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Hypovitaminos A And D In Young Animals

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Abstract: To study the prevalence, economic damage and etiology of hypovitaminosis A and D in young animals in cattle farms of our republic , as well as methods of diagnosing, treating and preventing the disease.

Keywords : A hypovitaminosis , hemeralopia, rhodopsin, hypovitaminosis, metabolis

INTRODUCTION

Currently, vitamin preparations given to farm animals ensure the normal course of metabolic processes in their bodies. Vitamins are biologically active substances that ensure the normal course of biochemical processes in the body of animals.

Retinol deficiency (hypovitaminosis A) is a disease caused by vitamin A deficiency , manifested by increased branching and metaplasia of epithelial cells, deterioration of vision and reproductive properties, and stunted growth in young animals.

Causes. The amount of vitamin A or its provitamin A carotene in the diet does not meet the animal's needs. Endogenous hypovitaminosis A can be observed in hepatitis, liver cirrhosis, gastroenteritis, some infectious and invasive diseases, or chronic poisoning. Tocopherol and some other antioxidants, as well as the element zinc, are substances that protect vitamin A from degradation.

Development. In cattle, carotene from food travels through the blood to the liver and is converted to retinol there, while in all other animals this process occurs in the wall of the small intestine.

With a deficiency of vitamin A, hyperplasia and branching develop in the epithelium of the skin, lacrimal glands, conjunctiva, respiratory tract, digestive tract, and genitourinary organs, as well as in the mucous membranes The protective functions decrease, metaplasia and dystrophy of the epithelium of the ovaries and testicles occur, follicle atrophy, spermiogenesis is inhibited, hyperplasia and branching of the skin epithelium develop, and atrophy of the sweat and sebaceous glands develops. Damage to the epithelium of the genital organs can cause death of the embryo and miscarriage . Dryness of the cornea and decreased vision are specific signs. This is caused by blockage of the tear duct , inflammation of the conjunctiva, the formation of ulcers on the cornea and its softening.

It is known that retinol, the active form of vitamin A, combines with the opsin protein in the retina to form rhodopsin (visual purple) , which ensures the eye's adaptation to darkness. Vitamin A is a growth factor, and its deficiency leads to a decrease in collagen synthesis in bone tissue, resulting in bone dystrophy and stunted growth. A Vitamin deficiency is accompanied by a decrease in

the synthesis of sex hormones and adrenal cortex hormones . There are also suggestions that the stability of cellular and mitochondrial membranes decreases during the disease .

Symptoms. Clinical signs characteristic of all types of animals include roughening of the skin, loss of shine and elasticity of the hooves and horns, the appearance of folds, rashes and areas of hair loss on the skin , stunted growth and development of young animals, decreased resistance to disease, decreased adaptation to darkness (hemeralopia), lacrimation, conjunctivitis , xerophthalmia, decreased reproductive characteristics and sexual activity in females and males, infertility, and embryonic death. During the disease, the serum carotene content of adult cattle and cattle older than 3 months is less than 0.4 mg/100 ml, and the retinol content is less than 20 μg/100 ml. The serum retinol content of beef cattle decreases to 16 μg/100 ml. Hypovitaminosis A occurs when serum retinol levels in calves during the lactation period drop to 4-8 mcg/100 ml.

pigs , the disease is accompanied by a deterioration or complete loss of vision. Piglets are often born blind or later become blind. They have symptoms such as tremors, impaired balance (ataxia), and paralysis of the hind legs. In pigs of all ages, itching and various rashes on the skin are noted, as well as dry skin. In sows , specific changes occur, such as decreased milk yield, nervous disorders, tremors and paralysis, and in male pigs, decreased movement, impaired spermiogenesis, and the appearance of pathologically shaped sperm cells.

Pathoanatomical changes. Metaplasia, glandular atrophy, and inflammatory and sometimes ulcerative lesions are noted in the epithelium of the skin, eyes, and mucous membranes of the hooves, as well as the respiratory tract, digestive tract, and genitourinary tract. In most cases , the disease develops in combination with fatty hepatosis, nephrosis, and osteodystrophy.

Diagnosis . The symptoms of the disease, the amount of carotene and vitamin A in the diet, the results of testing blood, urine (milk) and liver samples for carotene and retinol are taken into account. According to the scientific conclusions of Ya. P. Masalikina (2009), complex hypovitaminosis (A, C, E) in newborn calves In addition to the existing biochemical tests for monitoring the metabolic status, it is advisable to determine the levels of retinol, tocopherol and ascorbic acid in the blood serum to diagnose . Determination of serum carotene levels in calves of this age has low informative value.

Comparative diagnosis. The disease is differentiated from teliasis, rickettsial keratoconjunctivitis , and some infectious diseases of the respiratory and digestive systems.

