Volume 4, Issue 4, April 2026 ISSN (E): 2938-3765

BOVINE LUMPY SKIN DISEASE

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Abstract
An analysis of literature data and the results of our own research on the characteristics of the causative agent of bovine lumpy dermatitis (LD), distribution, clinical signs, cultivation, diagnosis and prevention of the disease are presented.

Keywords: Lumpy skin disease virus, cattle, spread, diagnostics, disease clinical signs.

Introduction

Lumpy dermatitis (cutaneous tubercle, nodular dermatitis, cutaneous nodular rash, cutaneous edema disease in buffaloes, patchy skin disease, viral, contagious skin tubercle, nodular exanthema of cattle) is a disease of cattle characterized by fever, lesions lymphatic system, swelling of the subcutaneous tissue and internal organs, the formation of skin nodes (bumps), damage to the eyes and mucous membranes of the respiratory and digestive tracts. Lumpy dermatitis in cattle is considered a particularly dangerous animal disease that can cause epizootics and cause significant economic damage. In accordance with the new classification, it is included in the OIE list of diseases subject to mandatory notification (notification) in the category “Diseases and infections of cattle” [5]. Historical reference. The disease was first discovered in Zambia in 1929 as an allergic reaction to multiple insect bites. Lumpy dermatitis was recognized as an infectious disease by F. Backström when an outbreak occurred in Nglamiland during 1943. By the end of 1944, the disease had manifested itself in the Transvaal, and over the following years, despite forced control measures, it quickly spread throughout South Africa. In Southern Rhodesia the presence of the disease was established in 1945 and by 1947 the disease had become enzootic in South Africa. The spread of infection was noted in Swaziland, Basutoland and Portuguese East Africa, and then in Madagascar, Tanganyika, Belgian Congo, Botswana and South Africa, where more than 8 million livestock were affected. In 1957, the disease appeared in Kenya simultaneously with an outbreak of sheeppox, and in the 70–80s. –in Sudan, Nigeria, Mauritania, Mali and other African countries, where it caused the death of up to 20% of infected livestock [21]. In 1988, the disease appeared in Egypt, and a year later in Israel [3]. ND was first reported outside Africa, in the Middle East, in 1991 [15]. 46 Current issues in veterinary biology. Current issues in veterinary biology. Distribution of nodular dermatitis in the world. Currently, the disease occurs in 34 countries in Africa and Asia [28]. The most recent outbreaks outside Africa occurred in the Middle East in 2006 and 2007, and on the island of Mauritius in 2008 [21]. Outbreaks of bovine ND were observed in Egypt (2006), Israel (2006 and 2007), Palestine (2007 and 2008) and Bahrain (2006–2009). It is possible that ND was introduced into this area through the import of live animals from countries where lumpy

