemical assay of blood and tests of biopsied hepar) [13, 17, 18, 19, 26, 31, 33].

The boundaries of hepatic obtrusion under fat hypertrophy of hepar are localized in the next limits: on 1st month of lactation from 13 to 9 rib; on 2-5th months of lactation from 12 to 9 rib; on 6-9th month of lactation from 11 to 8 rib; on 10-12th month after calving on 10from 8 rib [10, 11]. Besides the forms of percussion boundaries depending on calving also change according to course of reproductive cycle and the forms will be in form of half petal (lancet) slowly turning to extend rhombus and then turning to trapezium. These data of topographic organization hepar location along the whole current of reproductive cycle first of all are explained by biomedical indexes of lipidic exchange in hepar and blood [6, 7, 8, 42, 56], and also by pregnancy steps of fetus-bearing terms [11]. Depending on pregnancy terms and fetus location topography of hepar borders will alter in the following sequence: on 1-3rd month of pregnancy the borders will be from 12 to 9th rib; on 4-7th month of pregnancy – from 11 to 8 ribs; on 8-9 month – from 10 ribs to 8 ribs [9, 10, 31, 33].

General suppression, muscle weakness, sharp progressive syntexis on the back of production reduction, anorexia (belch and chew), upset of fore-stomaches (hypotonia and atony) and the digestive tract are marked as the general symptoms for sharp form of hepatic steatosis. Body temperature is normal or slightly decreased. In some cases hepar area hurts during palpation and percussion, but for the most part the increase of back percussion boundary is noticed. Sometimes one can find yellowness and asphyxia on mucous membranes and sclera of the eyes, and in other cases effusion of blood of various degree (from punctuate to extensive) and inclination to anemia (flexible, globulicidal) are determined [31, 33].

Additive of protein, high quantity of urobilin and indican (protein breakdown product) and sometimes – cholechromes are determined in stale. Not infrequently one can find crystal of leucine and tyrosine, speaking for hepar protein forming disorder, in hypostasis along with organic elements of nephrogenic. Death can occur under the acute type and quickly growing intoxication symptoms on the back of cardiovascular adequacy. Under the chronic process of hepatic steatosis clinical symptoms are less expressed. Such animals at first have nonspecific general symptoms: attenuation or syntexis does not progress and also general animal obesity, atony and hypotonia of fore-stomaches, stasis of feed masses in bible-bag, decrease of productivity and reproductivity

(propagation) are registered. There can be no biliousness syndrome. Ill-defined hemorrhage of mucosal, sclera, sometimes trophic ulcers and erosions on the skin are defined. Percussion boundary is increased under the hepar adult-onset obesity. Under atrophic obesity it is decreased. The course of a disease is fever-free, body temperature often declines to norm minimal boundaries [35].

High concentration of globulins, filtrate nitrogen, ammonia, serumal transaminase can be found in the patients' blood. Therewith concentration of albumins, fibrinogen and glucose significantly decrease. Hemophthisis (disorder of marrowy hematosis) and decrease of blood coagulability (deceleration of BSR) are marked [31, 33]. Pathological-anatomical clinical picture of hepar lipidosis is confirmed in the result of repeated necessary commission slaughter and prosection. The prosection should be conducted not earlier than 2 hours after mortality. Therewith the hepar on prosection has yellow or oatmeal (clayly) color and it is mildly dense or crumbly by-touch. Under hypertrophic form the hepar is significantly increased in size in comparison with norm its edges are rounded, capsule is strained, and the picture of lobulation is smoothed-out. Generally under the hepar palpation some divisions are squashed in hand or different size parts of crumbled hepatic tissue are detected [21, 25, 31, 33]. In rare cases the size of hepar is unchanged or decreased, consistence upon that is mildly dense. Kidneys of clayly colour with smudge crumbly surface of bast layer are often detected. During prosection blood is generally not congealed and in small volume. Mucous membrane of bowels are inflamed diffusely or partially, have ruby color, they are turgid and sometimes covered with viscous transparent slime, partially have noticeable, small hemorrhages. Erosions and ulcers are detected on the mucous membranes in hard cases [17, 18, 19, 26, 27, 31, 33].

