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Prevention and Management Strategies of Lumpy Skin Disease in Indian Dairy Cattle

Satyabrat Dutta[1], Rajashree Bhuyan[2], Kaushik Poran Bordoloi[3]

MVSc Student, Department of Veterinary Microbiology, CVSc & A.H, Selesih, Aizawl, India[1]

BVSc & A.H Student, Lakhimpur College of Veterinary Science, AAU, Joyhing, NLP, India[2]

MVSc Student, Department of Veterinary Medicine, CVSc & A.H, Selesih, Aizawl, India[3]

Abstract: Lumpy Skin Disease (LSD), also termed as Knopvelsiekte or Neethling is a highly infectious, eruptive, occasionally fatal vector born disease of cattle caused by a virus of the family Poxviridae under the genus Capripox virus. The causative agent of the disease seems to be spread mainly by arthropod vectors, including biting flies, midges, mosquitoes, ticks and various insects. In addition to vectors, transmission through consumption of contaminated feed or water, direct contact with infected animals and also through natural mating or artificial insemination has been reported. Clinical signs of the disease are characterized by enlarged lymph nodes, oedema of the skin and legs, multiple nodules on the skin, mucous membranes, and internal organs followed by eruption of nodules, fever, emaciation, and sometimes death. Although the mortality rate of the disease is generally low but the various factors like, highly contagious nature, carrier status in convalescent animals and substantial economic losses due to permanent damage to hides, the prolonged debilitating effect especially in severely affected animals with consequent losses resulting from reduced weight gain, temporary or permanent cessation of milk production as a result of mastitis, temporary or permanent infertility or even sterility in bulls as a consequence of orchitis, and abortion in approximately 10% of infected pregnant cows, reduction in meat and milk yields make it a disease of major concern. Moreover indirect losses stem from restrictions on cattle movements and trade. Till now no clinically approved vaccine is available in India for this disease. However, as the cattle become infective before appearance of clinical lesions on the skin, slaughter of clinical cases has not been very effective in preventing the spread of disease. Quarantine, movement control, dipping and spraying of cattle with insecticides and spraying of farm buildings have been found to limit the spread of infection up to certain extent.

Keywords: Lumpy Skin Disease (LSD), Knopvelsiekte, Neethling, vector born, multiple nodules, abortion, quarantine.

I. INTRODUCTION

Lumpy Skin Disease (LSD) is a viral disease of cattle caused by Capripox virus which is a member of Poxviridae family. Both *Bos indicus* and *Bos Taurus* are equally susceptible to the disease irrespective of age, sex and breed of the animal. It may be possible that water buffaloes are reservoir host and maintain the virus for a prolong period of time. LSDV has structural similarities with the other member of the family Poxviridae in having a dumbbell-shaped core enclosing dsDNA genome and two lateral bodies surrounded by a multilayered capsule. Presence of growing numbers of naïve (i.e. immune deficit) animals, abundance of active blood-sucking vectors, and uncontrolled animal movements are usually drivers for extensive LSD outbreaks. The disease is not zoonotic (meaning that it does not infect Human Beings) and spreads principally by blood sucking vectors even though direct contact and indirect contact may also serve as sources of transmission. Arthropod vectors, including biting flies, stable fly (*Stomoxys calcitrans*) and mosquitoes (*Culex mirificus* and *Aedes natronius*), midges, ticks (*Riphicephalus appendiculatus* and *Amblyomma hebraeum*) and various insects have been incriminated in transmission of the disease. Infected animals showing multiple lesions in the skin and mucous membranes of the mouth and nasal cavities excrete virus particle in saliva, as well as in nasal and ocular discharges, which may contaminate shared feeding and drinking sites. The virus persists in the semen of infected bulls so that natural mating or artificial insemination may be a possible source of infection for females. The virus may be transmitted to suckling calves through trans placental or trans mammary route, or from skin lesions in the teats.

II. EPIDEMIOLOGY

LSDV was originally endemic in Southern and Northern Africa and was confined to the continent but in the last decade has emerged to many other countries and is now endemic in Middle East and still continues to spread. LSD possesses a potential threat of causing economic impacts on livelihood of small holders even though the mortality is low as it causes a direct decline in production by causing lowered milk production, abortion, damage to animal hides, and indirectly by its restrictions on cattle trade. The disease was first reported in Zambia in 1929. In 2019 reports were received from

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Russia, West Bank, Turkey, Israel, China, Bangladesh and India. The first occurrence of LSD in India was reported to the OIE on 18th November 2019 in various states like Odisha, West Bengal and Assam.