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skin disease is endemic on the African continent. If proper veterinary and sanitary rules are not followed, cattle ND can quickly spread to Asia (to the east) and Europe (to the north) through the Middle East and Turkey [14]. More recently, outbreaks of nodular dermatitis have been reported in Guinea (2011), Israel (2012 and 2013), Turkey (2013 and 2014), Egypt, PAT, Lebanon, Jordan, Iraq, Azerbaijan (2014) [28, 29]. In July, in Azerbaijan, the disease lumpy skin disease was registered for the first time among cattle in the Bilasuvar, Ujar, Jalilabad and Agdash regions. It is believed that the infection was introduced by blood-sucking insects from Iran and Turkey [30]. FAO (FAO -Food and Agriculture Organization of the United Nations) -comments on the causes of the emergence of lumpy skin disease in the countries of the Eastern Mediterranean: "Uncontrolled movements of livestock increase the risk of possible spread of lumpy skin disease to new territories in Turkey or the country gions are free from this disease. Examples of these include the documented, seasonal, large-scale transhumances of livestock to new pastures conducted by the Kurds in the southern Anatolia region. In addition, the Tigris and Euphrates river deltas in Iraq have the potential to be vector transmission hotspots due to thepresence of riverine agriculture and related activities there. There is an immediate risk of introducing nodular dermatitis into the Islamic Republic of Iran and the Transcaucasian countries" [30]. For Russia, the indication of nodular dermatitis on the territory of Azerbaijan and the approach of the disease to the borders of Russia is of great concern [30]. Taxonomy of lumpy skin disease virus. The causative agent of the disease is the bovine lumpy skin disease virus, which is antigenically related to thestrains of viruses that cause smallpox in sheep and goats, which differ at the genetic level, and together with it form an independent genus Capripoxvirus, family Poxviridae. The genus Capripoxvirus (from the Latin capri -goat) contains sheeppox viruses (SPPV -Sheeppox virus (prototypic)), goatpox (GTPV -Goatpox virus) and tuberculosis viruses (LSDV -Lumpy skin disease virus) [10]. Neethling group viruses are the main causative agent of nodular dermatitis. They also cause cytopathic changes (CPI) in the culture of calf kidney cells, sheep embryos and testicular tissue of lambs and calves no earlier than 14 days after infection. In terms of its cytopathic action, the Neethling virus is similar to smallpox viruses. The structure of the virus. In 1966, Munz and Owen, studying negatively stained preparations at pH 6.5 in an electron microscope, discovered virus particles that were morphologically different from the M-forms of the vaccinia virus described by Westwood et al. These particles, in turn, were similar with C-form vaccinia virus, which were described by Westwood, or type 2 vaccinia virus particles by Nagington and Horne, Müller and Peters. In the modern concept, viruses of the genus Capripoxvirus have a brick-shaped shape, measuring 300 × 270 × 200 nm. At the center of the virion is a nucleoid containing DNA. Adjacent to it are lateral bodies, giving the nucleoid the appearance of a dumbbell, and an outer two-layer shell. The genome consists of 150–160 thousand base pairs [3, 6]. DNA is not infectious. 88% of the virion's mass is proteins, 5% is DNA, 4% is lipids and 3% is carbohydrates. Stability of the pathogen. According to Plowright, Ferris and Weiss, lumpy skin disease virus is stable between pH 6.6 and 8.6, showing a slight reduction in titer over 5 days at

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37°C. The Neethling virus tolerates 3 times freezing and thawing well, but is sensitive to a 20% solution of ether and chloroform. Weiss and A. Mayr proved that it is extremely stable in the environment and at room temperature the infectivity persists for 18 days, in the affected areas of the skin -at least 33 days, in saliva -11, in bull semen -for 22 days, in blood and in some internal organs -4 days, in cell culture at +4 °C -6 months, at a temperature of minus 80 °C, viability remains for more than 10 years. Neethling virus was detected in the blood of animals within 22 days after the onset of symptoms of the disease. Alexander and Weiss showed the presence of the virus in the blood for 4 days in experimentally infected animals, accompanied by the appearance of fever and the spread of skin lesions. Antigenic properties of lumpy skin disease virus. The antigenic properties, variability and relatedness of the ND virus have not been fully studied. Antigenically, the Neethling virus is related to the sheep and possibly goat pox virus.