During prosection of fallen and/or forcedly killed animals along with hepar pathology, farding bag side thinning can be determined. The given circumstance in its turn explains disorder of farding bag function, appeared in the result of fore-stomaches compression by fetus at the last month of pregnancy [8, 31, 33, 52]. The diagnosis is made according to the results of clinical research – increased hepatic obtrusion borders can be defined with a help of the percussion method, and hypotonia and atony of farding bag are defined by auscultation method [19, 26]. In addition subclinical blood indexes point to ketone bodies higher level and to decreased glucose and hemoglobulin level [52]. Under hepatic steatosis anamnestic information states dominant cow replacement at the first month after calving. Besides all animals have differential characteristic of fast syntexis [17, 18, 25, 27, 46]. Under blood biochemical analysis one can define high concentrations of unesteritied fatty acids (UNFA) and low concentrations of beta lipoproteins, but in hepar biopsied samples high tissues infiltration degree is defined by general lipids, generally by means of triacylglycerol fraction [31, 33]. Diagnosis is confirmed by the results of complex pathological-anatomical prosection of fallen and/or forcedly killed animals, and also by histologic examination of hepar collected samples [48]. Methods of toxicological analysis exclude reasons of toxic adipose degeneration by fodders or intravital research of biological and pathologic material [31, 33].

Forecasting characteristics of hepatic steatosis are following: acute type proceeds from 4 to 7 days; subacute type – proceeds from seven days to three weeks, and absence of active nosotropic therapy can lead to death of the animal or to forced slaughter and to rejection of high-producing cows from herd; hepar lipidosis chronic process does not lead ruminants to fatality, but without curative measures they can become the object of rejection according to productivity and reproductive power. Recently newly calved cows even with subclinical form of hepar obesity have an increased service period [13, 17, 32, 33]. Since lipidic hepar induces delay of normal estrus after calving at the begging of periodic ovarian activity, this increases the number of seminations to fertilization [19, 26, 32, 33]. Deficit glucose generated in hepar and metabolic imbalance of sex steroid – process which takes place directly in hepar are appeared to be immediate causes [32, 33, 76)]. In context one can find explanation of reproductive function interruption which as a rule more often occurs under cows high productiveness. In these cases an expressed carbohydrate (glucose) insufficiency and an excessive lipids mobilization take place that can disturb hepar functions and first of all glucose-synthesizing and steroid-metabolizing functions [10, 32, 33, 44, 72, 73, 74, 75, 77, 78]. Under the low concentration of glucose in blood its availability for peripheral tissues gets lower and necessary hypothalamo-pituitary-gonadial connections for reproductive function are defected [32,

33, 66]. From the other side cells of steatosis hepar cannot provide necessary metabolism and/or katabolism of sex steroids because of decreased functional activity that leads to reproductive function defection [14, 16, 22, 32, 33, 46].

Nowadays there is a row of complex treatments that include: nutrition dietary regimen, nosotropic and supportive care that in its turn influences on the general state of the organism and pointed to toxic metabolic product excretion from the organism, to fight with water deprivation, to normal work reconstruction of all organ systems [12, 37]. Correctly made diagnosis has a significant importance as disorder symptom of hepar function is accompanied by large catalogue of different aetiology and pathology diseases [17, 18, 27, 32, 33].

Nevertheless questions about treatment and prophylaxis of hepatic disorders are still appeared to be a topical problem for animals and their health safety including adult stock of cattle [3]. Firstly it is not always possible to determine the cause of hepatic steatosis orienting just only to transition period before and after calving. And in connection with this to remove and preclude manifestation of adipose (toxic or genetically conditioned) degeneration and hepar functional disturbance [17, 18]. Secondly, as a rule attending medical doctor receives the subclinical research results of biological material and fodders expertise very late. Thirdly complex therapy activity is aimed to toxins removal from the whole organism, bilis from the patient's hepar, pain relief, which in their turn no to the full reconstruct disturbed hepar functions and structural changes in the hepar cells [24, 32, 33].