Prognosis : With timely treatment, a sick animal recovers.

Treatment. Foods rich in carotene or vitamin A are included in the diet (hay, haylage, silage, grass , carrots, and green foods in the summer months). In the treatment of sick animals, oily solutions of retinol alcetate , microvit - A, vitamin fish oil, trivitamin, trivit, ayevit, te tromag and other preparations are used. When determining their dosage, the amount of retinol in their composition is taken into account, in particular, it is recommended to give retinol in the amount of 50,000 - 500,000 IU per day to cattle and horses, 50,000 - 100,000 IU to sows, sheep and calves, 3,000 - 10,000 IU to piglets and lambs, and 3,000 - 40,000 IU to dogs. The course of treatment is on average 15-20 days .

Prevention. Full - fledged feeding of animals is organized . Their needs for retinol and carotene are satisfied. In diseases of the liver, digestive system, endometritis, some infectious and invasive diseases, during stress and during

starvation , when the diet lacks proteins and energy substances, when nitrates and nitrites are present in excess , as well as in cases of tocopherol and zinc deficiency, the amount of carotene and retinol in the diet is increased . When it is impossible to satisfy the animal's need for carotene and vitamin A through natural food, their ready - made preparations are used. Prophylactic doses of such preparations are 2-4 times less than therapeutic doses . However, cows and heifers should be given 600,000-800,000 IU intramuscularly once a week starting 4-6 weeks before calving, and pigs should be given 600,000-800,000 IU intramuscularly once a week starting 4-6 weeks before calving. 250,000 - 350,000 IU of retinol are injected into calves and calves at a dose of 150,000 - 100,000 IU. Cows and heifers at a dose of 5-7, sows and sheep at a dose of 100,000 IU.

Trivitamin is injected intramuscularly in an amount of 2 - 3 ml. In order to prevent the disease in calves, retinol compounds and mixtures are given to them together with the first bite of cow's milk, or for this purpose, 75,000 - 125,000 IU of retinol oil concentrate is injected intramuscularly 1-2 times a week for calves, and 40,000 - 50,000 IU of retinol oil concentrate is injected intramuscularly for piglets and lambs. Starting from the age of 2-3 weeks, calves are given vitamin-rich hay, crushed grass and vitamin flour. Antioxidants (diludin, etc.) are used to protect retinol contained in feed from deterioration.

For the prevention of hypovitaminosis A and E and for the treatment of sick calves, it is necessary to use the drug "Betaviton" in the amount of 5 ml once a day for the first 7 days of the animal's life. Also, for the prevention of hypovitaminosis A, C and E and for the treatment of sick calves, it is recommended to use the drug "Betaviton- C " in the amount of 5 ml once a day for the first 7 days of the animal's life (Ya. P. Masalikina , 2009). The author states that the use of the drug "Betaviton- C " increases the level of vitamin A in the blood serum by 2.7 times.

Hypovitaminosis D - a disease of young growing animals associated with a deficiency of vitamin D, an imbalance of phosphorus and calcium in the body. Leads to the development of rickets. Also, rickets can occur against the background of a deficiency in the body of other vitamins, vital micro- and macroelements, as well as with ultraviolet radiation and gastrointestinal diseases. The main causes of rickets in young cattle:

Vitamin D deficiency; violation of the ratio or deficiency of calcium and phosphorus in the body of a young animal; gastrointestinal diseases; violation of the acid-base balance in the body; lack of physical exercise; lack of exposure to ultraviolet rays in the summer period (non-stop care), in the winter and spring periods - no ultraviolet radiation using mercury-quartz lamps; storage in dark, damp and cold rooms.

Vitamin D (calciferol) or antirachitic vitamin, together with parathyroid hormone, is involved in the metabolism of phosphorus and calcium, as well as in the mineralization and growth of bone tissue in a young animal.

Signs of rickets. Rickets in young cattle develops gradually, so it is very difficult to determine the presence of this disease in the first days. Calves born from cows with metabolic diseases are very weak. A clear sign of rickets in newborn calves is an underdeveloped skeleton. Pain is felt when palpating the hind legs, pelvic bones, and back.

Against the background of poor appetite, calves with rickets develop gastroenteritis and diarrhea. The wool of calves with rickets becomes dull and matted, and the skin loses elasticity. In calves affected by rickets, as a rule, the change of teeth is delayed. They also stagger and fall. In young cattle, sometimes choking and muscle cramps are common (tetany).

In calves aged 3-6 months, development slows down and weight gain does not occur. The animal moves little and remains in a lying position more. Sick calves stand slowly and often cross their legs. The front legs of an animal with rickets are widely spaced when standing.