Clinical signs of the disease. The largest outbreaks in Africa were caused by the Neethling virus. The disease caused by this type of pathogen is especially difficult. It is considered as a genuine cutaneous tubercle. The incubation period averages 7 days, but can last up to 5 weeks. It depends on the susceptibility of the animal, the type and virulence of the pathogen and the routes of its penetration into the body. The prodromal period is short and often goes unnoticed, especially when the first cases of the disease appear on the farm. In sick animals, the body temperature rises to 40 °C, watery discharge from the eyes, and lethargy appear. Animals refuse food and quickly become exhausted. The lymph nodes are enlarged and easily palpable on the thighs and especially in the prescapular area. Superficial lymph nodes sometimes have the appearance of swelling. In severe cases of the disease, the oral cavity, respiratory and digestive organs are affected. Thick viscoussaliva is released from the mouth, and purulent mucus with a fetid odor is released from the nose. The watery discharge from the eyes is replaced by mucous, and when it dries, crusts form. Erosion and ulceration appear on the eyelids. Conjunctivitis is sometimes observed; The cornea becomes cloudy, which can lead to partial or complete blindness. Ulcerations that appear in the respiratory tract cause severe swelling, and the animal dies from suffocation [2, 4, 8]. Throughout the body, and sometimes only onthe limbs and abdomen, intradermal tubercles with a flat surface are formed (diameter 0.5–7 cm, height up to 0.5 cm); the number of nodules ranges from 1–10 to several hundred. In some parts of the body, the tubercles merge. Sometimes they form under the skin and are detected only when palpated. Along the edges of the tubercles, the epidermis is separated, and in the center the tissue becomes necrotic and a characteristic depression is formed, bordered by a ridge of granulation tissue measuring 1–3 mm. 1–3weeks after the appearance of the tubercle, the necrotic tissue disappears. During an aseptic course, the cavity quickly fills with granulation tissue and is overgrown with hair of a slightly different color. With complications (secondary infection), swelling appears in the deep layers of the skin and subcutaneous tissue. In lactating cows, the udder is affected. It is increased in volume, nodules are visible on it; The milk is thick, with a pink tint, comes out in drops, and when heated turns into a gel. The disease lasts about 4 weeks, with complications –longer. Complications with tuberculosis often include tracheitis, pneumonia, accompanied by tracheal atresia and difficulty breathing,

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damage to the genital organs, missed 4-6 heats, and in males -temporary sexual sterility. The disease is often complicated by a secondary bacterial infection, affecting the joints, lungs and other organs.

Pathological: changes are characterized by the presence of widespread necrotic nodular lesions on the skin. Lymph nodes are swollen and juicy when cut. Under the visceral pleura, sometimes on the turbinates of the nasal passages, in the spleen, liver and scar, stellate hemorrhages can be found. The lungs are swollen, sometimes nodules are visible in them. In the nasal passages and in the omentum, signs of stagnant processes are found, under the kidney capsule there are small nodules (2–3 mm), in the abomasum there is diffuse inflammation, sometimes ulceration of the fundus and pylorus, in the ligament 50 Histomorphological changes depend on the stage of development of the process. In the initial stage of the disease, epithelial cells are enlarged and vacuoles appear in them. In histosections of the tubercles, cytoplasmic inclusions of round or oval shape are found, often larger than the nucleus. These inclusions are contained in epithelial cells and histiocytes.

Diagnostic methods: According to foreign authors, the diagnosis is made based on the analysis of epizootic and clinical data, pathoanatomical and histological changes, as well as the results of laboratory tests. At various phases of disease development, antigen determination is carried out by solid-phase ELISA, immunofluorescence reaction, immunodiffusion reaction in agar gel, polymerase chain reaction (PCR), virus neutralization reaction, indirect fluorescent antibody reaction, Western blotting, and virus detection is by infection of cell culture and electron microscopy.

Differential diagnosis. Lumpy skin disease virus should be differentiated from other smallpox viruses that cause clinically similar diseases in cattle, also manifested by damage to the skin and mucous membranes. These include vaccinia viruses, cowpox and buffalopox, which are part of the genus Orthopoxvirus, bovine papular stomatitis viruses and bovine pseudopox, which form the genus Parapoxvirus. The group of orthopoxviruses has the same virion structure (brick shape) as the ND virus, and parapoxviruses differ in the shape of the virion, which looks like a “cocoon” measuring (220–300) × (140–170) nm with lateral flaps adjacent to it on both sides bodies and an outer shell, consisting of tubular protein structures that wrap around the virion with 10–12 turns. In addition, the ND virus should be distinguished from the bovine herpes virus (Allerton), which also causes skin pathology, the so-called “pseudo-tubercular dermatitis”, accompanied by nodular and ulcerative lesions of the skin of the teats and udder of cows and other parts of the body. Immunity in nodular dermatitis. Weis showed that virus-neutralizing antibodies in recovered animals persisted for at least 5 years, and immunity was likely “lifelong.” Passive immunity obtained by taking colostrum lasted for 6 months (V. D. Westhuizen) [18, 19]. According to Adelaar and Neitz, immunity after infection with nodular dermatitis was short-lived (11 months) [26]. Animals that have recovered from the disease are resistant to re-infection.