III. CLINICAL SIGNS

LSD signs range from inapparent to very severe disease. There is no current evidence of variation in virulence among the different LSDV strains.The incubation period in experimentally infected animals varies between only 4-7 days, but in naturally infected animals it may be up to 2-5 weeks. Clinical signs of the disease include:

- Severe nasal discharge and lachrymation followed by rhinitis, conjunctivitis and excessive salivation. –Usually observed first.
- Swelling of Subscapular and prefemoral lymph nodes and are easily palpable.
- Very high rise of temperature (>41C) may persist for several days.
- Limbs and other ventral parts of the body, such as the dewlap, brisket, scrotum and vulva, may be oedematous, causing the animal to be reluctant to move.
- Marked reduction in milk yield of lactating animals.
- Generalized lymphadenitis, anorexia and oedema of limbs, abdomen and brisket followed by lumps.

Appearance of highly characteristic, distinguishable nodular nodules (number of lesions varies from a few in mild cases, to multiple lesions in severely infected animals) measuring 1-3 cm in diameter and 1-2 cm deep over the entire skin. The lumps are circular and hard with well-demarcated edges. Sites of predilection are mainly skin of the head, neck, perineum, genitalia, udder and limbs. These nodules are circumscribed, firm, round and raised, and involve the skin, subcutaneous tissue and sometimes even the underlying muscles.

- Nodules in the skin may persist for longer period of time.
- Within a week, the edge of nodules begins to separate from the surrounding skin by a necrotic zone eventually by fibrosis. This stage is known as ‘sit fast’ stage. The sit fast lesions become necrotic and in next 4-6 weeks sloughs off leaving a deep granulating pock
- Sometimes, painful ulcerative lesions may develop in the cornea of one or both the eyes, leading to keratitis followed by blindness in worst cases.
- The lesions on the mucosa are of punched-out type and are mostly seen on the dental pad, commissures, edge of the dental pad and occasionally in the gum.
- Skin lesions in the legs and on top of the joints may lead to deep subcutaneous infections complicated by myiasis, secondary bacterial infections and lameness. Pneumonia caused by the virus itself or secondary bacterial infections, mastitis and abortion in pregnant animals are common sequelae.
- Subclinical infections are more common in the field condition.
- Bulls may become permanently or temporarily infertile.
- When an animal with multiple skin lesions is sent to a slaughterhouse, subcutaneous lesions are clearly visible after the animal is skinned.
- In autopsy, pox lesions can be found throughout the entire alimentary and respiratory tracts and on the surface of almost any internal organ.

IV. PATHO-PHYSIOLOGY

The lumps on the skin are hard fibrous mass and situated deep enough within the corium and in some cases could involve the underlying muscles still. The necrotic lesions are found within the mucous membrane of cavum, rumen, reticulum, omassum, abomasum, nasal mucous membrane and lungs. The lesions within the respiratory organ and trachea are typically scattered and within the type of yellowish-grey mass of one cm diameter.
Microscopically, the affected space of skin shows foci of necrosis within the corium related to aggregation of mononucleate cells significantly the lymphocytes, macrophages, some plasma cells and fibroblasts. The massive intracytoplasmice eosinophillic inclusion bodies are typically seen in macrophages and fibroblasts and infrequently within the epithelial tissue cells of minute vessels. As a result, the coagulum formation of vascular wall becomes a constant finding. In some cases, the cuticle may be involved in conjunction with corium and therefore the cells of basal layer exhibits vacuolation, margination of nuclear chromatin granule and infectious agent fingerprints. Most of the lesions on the skin significantly at sit fast stage involve all layers of skin and edema, hemorrhages and marked cellular infiltration are seen within the upper layers of dermis leading to destruction of cuticle and formation of a death plug that extends from original focus to cuticle. The healing starts from edges of the lesions, as is evidenced by proliferation of fibroblasts. In most cases, the granulating-tissue step by step extends beneath the death plug and eventually expels it out leaving a raw healing lesion. The lesions on the secretion membranes exhibit similar pathological changes.