In connection with upper discussed problems and on the assumption of requirements to treatment and prophylaxis, during creation and appliance of new ecologically clean medications from natural resources [2, 63], at the present time a new medication – hepar hydrolyzate is offered, it has antiseptic and antiphlogistic action, it improves and reconstructs lipidic-carbohydrate (energetic) metabolism, angiogenesis and appears to be a good antidote and antloxidant and most of all it is not an antagonist of remedies, applied in the complex therapeutic regimens and/or for hepatosis precaution, including vaccines [17, 18, 19, 21, 24, 25, 26, 27, 32, 33].

The advantage of the offered medication and method of its appliance consists in that this medication with a help of hepar percussion method allows to control clinical state of its sizes more executive so as results of subclinical (laboratory) analysis and more over of histologic study, as practical training shows, enter into farm in 7-14 days [32, 33, 41, 48, 50, 61]. The advantage of this medication appears to be that it consists of hepatic tissue amino acids of clinically healthy animals, received by hydrolytic way and that is why injection of it into organism allows to direct medicine to hepar selectively, so as genetically corrected dependence in ingredients usage, received from analogous tissues exists in the organism. Under hydrolytic degradation of hepar extract protein structures the abruption of long polypeptide chains of protein to amino acids takes place. In the process of decomposition protein loses its specific peculiarity, colloidal properties do not already have neither primary toxicity, nor antigenic and anaphylactic properties. However, amino acids have their belonging to the hepar tissue [40, 41].

Injection method of treatment and precaution of hepatosis, including adipose degeneration of cows, applied on the ground that under the hepar disease entrance of medicated product in combination with fodder and digestion of it will be ineffective because of ruminants digestive peculiarities, besides, as a rule an affected animal has no appetite. According to ruminants (cows, sheep, goats, camels ant others) digestive peculiarities, then entered to digestive system medicated product solution, containing amino acids, including indispensable, can be used by microorganisms and by the animal organism itself as nutritional ingredients, besides, it is impossible to exactly control the dose of injected medication to the organism in combination with fodder. Injection of hepar hydrolyzate solution contributes to faster and better diffusion process (penetration) into the hepar, than in combination with fodder, in the result of which regenerative processes and toxic materials fixation realize more actively [24, 32, 33, 41, 61].

Medical and prophylactic efficacy is reached by hepatoprotector remedy injection, subcutaneously, intramuscularly or intravenously one time in 24 hours. With medical goal newly calved cows with live weight 350-550 kg, which hepatic alterations proceed with concomitant variations of fore-stomaches diastalsis, general attenuation and metabolic disease, medication dose composes

20,0-40,0 ml, besides, 0,5 ml of dose is injected subcutaneously and 0,5 ml of dose is injected intramuscularly and simultaneously during 5-6 days in succession. During birth palsy the medication is injected every day intravenously in dose of 100-200 ml in delution 1:1 with 40% glucose to animals with obvious symptoms of rookery and also to avoid fatality [13, 17, 18, 24, 27], till a threat of compulsory slaughter is removed, and then according to the scheme of newly calved cows. For the purpose of precaution – nonmilking cows with weght 350-550 kg in dose 10,0-20,0 ml with interval 7-10 days, have in whole 5-7 injections [24, 32, 33, 35, 41, 43, 50, 61]. As a practice of broad implementation of treatment method and hepatosis precaution has shown, this hepatoprotector medication can be successfully used not only for hepatic steatosis treatment, but also for other varieties of hepatic disorders [17, 18, 32, 33, 35, 43, 49, 50, 61].

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August, 29, 2015