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However, according to some researchers, the duration and intensity of post-infectious immunity vary. There is no cross-trained immunity between the above types of virus [2, 3, 21]. Specific prevention of nodular dermatitis. To prevent tuberculosis, a vaccine is used from the attenuated Neethling strain of lumpy skin virus, obtained by passage in lamb kidney cell culture andon the chorioallantoic membranes of chicken embryos, as well as from 3 Kenyan strains of sheeppox virus (Kedong, SP-143 and Isiolo). Immunity to nodular dermatitis is formed starting from the 10th day and reaches its maximum value 21 days after vaccination. Duration of post-vaccination immunity –1 year [22]. At various phases of disease development, antigen determination is carried out by solid-phase ELISA, immunofluorescence reaction, immunodiffusion reaction in agar gel, polymerase chain reaction (PCR),virus neutralization reaction, indirect fluorescent antibody reaction, Western blotting, and virus detection is by infection of cell culture and electron microscopy. Differential diagnosis. Lumpy skin disease virus should be differentiated from other smallpox viruses that cause clinically similar diseases in cattle, also manifested by damage to the skin and mucous membranes. These include vaccinia viruses, cowpox and buffalopox, which are part of the genus Orthopoxvirus, bovine papular stomatitis viruses and bovine pseudopox, which form the genus Parapoxvirus. The group of orthopoxviruses has the same virion structure (brick shape) as the ND virus, and parapoxviruses differ in the shape of the virion, which looks like a “cocoon” measuring (220–300) × (140–170) nm with lateral flaps adjacent to it on both sides bodies and an outer shell, consisting of tubular protein structures that wrap around the virion with 10–12 turns. In addition, the ND virus should be distinguished from the bovine herpes virus (Allerton), which also causes skin pathology, the so-called “pseudo-tubercular dermatitis,” accompanied by nodular and ulcerative lesions of the skin of the teats and udder of cows and other parts of the body. The morphological features of this virus correspond toherpes viruses. Immunity in nodular dermatitis. Weiss showed that virus-neutralizing antibodies in recovered animals persisted for at least 5 years, and immunity was likely “lifelong.” Passive immunity obtained by taking colostrum lasted for 6 months (V. D. Westhuizen). According to Adelaar and Neitz, immunity after infection with nodular dermatitis was short-lived (11 months) [26]. Animals that have recovered from the disease are resistant to re-infection. However, according to some researchers, the duration and intensity of post-infectious immunity vary. There is no cross-trained immunity between the above types of virus. Specific prevention of nodular dermatitis.

Conclusion:

Tubercles use a vaccine from an attenuated Neethling strain of lumpy skin virus obtained by passage in lamb kidney cell culture and on the chorioallantoic membranes of chicken embryos, as well as from 3 Kenyan strains of sheeppox virus (Kedong, SP-143 and Isiolo). Immunity to nodular dermatitis is formed starting from the 10th day andreaches its maximum value 21 days after vaccination. The duration of post-vaccination immunity is 1 year.

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References

1.Epizootology of farm animals. Ministry of Agriculture of the USSR as a textbook for students of higher agricultural education in the specialty of Veterinary Medicine, p. 308 Moscow “Spike” 1984.

2.Мухамедова, М. Г. (2023). Распространенность Электрокардиографических Предикторов Внезапной Смерти У Здоровых Мужчин Призывного Возраста. Central Asian Journal of Medical and Natural Science, 4(3), 1172-1180.

3.Food and Agriculture Organization of the United Nations, FAO Livestock and Animal Health Service. Practical guidelines for veterinarians, Bulgarian Food Safety Authority BFSA.

4.Infectious diseases are one of the main obstacles in the development of rabbit breeding “Veterinary Journal” 7 sleep, 2023. Ashurov S.A.