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I. **Pathogenesis**

After exposure of animals to the virus significantly through bite of insects, the febrile stage develops 5-7 days later and nodules seen on the skin by day 7-9 PI. The multiplication of virus takes place within the corium and leads to viraemia that coincides well with pyrexia that lasts for 4-5 days. Throughout terminal stages of febrile reaction, the lesions develop over the skin and sometimes in internal organs. The virus is excreted for 11-12 days in saliva and for 22-25 days in seminal fluid but not in the urine or faecal matter. The virus has additionally been isolated from regional lymph nodes, spleen, muscle and normal uninfected skin. The spread of infection to other healthy animals is of minor importance and primarily depends on accessibility of animals to invertebrate vectors.

V. DIAGNOSIS

Diagnosis is especially based on signs and symptoms such as characteristic cutaneous lesions. Demonstration of classic poxvirus particle in the skin nodules by transmission electron microscopy however cannot differentiate to genus or species level. The tissue samples collected for microscopical examination may be used for electron microscopy. The massive cytoplasmic inclusion bodies in early stages of infection and isolation of virus on CAM of developing chicken embryos/cell culture or electron microscopy of skin sections ensure the diagnosis. The confirmation may be achieved by demonstrating the viral antigen within the cryostat sections of skin or infected monolayer as early as 48 hour PI by indirect immunofluorescence. Additionally, the cytopathological changes significantly the moth eaten appearance of monolayer due to rounding, shrinking and detachment of cells and presence of intracytoplasmic inclusion bodies provide robust proof of LSDV infection. Polymerase chain reaction (PCR) is the cost effective and fastest method for detection of LSDV. Skin nodules and scabs, saliva, nasal secretions, and blood are appropriate samples for PCR detection of LSDV. Serological test like virus neutralization test is also considered as gold standard test for the detection of antibodies raised against capripox viruses. Capripox virus antibody enzyme-linked immunosorbent assay is a new commercial kit for detection of capripox virus antibodies are presently being developed and released on to the market.

Misdiagnosis of skin lumps and misreporting of infection have most likely been common over the years due to veterinarians not having previous expertise of the illness. Though severe LSD is VERY characteristic, but milder forms can be confused and misdiagnosed with VARIOUS diseases and infections such as pseudo lumpy skin disease (*Bovine Herpes virus*), *bovine papular stomatitis* (Para poxvirus), *pseudo cowpox* (Para poxvirus), *Vaccinia* virus and *Cowpox* virus (Orthopoxviruses) infections, *dermatophilosis*, insect or tick bites, *besnoitiosis*, *rinderpest*, *demodicosis*, *Hypoderma bovis* infection, *photosensitisation*, *urticaria*, cutaneous tuberculosis and *onchocercosis*.

VI. TREATMENT, PREVENTION & CONTROL

Till this moment, no specific antiviral treatment has been found against Lumpy Skin Disease. Sick animals should be eliminated from the herd and supportive and symptomatic treatment like antibiotics, anti-inflammatory drugs, and vitamin injections should be given. These therapies are used to to cut back possibilities for the event of secondary microorganism infections, inflammation and fever, and so rising the appetite of the animal. Generally, animals infected with LSD can recover as mortality is usually less than 3% though morbidity is incredibly high. If secondary microorganism infection developed, complete recovery could takes over a months or Longer. Successful control and obliteration of LSD relies on early detection of the all clinical and subclinical index case, followed by a speedy and widespread mass vaccination campaign. The simplest protection comes from prophylactic vaccination of the whole cattle population, allotted well beforehand in at-risk areas. Currently used vaccines against LSD in different part of the world are:

Attenuated LSDV vaccines

Currently, vaccine producer manufacturing attenuated LSDV vaccines. Live, attenuated LSDV vaccines offer smart protection in cattle if 80 % vaccination coverage is earned. In practice, all cattle population need to be vaccinated, including small calves and pregnant cows. Regional vaccination campaigns should be preferred to ring vaccination. Example of live attenuated strains of capripox virus vaccines has been utilized in order to regulate LSDV outbreaks (not authorised to be used within the United States) are:

- Kenyan sheep and goat pox virus strain,
- Yugoslavian RM sixty five sheep pox strain,
- Romanian sheep pox strain, and
- LSDV strain from Republic of South Africa.

Attenuated SPPV vaccines

Sheep pox virus vaccines are utilized in cattle against LSDV in those regions wherever LSD and SPP are both present. Because the protection provided by SPPV vaccines against LSDV is believed to be partial, choice of the immunizing agent should be based on demonstrated efficacy of the vaccine against LSDV by a challenge trial allotted in a controlled

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atmosphere. If acceptable efficacy of the SPPV/GTPV vaccines is demonstrated, SPP vaccines may be used only if full vaccination coverage and alternative applicable management measures are in place.