In severe cases of rickets in calves, the following are observed: respiratory failure; myocardial dystrophy; tachycardia; anemia.

The rare movements of a calf with rickets are accompanied by characteristic crunching and limping in the joints. The movements of the sick animal are very slow, jerky, and the steps are shortened. Pain is felt when palpating the joints. Bone fractures are common among very heavy animals.

Young cattle, up to one year old, also suffer from this disease. In well-developed and well-fed animals, body weight indicators decrease as a result of poor nutrition (lack of appetite) and reduced feed digestibility. Heifers with rickets lie for a long time, show no interest in feeding, move in short steps. When examining the heifer, an increase in the size of the joints, curvature of the spine, limbs are brought to the body.

Diagnosis of the disease. During the diagnosis, the veterinarian evaluates the animal's diet, analyzes the clinical signs of the disease. During the diagnosis, laboratory (biochemical) blood tests are also taken into account with the following definitions : the concentration of calcium and phosphorus in the blood of a sick animal; reserve blood alkalinity; alkaline phosphatase activity.

If necessary, the veterinarian should conduct an X-ray or histological examination of the tissue of the epimetaphyseal zone of the bones . Rickets in young animals has similar symptoms: painful rheumatism; white muscle disease; Urovsky's disease; hypocuprosis (or acuprosis). Therefore, in the differential diagnosis of rickets in young cattle, the veterinarian should exclude these diseases. Treatment of rickets in calves When rickets is detected in newborn calves and young cattle, sick animals should be separated from healthy animals and placed in a dry, warm and spacious room. First of all, the diet of young animals should be reviewed. It should consist of easily digestible feeds rich in protein, vitamins A, D, calcium, phosphorus, macro- and microelements. Sick animals are introduced into the diet and the diet is increased: water grass; alfalfa and alfalfa vitamin hay; red carrots; whole milk and skim milk; yeast feed. Mineral compounds used: shell and bone meal; tricalcium phosphate, calcium glycerophosphate. Oil, alcohol solutions, and vitamin D emulsions are prescribed for the treatment of rickets in young cattle.

Ergocalciferol (vitamin D2) is administered intramuscularly: long-term treatment for a month or more with fractional doses of 5-10 thousand IU;
75-200 thousand IU every 2-3 days (within 2-3 weeks); single dose 500-800 thousand IU.

Complex drugs are also used in the treatment of rickets: orally administered "Trivitamin" (a solution of vitamins D3, A and E) 5-10 drops per day or intramuscularly 1-2 ml once or three times a week; "Tetravit" (a solution of vitamins D3, F, E and A) intramuscularly 2 ml once or twice a week.

Calves with rickets are prescribed fortified fish oil at a rate of 0.4-0.5 g per 1 kg of animal weight orally during feeding three times a day for 7-10 days.

Calves with rickets are irradiated with ultraviolet lamps. Group irradiation of calves is carried out in special rooms. In good sunny weather, young animals should be released for walks in wide open yards.

Prognosis. With timely detection of the disease (especially at an early stage), as well as with proper treatment, an animal suffering from rickets recovers quickly. With late detection of symptoms

of the disease, incorrect diagnosis and the development of complications, the prognosis is poor or doubtful.

In young cattle, the course of the disease is chronic. Rickets in calves is dangerous with the following complications: bronchopneumonia; anemia; severe fatigue; myocardial dystrophy; chronic gastroenteritis; stomach and intestinal catarrh; decreased resistance of the young animal's body to infectious diseases.

Preventive measures. Prevention of rickets in young cattle provides a whole complex of veterinary and zootechnical measures. First of all, calves should be provided with full nutrition. The lack of vitamins, micro and macroelements is compensated by the introduction of vitamin-mineral complexes into the diet of young animals. Calcium, phosphorus, vitamins of group B, D, A and E are especially necessary for animals during pregnancy and when feeding calves with colostrum. Pregnant cows are injected intramuscularly with vitamin D - 250-1000 thousand IU approximately 4-6 weeks before the calving date. If cows have a mineral or vitamin D deficiency, a newborn calf should be fed 50 thousand IU of vitamin D when colostrum is first given.

The room where the young are kept should be spacious, light and warm. It is not allowed to keep animals in crowded, damp, dark rooms. In summer and sunny weather, young animals should be provided with physical exercise in the fresh air. In spring, autumn and winter, it is necessary to organize irradiation under special ultraviolet lamps.

Conclusion. Rickets in young animals occurs as a result of impaired mineral metabolism in the body, as well as a deficiency of vitamin D, calcium and phosphorus . This dangerous disease is primarily a consequence of violations of the norms of feeding and keeping calves and pregnant cows. With timely treatment, sick calves recover quickly, in severe cases they die from severe complications.

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