Attenuated Gorgan GTPV vaccine

Commercially available GTPV Gorgan strain has been incontestable to produce equal protection against LSD as the LSDV vaccines (Gari *et al.*, 2015). Gorgan GTPV vaccine may be a good, cost-efficient alternative in those countries wherever GTP and LSD usually overlap.

No Differentiation of Infected from Immunised Animals (DIVA) vaccines have been developed against LSD so far. Vaccines don't seem to be obtainable in each part of the world. It is restricted in some part of the globe, Moreover Live attenuated vaccines are allowed for use in bovine in Africa, however in alternative presently affected regions specific authorization is needed before their use. Measures to be taken for prevention and controls where no vaccines are clinically approved (Including India):

- If a suspected case of LSD is detected by an owner, personal vet, animal trader, cattle truck driver, artificial inseminator, or any other visitant, a competent veterinary authority should be informed at once and an official veterinarian/veterinary team should visit the farm to carry out an epidemic investigation. If possible, separate the suspected case(s) from the rest of the herd. If attainable, separate the suspected case(s) from the remainder of the herd and separate by feeding them on the farm and avoiding communal grazing. Severely affected animals should be aloof from the herd as a result of they function as continuing supply of contamination.
- Stop animal movement from/to the farm and limit visitors to essential services.
- Movements of animal within the country and across borders ought to be strictly controlled or all prohibited. Approved animal movements ought to be in the midst of a veterinary certificate
- In affected villages, dairy herds ought to be unbroken break free alternative herds by avoiding communal grazing, if attainable while not animal welfare problems. However, in some cases the total village forms one epidemiological unit and so the practicability of separation must be evaluated on an individual basis.
- Cattle should be treated frequently with insect repellents to reduce the chance of vector transmission of the disease. This measure cannot totally stop transmission however could cut back the chance.
- Once a stamping-out policy is enforced, culling and disposal of carcasses should take place as shortly as attainable in compliance with all animal welfare and safety necessities. Disposal of carcasses ought to be conducted by burial, burning or rendering, in line with national procedures.
- Lumpy skin condition virus is incredibly stable and survives well in extremely cold and dry environments. Infected animals shed scabs from skin lesions. Within the scabs, the virus could stay infectious for many months. Personnel ought to conjointly endure medical care.
- Though LSDV is sensitive to most disinfectants and detergents, so as to effectively cleanse animal facilities and holdings, mechanical removal of surface material like dirt, manure, fodder and straw is needed beforehand.
- Efficient insect management on dairy herd could cut back the speed of mechanical transmission, though unable to prevent it completely. Anti-mosquito nets are often thought-about in cases once bovine area unit for good unbroken inside. The appliance of spot-on repellents will defend animals from insects and ticks for brief periods.
- Limiting vector breeding sites like standing water sources, slurry and manure, and rising emptying in holdings area unit property, reasonable and environment friendly ways that of reducing the quantity of vectors on and around cow
- In the event of LSD entering a country, farm biosecurity should be raised to the best possible level, taking into thought the boundaries of the epidemiological unit in every case. As the disease is spread by vectors, such measures might not totally prevent an incursion, however the chance are often reduced.
- Purchase of new animal that are either incubating the disease or are carrier while not exhibiting any symptoms presents a significant risk of introducing the disease into naïve cattle shed. Introduction of new animals into herds should therefore be restricted. Stock should be bought solely from sure sources. New animals should be examined and declared freed from clinical signs before movement and on arrival, and should be kept isolated from the herd for a minimum of twenty-eight days.
- All visitant vehicles and instrumentality should be cleansed during a wash-down bay once getting into farms. Boots should even be cleansed or, instead, shoe covers should be worn. guests getting into farms ought to wear clean protecting article of clothing.
- Awareness campaigns should be targeted at official and personal veterinarians, field and building, veterinary students, farmers, herdsmen, animal traders, animal truck drivers and artificial inseminators.

Surveillance programmes are based on active and passive clinical surveillance and laboratory testing of blood samples, nasal swabs, or skin biopsies collected from suspected cases. As there are no DIVA vaccines against LSD, serological surveillance is of no use in affected countries or zones wherever the complete cattle population is immunised. However, serology can be used whenever the presence of unnoticed/unreported outbreaks are investigated in disease-free regions either bordering, or in close proximity to, affected areas with unimmunised animal. In such areas, the presences of seropositive animals are often thought-about as a sign of recent outbreaks.

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